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Quantitative Analysis of Capillary Density in High Myopia Patients with Diabetic Retinopathy: A Retrospective Monocentric Study Using OCT-A

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Abstract

Objective - Diabetic retinopathy (DR) is a major microvascular complication of diabetes, characterized by retinal capillary alterations and progressive ischemia. Some epidemiological studies based on fundus examination have suggested a protective effect of high myopia (HM) against the development and progression of DR. This study aims to quantitatively analyse retinal capillary density and non-perfusion areas in highly myopic patients with DR, comparing them to emmetropic diabetics and highly myopic non-diabetics using Optical Coherence Tomography Angiography (OCT-A).

Methods - In this retrospective, monocentric, observational study, we included patients with type 1 or type 2 diabetes and high myopia (HMDB). We also included matched emmetropic diabetic patients (DB), as well as matched non diabetic subjects with high myopia (HM). High myopia was defined as an axial length > 26 mm and/or refraction ≤ -6D. Superficial and deep capillary density, as well as the area of the foveal avascular zone, were assessed using a 3x3 mm OCT-A scan (Optovue®, AngioAnalytics software).

Results – Twenty-six eyes of 15 patients were included in the HMDB group, 26 eyes of 15 patients in the DB group and 26 eyes of 16 patients in the HM group. Superficial capillary plexus (SCP) density was significantly lower in both HM and HMDB groups compared to DB. However, no significant differences were found between HM and HMDB. Deep capillary plexus (DCP) density was significantly lower in HM compared to DB, while HMDB showed higher DCP values than HM, suggesting a distinct vascular profile. No significant differences were observed in foveal avascular zone (FAZ) area across groups. Adjusted models showed SCP decreased with diabetic retinopathy (DR) severity, while DCP showed a non-significant decreasing trend. In the HMDB group, DCP appeared elevated at earlier stages but declined with increasing pathology.

Discussion – Our findings suggest that diabetes and HM have distinct and potentially interacting effects on retinal microvasculature. The preservation of DCP in HMDB may reflect early compensatory vascular remodeling. This supports a model where DCP is relatively spared in early-stage DR among individuals with HM, followed by capillary dropout as disease progresses.

Introduction

Diabetic retinopathy (DR) is a leading cause of preventable vision loss worldwide and is characterized by progressive microvascular damage, capillary non-perfusion, and retinal ischemia[1]. Chronic hyperglycemia induces structural and functional alterations of the retinal vasculature, including pericyte loss, endothelial dysfunction, and increased vascular permeability, ultimately leading to capillary dropout and neovascular complications[2,3]. Optical Coherence Tomography Angiography (OCT-A) enables non-invasive, high-resolution assessment of retinal microvasculature, allowing quantitative evaluation of capillary density within the superficial capillary plexus (SCP) and deep capillary plexus (DCP), as well as measurement of the foveal avascular zone (FAZ). The SCP primarily reflects the inner retinal circulation, whereas the DCP supplies deeper retinal layers and exhibits distinct structural and functional

characteristics. Previous studies have demonstrated reductions in capillary density in both plexuses in patients with DR compared with healthy subjects[4,5].

Epidemiological studies have suggested a lower prevalence and slower progression of DR in patients with high myopia (HM), particularly in association with increased axial length (AL)[6,7]. However, most of these observations are based on fundus photography and clinical grading systems, such as the Early Treatment Diabetic Retinopathy Study (ETDRS) classification, without detailed assessment of retinal microvascular perfusion. More recent OCT-A studies have reported decreased macular capillary density in highly myopic eyes[8], but data remain limited and heterogeneous, particularly in patients with co-existing diabetes. In addition, few studies have included appropriate comparison groups to disentangle the respective effects of myopia and diabetes on retinal microvasculature[9].

As a result, the interaction between high myopia and diabetic microvascular alterations remains incompletely understood. In particular, it remains unclear whether high myopia exerts a protective, neutral, or modifying effect on capillary density changes associated with DR, and whether such effects differ between retinal vascular layers.

This study aims to quantitatively compare macular capillary density and foveal avascular zone area among highly myopic patients with diabetes, emmetropic patients with diabetes, and highly myopic non-diabetic subjects using OCT-A.

Methods

Study Population: This retrospective monocentric study was conducted at Hôpital Lariboisière between 2018 and 2023. The inclusion criteria for the High Myopia and Diabetes (HMDB) group were: type 1 or type 2 diabetes and high myopia (HM). High myopia was defined as AL > 26.0 mm and/or spherical equivalent ≤ -6.0 diopters. The Diabetes (DB) group included type 1 or type 2 diabetic patients without high myopia. The High Myopia (HM) group included non-diabetic subjects (HbA1c < 6.5% and no antidiabetic treatment) with high myopia. Matching was performed within ± 5 years based on age, DR stage, and diabetes type. Matching was performed at the patient level, with both eyes included when eligible and of sufficient image quality. Exclusion criteria included: (i) active or prior diabetic macular edema; (ii) myopic maculopathy (e.g., patchy atrophy, chorioretinal atrophy, or myopic choroidal neovascular membrane); (iii) previous vitreoretinal surgery; (iv) significant media opacity; and (v) poor-quality OCT-A scans (signal strength index < 6/10) or scans with major artifacts/segmentation errors.

The study was conducted in accordance with the principles of the Declaration of Helsinki and complied with French data protection regulations (MR-004). All patients received appropriate information and did not oppose the use of their anonymized clinical data.

Measurements : Each patient underwent AL measurement using ZEISS® IOLMaster 700. OCT-A imaging was performed using the Optovue® system (3×3 mm scan, AngioAnalytics). Superficial and deep capillary plexus densities (SCP, DCP) were quantified, and the foveal avascular zone (FAZ) area was

measured. The nominal 3×3 mm retinal scan area is based on the linear distance across the retinal surface corresponding to a fixed angular field of view of 10°×10°, calculated using a reference AL of 23.82 mm, as defined by the RTVue® XR Avanti system. To account for ocular magnification differences due to variations in AL, FAZ area was corrected using the Littmann-Bennett formula.[10] Vessel density measurements were obtained using the device's built-in AngioAnalytics software (Optovue), based on automated segmentation of the superficial and deep capillary plexuses. No manual correction or external image processing (e.g., ImageJ/Fiji) was applied in order to preserve reproducibility and consistency.

Statistical analysis: A descriptive analysis of the study population was performed. The normality of quantitative variables was assessed using Shapiro-Wilk tests. Normally distributed data were expressed as means ± standard deviations (SDs), while non-normally distributed data were reported as medians with interquartile ranges (IQRs). To account for inter-eye correlations, generalized estimating equations (GEE) were used[11] with pairwise comparisons with Bonferroni correction within the GEE model for between-group comparisons. Additionally, GEE models were applied to evaluate the effect of DR stratification and diabetes type on the studied variables while adjusting for inter-eye dependencies. All statistical analyses were performed using R version 4.0.3 for Mac (2020).

Results

Patients

A total of 78 eyes from 46 patients were included in the study. These eyes were classified into three groups: 26 eyes from 15 patients with emmetropic diabetes (DB), 26 eyes from 15 patients with highly myopic diabetics (HMDB), and 26 eyes from 16 patients with highly myopic non-diabetics (HM). The demographic and clinical characteristics of the study population are summarized in Table 1. The three groups were comparable in terms of age, gender, diabetes duration, and diabetic retinopathy (DR) stage. There were no significant differences in axial length (AL) between the HM and HMDB groups. Additionally, no significant differences in AL were observed between patients with type 1 and type 2 diabetes within either group. Specifically, in the DB group, the mean AL was 23.3 ± 0.35 mm for type 1 (n = 4) and 23.3 ± 0.90 mm for type 2 (n = 22), with no significant difference (t(12) = 0.02, p = 1.00; 95% CI: −0.561 to 0.572). Similarly, in the HMDB group, the mean AL was 29.0 ± 0.64 mm for type 1 (n = 4) and 28.6 ± 1.31 mm for type 2 (n = 22), showing no significant difference (t(9) = 1.00, p = 0.30; 95% CI: −0.527 to 1.414). **Figure 1** shows representative OCT-A images for one individual from each group.

Univariate Between-Group Comparison

Univariate analyses confirmed that the data for all five key variables followed a parametric distribution, as verified by the Shapiro-Wilk test (p > 0.05). When comparing between groups, both the HMDB and HM groups exhibited significantly lower SCP_wh and SCP_pf values compared to the DB group. However, no significant differences were observed between the HMDB and HM groups in these parameters. Regarding DCP_wh and DCP_pf, the HM group showed significantly lower values than the DB group, but

the HMDB group did not differ significantly from the DB group in these parameters. Notably, the HMDB group demonstrated a significant increase in DCP_wh compared to the HM group. No significant differences were observed in FAZ area among the three groups (**Figure 2**). Post-hoc between-group comparisons are shown in **Table 2.**

Adjusted Models on Diabetes Type and DR Stages

Table 3 presents the results of the adjusted models using GEE to examine the effects of diabetes type and DR stage on vessel densities and FAZ area. A decrease in SCP was observed in the DB group with advancing DR stage, particularly in proliferative DR. Although not statistically significant, there was a trend toward decreased DCP in the DB group with increasing DR severity. In the HMDB group, SCP perfusion was higher in type 2 diabetes compared to type 1 diabetes. Conversely, in the DB group, DCP perfusion was higher in type 2 diabetes than in type 1. Of note, all individuals with type 1 diabetes were at stage 3 DR.

Discussion

This study evaluated retinal microvascular changes across three groups: patients with high myopia and diabetes (HMDB), patients with high myopia without diabetes (HM), and emmetropic patients with diabetes (DB). Our findings suggest that high myopia and diabetes exert distinct—and potentially interacting—effects on retinal microvasculature, with differential involvement of the superficial and deep capillary plexuses.

No significant differences were observed between the HM and HMDB groups in terms of SCP density or FAZ area. These findings suggest that axial elongation may play a predominant role in SCP alterations, potentially outweighing the additional impact of diabetes at this level. In contrast, DCP density was significantly higher in HMDB compared with HM, despite similar SCP profiles. This finding points toward a distinct influence of diabetes on the deeper retinal vascular layers in highly myopic eyes. One possible interpretation is that the increased DCP density observed in HMDB reflects adaptive microvascular changes in response to metabolic stress or localized hypoxia, or alternatively early microvascular remodeling associated with DR. We cannot distinguish between these two mechanisms based on the cross-sectional nature of our data; longitudinal studies are required to clarify this point. These results highlight a differential behavior of the two vascular plexuses and suggest a complex interaction between axial elongation and microvascular regulation.

We also observed that SCP density decreased with increasing DR severity, consistent with previous reports[12]. In contrast, within the HMDB group, DCP rather than SCP showed a decreasing trend with increasing disease severity. This pattern may suggest that in highly myopic diabetic eyes, the DCP is relatively preserved—or even may appear elevated—at earlier stages of DR, followed by a decline as disease progresses. Such a trajectory could reflect an initial compensatory phase, potentially involving capillary dilation or redistribution of flow, followed by progressive capillary dropout. However, this interpretation remains speculative and would require confirmation in longitudinal studies.

The relationship between HM and DR has been explored in epidemiological studies, which have reported a lower prevalence of DR in highly myopic eyes[6,7]. Several mechanisms have been proposed to explain this observation. Axial elongation may lead to stretching and thinning of retinal tissues, resulting in reduced retinal blood flow, as demonstrated in non-diabetic eyes[13–15] consistent with our findings of reduced SCP in the HM group. Reduced perfusion may in turn decrease capillary hydrostatic pressure, potentially limiting vascular leakage and microaneurysm formation. Additional hypotheses include dilution of inflammatory mediators within a larger ocular volume[16] and reduced metabolic demand associated with retinal thinning and neurodegenerative changes[7,17,18]. However, these mechanisms remain incompletely understood, and their impact on OCT-A–derived microvascular metrics is still uncertain[19–21].

Several limitations should be acknowledged. First, the retrospective design and relatively small sample size limit statistical power, particularly after subgroup stratification. No formal sample size calculation was performed, and the results should therefore be considered exploratory and hypothesis-generating. Second, all patients with type 1 diabetes in our cohort were classified as having stage 3 DR, precluding separation of the effects of diabetes type and disease severity. Consequently, some observed differences may reflect disease stage rather than intrinsic differences between diabetes types. Third, although FAZ area was corrected for ocular magnification using AL–based scaling, vessel density measurements were not adjusted for image size. Variations in AL can introduce magnification-related bias in OCT-A measurements, potentially affecting comparisons between myopic and non-myopic eyes. While axial length–based correction methods exist, their application to OCT-A vessel density measurements remains methodologically challenging and is not standardized across devices[22]. This limitation should therefore be considered when interpreting intergroup differences. Additional limitations include the absence of a healthy emmetropic control group, which restricts the ability to distinguish disease-specific changes from baseline physiological variation. Systemic factors such as glycemic control and duration of diabetes were not incorporated into the analysis and may have influenced retinal vascular parameters. Furthermore, although scans with poor signal strength were excluded, OCT-A acquisition in highly myopic eyes is technically challenging, and residual artifacts—including projection artifacts affecting DCP visualization—may have affected measurements. Finally, multiple vascular parameters were analyzed, increasing the risk of type I error despite Bonferroni adjustment within each outcome. These findings should therefore be interpreted within the context of these methodological constraints.

In conclusion, this study suggests that high myopia and diabetes may have distinct and layer-specific effects on retinal microvasculature, with a predominant influence of axial elongation on the superficial plexus and a more complex response in the deep plexus. These findings support the hypothesis that HM may modify microvascular alterations associated with diabetic retinopathy. However, larger prospective studies with appropriate axial length correction and standardized OCT-A analysis are required to confirm these observations and clarify the underlying mechanisms.

Declarations

- **Ethics approval and consent to participate**: The study was approved by the Ethics Committee of the French Society of Ophthalmology (00008855) and adhered to the tenets of the Declaration of Helsinki.
- **Consent to Participate:** All patients received appropriate information and provided oral informed consent prior to inclusion.
- **Availability of data and materials**: The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.
- **Competing interests**: The authors declare no competing interests.
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- **Authors' contributions**: M.K.: Conceptualization, Methodology, Investigation, Screening, Data curation, Writing the original draft, Writing – review & editing. A.V.J: Formal Analysis, Writing the original draft, Writing – review & editing. A.C. Conceptualization, Methodology, Investigation, Writing – review & editing, Supervision.

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References

1. Kropp M, Golubnitschaja O, Mazurakova A, et al. Diabetic retinopathy as the leading cause of blindness and early predictor of cascading complications—risks and mitigation. EPMA J. 2023;14(1):21–42. 10.1007/s13167-023-00314-8.
2. Aiello LP, Northrup JM, Keyt BA, Takagi H, Iwamoto MA. Hypoxic regulation of vascular endothelial growth factor in retinal cells. Arch Ophthalmol Chic Ill 1960. 1995;113(12):1538–44. 10.1001/archopht.1995.01100120068012.
3. Cheung N, Mitchell P, Wong TY. Diabetic retinopathy. Lancet. 2010;376(9735):124–36. 10.1016/S0140-6736(09)62124-3.
4. Pamulapati P, Das MK, Mohanty G. Assessing Preclinical Diabetic Retinopathy: The Role of Optical Coherence Tomography Angiography. Cureus. 2024;16(10):e72747. 10.7759/cureus.72747.
5. Waheed NK, Rosen RB, Jia Y, et al. Optical coherence tomography angiography in diabetic retinopathy. Prog Retin Eye Res. 2023;97:101206. 10.1016/j.preteyeres.2023.101206.
6. Man REK, Sasongko MB, Wang JJ, Lamoureux EL. Association between myopia and diabetic retinopathy: a review of observational findings and potential mechanisms. Clin Exp Ophthalmol. 2013;41(3):293–301. 10.1111/j.1442-9071.2012.02872.x.
7. Lim LS, Lamoureux E, Saw SM, Tay WT, Mitchell P, Wong TY. Are Myopic Eyes Less Likely to Have Diabetic Retinopathy? Ophthalmology. 2010;117(3):524–30. 10.1016/j.ophtha.2009.07.044.

8. Al-Sheikh M, Phasukkijwatana N, Dolz-Marco R, et al. Quantitative OCT Angiography of the Retinal Microvasculature and the Choriocapillaris in Myopic Eyes. Invest Ophthalmol Vis Sci. 2017;58(4):2063–9. 10.1167/iovs.16-21289.
9. Su R, Jia Z, Fan F, Li J, Li K. Clinical Observation of Macular Vessel Density in Type 2 Diabetics with High Myopia. Ophthalmic Res. 2023;66(1):124–30. 10.1159/000526487.
10. Bennett AG, Rudnicka AR, Edgar DF. Improvements on Littmann's method of determining the size of retinal features by fundus photography. Graefes Arch Clin Exp Ophthalmol Albrecht Von Graefes Arch Klin Exp Ophthalmol. 1994;232(6):361–7. 10.1007/BF00175988.
11. Zeger SL, Liang KY. Longitudinal data analysis for discrete and continuous outcomes. Biometrics. 1986;42(1):121–30.
12. Durbin MK, An L, Shemonski ND, et al. Quantification of Retinal Microvascular Density in Optical Coherence Tomographic Angiography Images in Diabetic Retinopathy. JAMA Ophthalmol. 2017;135(4):370–6. 10.1001/jamaophthalmol.2017.0080.
13. Benavente-Pérez A, Hosking SL, Logan NS, Broadway DC. Ocular blood flow measurements in healthy human myopic eyes. Graefes Arch Clin Exp Ophthalmol Albrecht Von Graefes Arch Klin Exp Ophthalmol. 2010;248(11):1587–94. 10.1007/s00417-010-1407-9.
14. Berisha F, Findl O, Lasta M, Kiss B, Schmetterer L. A study comparing ocular pressure pulse and ocular fundus pulse in dependence of axial eye length and ocular volume. Acta Ophthalmol (Copenh). 2010;88(7):766–72. 10.1111/j.1755-3768.2009.01577.x.
15. Shimada N, Ohno-Matsui K, Harino S, et al. Reduction of retinal blood flow in high myopia. Graefes Arch Clin Exp Ophthalmol Albrecht Von Graefes Arch Klin Exp Ophthalmol. 2004;242(4):284–8. 10.1007/s00417-003-0836-0.
16. Xie XW, Xu L, Wang YX, Jonas JB. Prevalence and associated factors of diabetic retinopathy. The Beijing Eye Study 2006. Graefes Arch Clin Exp Ophthalmol. 2008;246(11):1519–26. 10.1007/s00417-008-0884-6.
17. Luu CD, Lau AMI, Lee SY. Multifocal electroretinogram in adults and children with myopia. Arch Ophthalmol Chic Ill 1960. 2006;124(3):328–34. 10.1001/archopht.124.3.328.
18. Wolsley CJ, Saunders KJ, Silvestri G, Anderson RS. Investigation of changes in the myopic retina using multifocal electroretinograms, optical coherence tomography and peripheral resolution acuity. Vis Res. 2008;48(14):1554–61. 10.1016/j.visres.2008.04.013.
19. Yamamoto N, Itonaga K, Marunouchi T, Majima K. Concentration of transforming growth factor beta2 in aqueous humor. Ophthalmic Res. 2005;37(1):29–33. 10.1159/000083019.
20. Chua J, Vania M, Cheung CMG, et al. Expression profile of inflammatory cytokines in aqueous from glaucomatous eyes. Mol Vis. 2012;18:431–8.
21. Jonas JB, Tao Y, Neumaier M, Findeisen P. VEGF and refractive error. Ophthalmology. 2010;117(11):2234e1. 10.1016/j.ophtha.2009.12.006.
22. Sampson DM, Gong P, An D, et al. Axial Length Variation Impacts on Superficial Retinal Vessel Density and Foveal Avascular Zone Area Measurements Using Optical Coherence Tomography

Angiography. Invest Ophthalmol Vis Sci. 2017;58(7):3065–72. 10.1167/iovs.17-21551.

Tables

Table 1 – Characteristics of Included Patients

	HMDB	DB	HM
N eyes (N patients)	26 (15)	26 (15)	26 (16)
Gender F (%)	62%	62%	62%
Age	66.4 ± 12.9	65.4 ± 12.2	66.6 ± 12.3
AL (mm)	28.3 ± 1.75	23,3 ± 0.84	29.4 ± 1.51
Diabetes duration (y)	25	22	
Diabetes type and DR stage			-
Type 1	4 4 stage 3	4 4 stage 3	-
Type 2	22 10 stage 0-1 4 stage 2 8 stage 3	22 10 stage 0-1 4 stage 2 8 stage 3	-
Quality Index (/10)	7.2 ± 1	7.5 ± 0.6	7.3 ± 0.7

Table 2 – Between-Group Comparisons

Analyses were performed using gerealized estimating quations (GEE) to account for inter-eye correlations and pairwise comparisons with Bonferroni correction were used for between-Group Comparisons.

	Mean ± SD			p-value		
	HMDB	**DB**	**HM**	**HMDB vs DB**	**HMDB vs HM**	**DB vs HM**
SCP_wh	38.1 ± 5.3	43.9 ± 4.3	37.4 ± 4.6	**0.0008**	1.0	**0.0001**
SCP_pf	39.7 ± 5.6	45.9 ± 4.4	39.8 ± 4.4	**0.003**	1.0	**0.0001**
DCP_wh	45.8 ± 5.8	46.9 ± 2.7	43.0 ± 4.7	1.0	0.2	**0.0008**
DCP_pf	47.0 ± 6.9	48.4 ± 3.1	45.0 ± 5.0	1.0	0.8	**0.01**
FAZ	0.31 ± 0.14	0.30 ± 0.12	0.32 ± 0.11	1.0	1.0	1.0

Table 3 – Results for the adjusted models considering the effect of the group, the diabetes type and the diabetic retinopathy stage as predictors. Patients with diabetes without high myopia (DB) serve as the reference group. The reference diabetes type is Type 1, and the reference diabetic retinopathy (DR) stage is Stage 1. Generalized Estimating Equations (GEE) were applied. Note: Stage 3 DR was observed exclusively in patients with Type 1 diabetes.

	Diabetes type		DR Stage 2		DR Stage 3	
	Estimate ± SD	**p**	**Estimate ± SD**	**p**	**Estimate ± SD**	**p**
HMDB						
SCP_wh	7.94 ± 5.2	0.127	-1.49 ± 2.39	0.534	1.58 ± 2.6	0.544
DCP_wh	0.3 ± 3.76	0.936	-4.44 ± 2.34	*0.058*	-4.07 ± 2.96	0.17
SCP_pf	9.42 ± 3.87	**0.015**	-2.05 ± 2.17	0.345	0.88 ± 3	0.771
DCP_pf	4.66 ± 2.91	0.11	-5.28 ± 2.9	*0.069*	-5.57 ± 3.43	0.105
FAZ	-0.07 ± 0.09	0.44	0.04 ± 0.05	0.444	0.06 ± 0.09	0.525
DB						
SCP_wh	-5.27 ± 4.11	0.199	-1.78 ± 2.1	0.395	-5.17 ± 1.72	**0.003**
DCP_wh	-1.24 ± 2.07	0.549	1.11 ± 0.85	0.19	-1.55 ± 1.56	0.32
SCP_pf	-5.26 ± 3.96	0.184	-1.83 ± 1.79	0.308	-5.62 ± 1.98	**0.005**
DCP_pf	2.74 ± 1.36	**0.045**	-0.46 ± 1.31	0.726	-0.97 ± 1.71	0.571
FAZ	-0.01 ± 0.05	0.86	-0.07 ± 0.05	0.188	0.03 ± 0.07	0.594

Figures

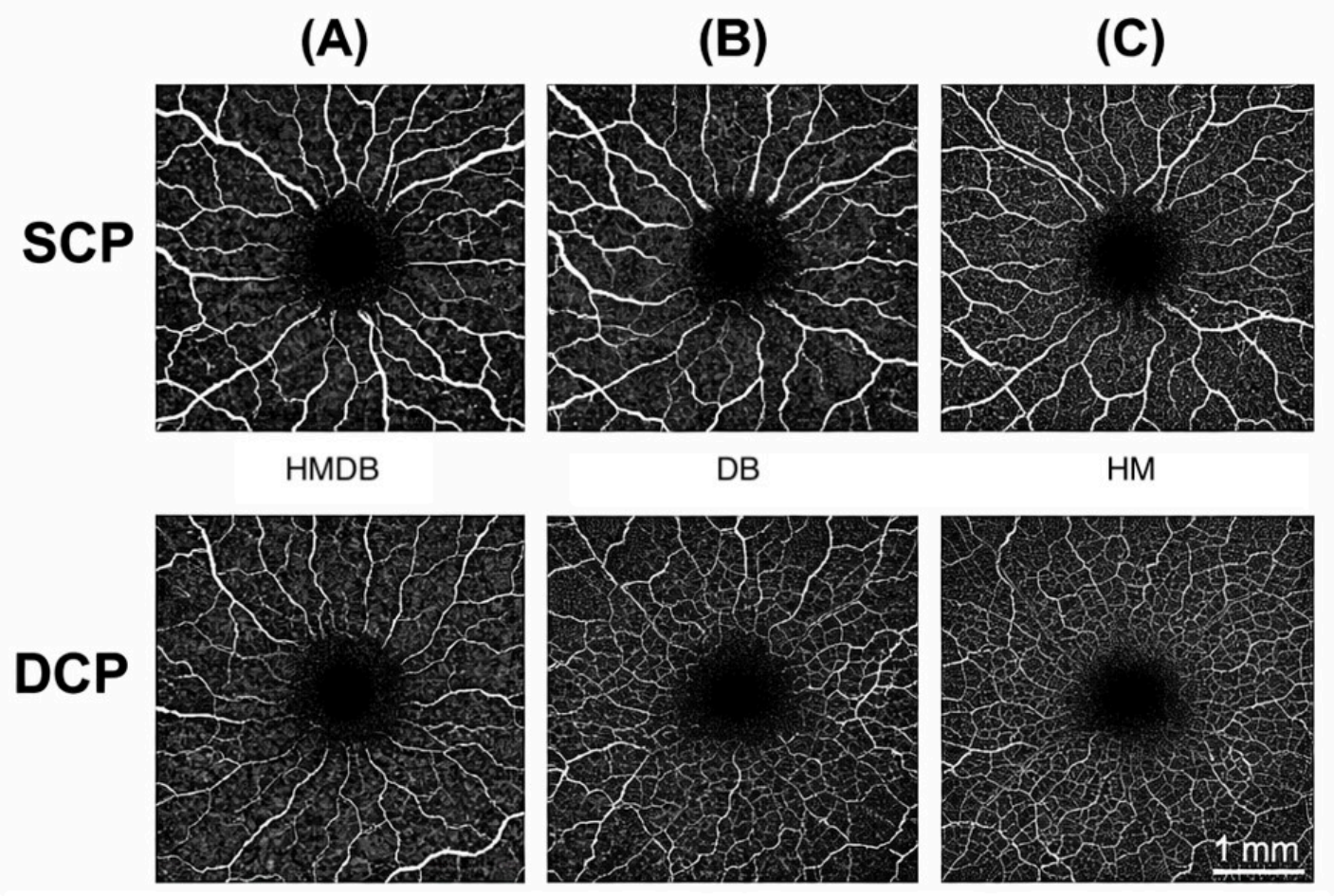


Figure 1

Representative 3×3 mm OCT-A images of the superficial capillary plexus (SCP, upper row) and deep capillary plexus (DCP, lower row) for one individual from each study group. (A) Highly myopic diabetic

patient (HMDB group). (B) Emmetropic diabetic patient (DB group). (C) Highly myopic non-diabetic patient (HM group). Scale bar = 1 mm. All images acquired using the Optovue® system (AngioAnalytics software).

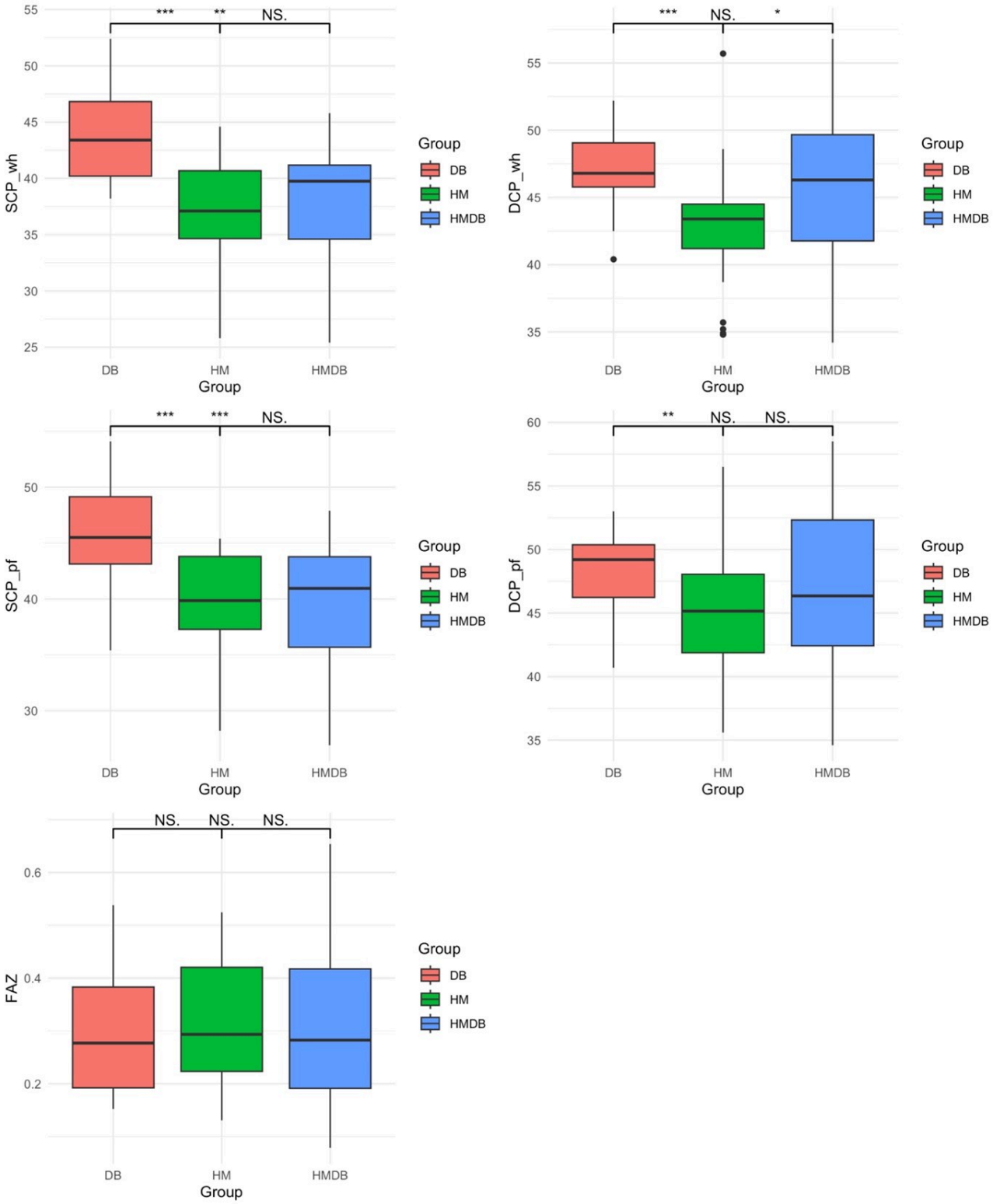


Figure 2

Box plots illustrating intergroup comparisons for the vascular density and the foveal avascular zone.