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ORIGINAL RESEARCH

The Correlation Between Macular Vessel Density and Its Clinical Parameters in Diabetes Mellitus Type 2

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Purpose: To evaluate macular vessel density using clinical parameters in patients with type 2 diabetes mellitus (DM) without retinopathy.

Patients and Methods: This cross-sectional study enrolled 32 participants (63 eyes) aged 40–60 years who met the inclusion criteria. Group 1 included 32 eyes of type 2 DM, whereas the rest had no DM. Ophthalmic examination was performed. Macular vessel density was measured using optical coherence tomography angiography (OCTA) with a 6 × 6 mm scan. Macular vessel density is correlated with patient age, HbA1c values, disease duration, and contrast sensitivity. The data were analyzed using Spearman's rank correlation and an independent *t*-test.

Results: Central (foveal area) macular vessel density in the diabetic group (5.36 ± 2.87) was significantly lower than that in healthy participants (7.82 ± 3.05) (p = 0.002). The whole macular vessel density (foveal and parafoveal areas) was also lower in the DM group than in the healthy subjects (15.76 ± 3.38 vs 17.18 ± 1.92) but the difference was not statistically significant (p = 0.070). The age of DM patients has correlated with central (r = −0.522; p = 0.002) and whole macular vessel density (r = −0.369; p = 0.038). HbA1c levels, diabetes duration, and contrast sensitivity were not correlated with macular vessel density.

Conclusion: Macular vessel density was lower in DM patients without retinopathy than in healthy subjects. In patients with DM, macular vessel density decreases with increasing age. We failed to find a correlation between macular vessel density and the HbA1c value, disease duration, or contrast sensitivity.

Keywords: foveal density, parafoveal density, optical coherence tomography angiography, diabetes mellitus type 2

Introduction

Data collected by the International Diabetes Federation (IDF) show more than one billion people worldwide are living with diabetes. The global prevalence of diabetes will rise to 783.2 million (12.2%) in 2045.[1] Diabetics' condition can cause complications in the retina through several biochemical pathways, thus damaging the retinal microvascular and neurovascular unit.[2–5] Retina, especially in the macular area, has high metabolic and oxygen demands, and is very susceptible to microvascular damage. Macular disorders cause visual impairment, including visual acuity and contrast sensitivity.[5,6] Many studies have found that damage to the retinal microvascular and neurovascular units already exists before clinical signs of retinopathy are observed.[4,7,8] In hyperglycemic conditions, macular perfusion is altered. Decreased retinal blood flow has been reported in patients with diabetes.[3,5,7,8]

After the discovery of Optical Coherence Tomography Angiography (OCTA), visualization of retinal capillaries and perfusion status of the retinal vasculature has become easier to evaluate. Retinal vessel density, defined as the proportion of blood vessel area showing blood flow relative to the total area, can be measured.[8–11] Decreasing perifovea capillary

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blood flow, seen in changes in the morphological area of the fovea avascular zone (FAZ) and macular vessel density, may reflect macular ischemia, which is a risk factor for diabetic retinopathy progression.[12,13]

The duration of diabetes and uncontrolled diabetes are also risk factors for the progression of diabetic retinopathy. Several previous studies have correlated glycemic control in diabetic conditions where retinopathy has not yet occurred with macular vessel density, but the results are conflicting.[14–17]

Understanding the correlation between macular vessel density and clinical parameters is important because it could explain the pathophysiology of diabetic eye disorders and provide information about management strategies, including the prevention of normal retinal structure and visual function. The detection of early microvascular damage in patients with diabetes is also important for predicting the progression of diabetic retinopathy. Therefore, this study aimed to evaluate macular vessel density using certain clinical parameters in patients with type 2 diabetes mellitus (DM) without retinopathy using OCTA.

Materials and Methods

Participants

This cross-sectional study was conducted on outpatients diagnosed with DM type 2 at the Endocrinology Department Saiful Anwar General Hospital and was approved by the Institutional Ethics Committee of the General Hospital Saiful Anwar Malang, Indonesia (400/107/K3/302/2021). This study was conducted in compliance with the principles and practice of the Declaration of Helsinki.

It enrolled 32 participants (63 eyes) between 40 and 60 years of age, divided into two groups. Group 1 included 16 patients with diabetes mellitus who met the inclusion criteria (32 eyes). The inclusion criteria were type 2 DM without retinopathy (examined by an ophthalmologist) with HbA1c > 6.5% (diagnosed diabetes by an endocrinologist), who were 40–60 years old, and best-corrected visual acuity better than 6/18. History of neuritis, glaucoma, multiple sclerosis, hypertension, epilepsy, ocular trauma, and consumption of ethambutol, levodopa, and antiepileptic drugs were not included in this study. Patients with optic neuropathy were excluded from the study.

Group 2 was normal control group consisting of 16 healthy staff members and volunteers without diabetes or other ophthalmological disorders (31 eyes). The other criteria were the same as those used for inclusion. One eye of normal control group was excluded because there was an error occurred in capturing the image. All participants provided written informed consent to participate in this study.

Ocular Examination

An ophthalmologist performed all ophthalmic examinations. Visual acuity was examined using an ETDRS chart. The LEA NUMBER low-contrast flip chart was used for contrast sensitivity (CS) examination. Contrast sensitivity decreases when the results value is more than 1.25%. All pupils were dilated using mydriatyl 0.5% eye drops (Cendo), and all funduscopic examinations were performed using indirect slit-lamp biomicroscopy with a 90D lens (Volk).

Optical Coherence Tomography Angiography Imaging

Macular vessel density was measured by one operator using optical coherence tomography angiography (OCTA) with Cirrus 6000 OCT-Angiography scans (Zeiss OCT, Carl Zeiss Meditec, Jena, Germany) with 6 × 6 mm foveal-centered scan patterns at an acquisition frequency of 100,000 A-scans per second, wavelength centered at 840 nm, and axial and transverse resolutions of 5 and 12 µm, respectively. Macular vessel density divided into central (foveal area) and whole macular vessel density (foveal and parafoveal areas or full area in ETDRS region). The measurements are read by single person based on OCTA analysis.

Macular vessel density is correlated with patient age, HbA1c values, disease duration, and contrast sensitivity.

Statistical Analysis

Before the data were analyzed, the normality test for each group was performed using the Kolmogorov–Smirnov test. The data were analyzed using Spearman's rank correlation and an independent *t*-test. Statistical analyses were performed using SPSS21 for windows. Statistical significance was set at $p < 0.05$.

Results

This study included 32 participants (63 eyes); 32 had diabetic eyes and the rest had normal and healthy eyes. The mean visual acuity in the diabetic and control groups was similar. The mean diabetes duration was <5 years. Age, HbA1c levels and contrast sensitivity in the diabetic and control groups were significantly different ($p < 0.05$). The distribution of participants' basic characteristics is presented in Table 1.

Our study focused on the correlation between macular vessel density (central and whole macular vessel density) and the risk factors for diabetic retinopathy. We measured the central macular vessel density (fovea avascular zone area) and whole macular vessel density of each eye, as shown in Figure 1. Before analyzing the data, we assessed the differences in central macular vessel density and whole macular vessel density (full foveal and parafoveal areas) between non-diabetic and diabetic participants (Figures 2 and 3). Macular vessels were denser in the eyes of non-diabetic patients than in diabetic patients, whether in the central or whole vessels, but the result was significant only for central macular vessel density ($p = 0.002$).

We analyzed the correlation between macular vessel density (central and whole areas) and the risk of diabetic retinopathy (HbA1c and duration of diabetes), clinical characteristics (age of the patients), and contrast sensitivity as a clinical parameter. No correlation was found between the HbA1c level with central macular vessel density. We found correlation between HbA1c with whole macular vessel density, but the correlation is positive that means the higher HbA1c, the denser macular vessel density (Figure 4). This result is contrary to the hypothesis. There is no correlation between duration of diabetes with macular vessel density (Figure 5).

Comparing the age of patients in both the DM and non-DM groups correlated with macular vessel density, we found a significant correlation between both areas (central and whole vessel density), ($p = 0.002$ vs $p = 0.038$) in DM patients (Figure 6).

For the clinical parameters, we measured the contrast sensitivity and related macular vessel density and found no significant correlation in either area of the macular vessel density (Figure 7).

Table 1 Characteristics of Study Participants

Variable	Group	N	Mean	P-value
Age	DM	16	52.88 ± 6.5	0.001*
	Normal	16	47.13 ± 5.8	
DM duration	DM	16	4.9 ± 2.1	
HbA1c	DM	16	8.03 ± 1.0	0.000*
	Normal	16	5.69 ± 0.5	
Visual acuity	DM	16	0.18 ± 0.1	0.389
	Normal	16	0.13 ± 0.2	
Contrast sensitivity	DM	16	1.99 ± 0.6	0.000*
	Normal	16	1.41 ± 0.4	

Notes: The asterisk (*) denotes the significant difference, as indicated by P < 0.05. Statistically significant at P < 0.05.

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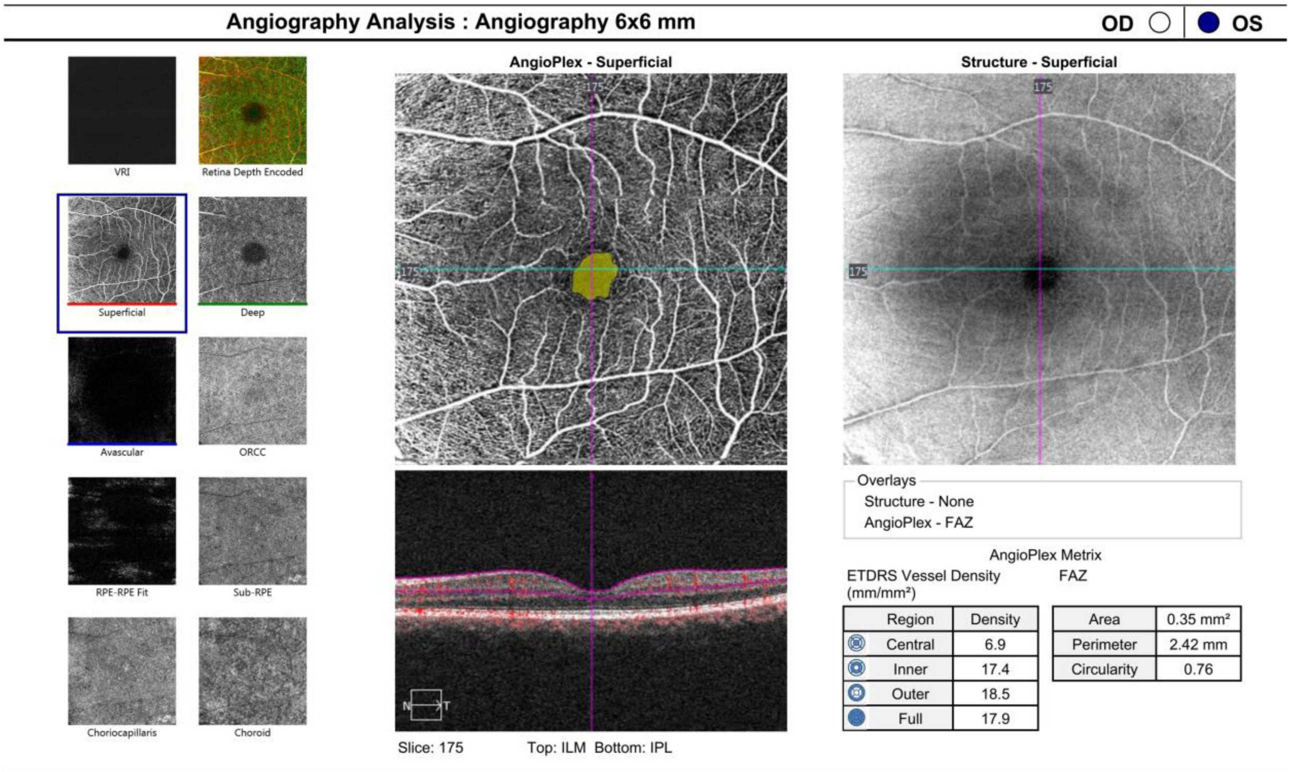

Figure 1 Left eye OCTA scan of 59 year-old DM patients with measures of superficial central macular vessel density, and whole (full) macular vessel density in mm/mm2.

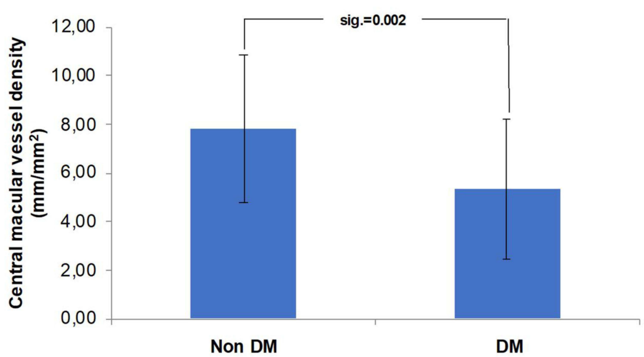

Figure 2 Central macular vessel density in DM significantly lower than non DM (p = 0.002).

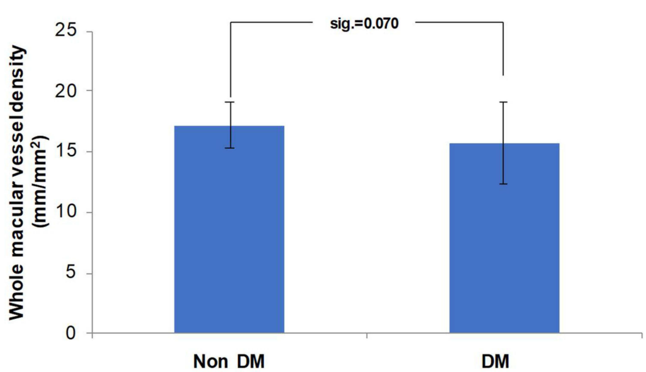

Figure 3 Whole macular vessel density in DM is lower than non DM, but not significant (p = 0.070).

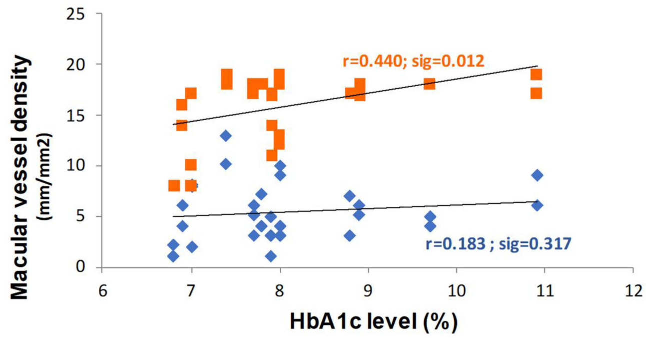

Figure 4 No correlation between central macular vessel density (diamond shape) (p = 0.317) with HbA1c. There is significant positive correlation between whole macular vessel density (Orange square) (p = 0.012) with HbA1c, but the correlation does not correspond to the hypothesis.

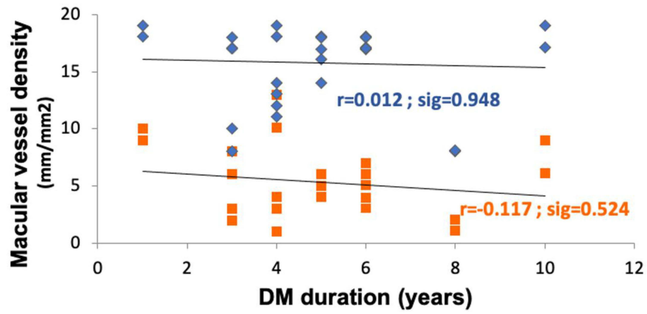

Figure 5 No correlation between central (Orange square) (p = 0.524) and whole macular vessel density (diamond shape) (p = 0.948) with DM duration.

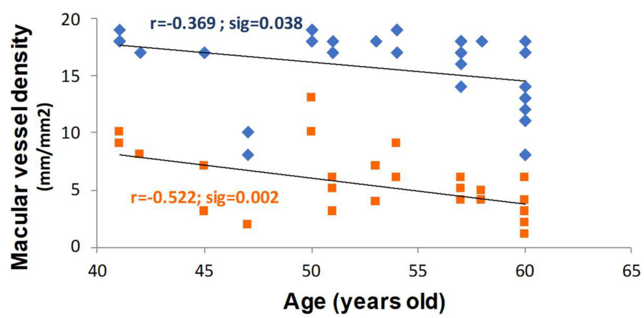

Figure 6 There is significant correlation between age of the DM patients and central macular vessel density (Orange square) (p = 0.002), and with whole macular vessel density (diamond shape)(p = 0.038).

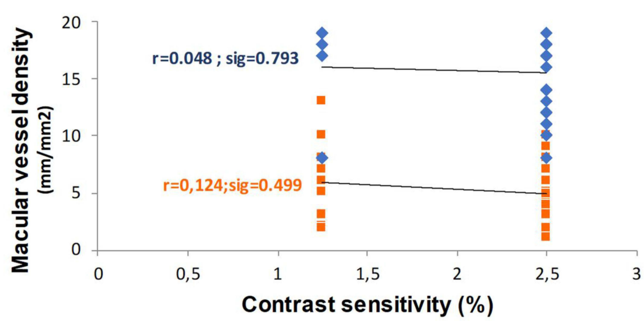

Figure 7 There is no correlation whether between central macular vessel density (Orange square) (p = 0.499), whole macular vessel density (diamond shape) (0.793), and contrast sensitivity.

Discussion

The first funduscopic finding of diabetic retinopathy was the presence of a microaneurysm. It is a clinical sign of microangiopathy, a microvascular complication of diabetic retinopathy. Recent studies have found that before the clinical signs of diabetic retinopathy are observed, there are changes in the microvascular capillary structure, including the fovea avascular zone and macular vessel density, and degeneration of the neurovascular unit of the retina.

In this study, we analyzed the parameters of diabetic patients without retinopathy, including HbA1c level, duration of diabetes, age, and contrast sensitivity, correlated with macular vessel density, using OCTA. Ocular Coherence Tomography Angiography can be used to identify retinal microvascular changes in a noninvasive and rapid manner. Our results showed that the central macular vessel density in diabetic patients was lower than that in non-diabetic patients. These findings are consistent with the results of other studies that show that there is foveal avascular zone enlargement and a decrease in macular vessel density in diabetic patients compared to non-diabetic patients, even before the clinical signs of diabetic retinopathy appear.[14,18–20] The same result was obtained by counting the foveal and parafoveal areas (whole vascular density) in diabetic patients, but the difference was not statistically significant. This could be because the central area is more sensitive to changes in hyperglycemia because the central area or foveal avascular zone (FAZ) is vascularized by choroidal vessels. According to a previous study, choroidal vessels are more susceptible to hyperglycemia-induced ischemic conditions. The choroid provides nutrition and oxygen to the outer layer of the retina and retinal pigment epithelium, so choroids reflect the metabolic state of the choroid and retina itself.[7,21] It is consistent with a previous study that showed that choroidal perfusion in diabetes is low and there is a decrease in choroidal thickness in diabetic patients, although it is still unclear whether the thinning of the choroid is primary or secondary to retinal ischemia.[10,22–25] The mechanism of vessel density loss is also based on retinal ischemia caused by chronic hyperglycemia causing an imbalance in the biochemical pathway rather than cellular changes, breakdown of the blood-retinal barrier, pericyte loss, endothelial dysfunction, red blood cell changes, basement membrane thickening, and increasing platelet stickiness.[2,3] All of these pathologies represent low perfusion and decreased vessel density on OCTA.

The age of the diabetic patients in this study was correlated with macular vessel density. These results are consistent with those of a previous study that reported that FAZ circularity significantly decreased after 40 years of age, but not in healthy patients.[13,26,27] The reduction in blood flow in older patients with and without diabetes could be due to oxidative stress that can occur in aging patients. Diabetes also accelerates aging. Therefore, the results of this study could be attributed to oxidative stress in patients with diabetes and aging. However, the results of these studies have been conflicting. The architecture of the FAZ is related to hormones, ethnicity, and imaging techniques.[11,26]

A previous study correlated vessel density with glycemic control. There were significant differences between lower vessel density in patients with diabetes, even with intensive control (HbA1c $< 7\%$) and no diabetes. Choriocapillary flow deficit is correlated with HbA1c levels shown in OCTA.[14] Oltea (2024) and Wysocka Mincewicz (2020) also showed that higher levels of HbA1c corresponded to larger FAZ areas and decreased foveal vessel density. HbA1c represents glycemic control. Uncontrolled hyperglycemia in patients with DM increases metabolic demand and leads to hypoxia rather than ischemia.

In contrast to other studies, this study failed to show a correlation between HBA1c and macular vessel density.[13,15,20,28] The reason for the differences could be that this study only measured HbA1c at a single visit; therefore, it does not represent the glycemic control of the patient. Similar to our results, a study by Golebiewska (2017) showed no statistically significant difference between the FAZ area and vessel density of the superficial and deep capillary plexuses with HbA1c levels in children. However, the subjects of this study were patients with DM type 1, so the autoregulation and retinal circulation state may still be better compared to adult patients with DM type 2.[29] Shaw et al also showed no significant association between HbA1c levels in OCTA measurements. This may be because some eyes respond well to glycemic control changes, whereas others are less affected or more tolerant to these changes.[30]

The loss of capillary vessels is caused by chronic or prolonged exposure to hyperglycemia. It induces capillary wall damage, such as pericyte loss, microvascular occlusion, endothelial damage, and breakdown of the blood ocular barrier. The loss of capillaries leads to a decrease in the central macular vessel density. However, in this study, no correlation was found between macular vessel density and the duration of diabetes. This may be because all patients with diabetes in this study had diabetes for less than five years (mean duration, 4.9 years). Several studies have shown similar results, even when the duration of DM is divided into less than 5 years and more than 5 years.[15,29,30]

The contrast sensitvity test results were lower in patients with diabetes than in healthy patients. This correlated with other study findings that the retinal nerve fiber layer had already decreased in diabetic patients without retinopathy, showing that neurodegeneration occurred before the appearance of clinical signs of diabetic retinopathy. However, the results of this study showed that there was no correlation between central and whole macular vessel density and CS. This is because a decrease in contrast sensitivity is most likely related to ganglion cells or neurodegeneration of the retina, but not vascular density.[31,32] Another study measuring contrast sensitivity in diabetic patients with and without retinopathy also showed a decrease in contrast sensitivity, but the mean duration in the population with diabetes was more than 10 years compared to this study, which was no more than 5 years.[33]

The strength of our study is that few studies have examined the sensitivity related to macular vessel density in diabetic patients without retinopathy. Several limitations of the current study include the following: we only measured the superficial vascular density on OCTA; therefore, the deep vascular density should be measured, because previous studies have shown that deep retinal vessels are preferentially affected in DM patients compared to superficial layers that are less vulnerable to ischemic change and are usually damaged after the onset of diabetic retinopathy.[6] The number of participants was relatively small because we had to fulfil the inclusion criteria in the tertiary hospital setting (not many patients with diabetes do not have other pathological findings). We only measured HbA1c levels at a single visit, whereas another study calculated four measurements per year.[14,15,28,29] Therefore, we hope that health-care workers not only rely on optimizing HbA1c levels but also control the stability of glycemic levels in diabetes management to prevent further retinal vascular damage.

Conclusion

In conclusion, this study showed that the central and whole areas of macular vessel density are lower in patients with diabetes, even in those without retinopathy. It also decreases with increasing age but has no correlation with HbA1c level, disease duration, and contrast sensitivity.

These data may help us to understand the pathology of diabetic retinopathy, even though retinopathy has not yet occurred. These findings also suggest that the central macular area can be used as a marker because it is sensitive to hyperglycemic changes and is susceptible to ischemic conditions. In the future, it will not only be used to prove a pathological and mechanism concept but also can be used for early therapy or intervention.

Disclosure

The authors declare that they have no conflicts of interest in this work.

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