ORIGINAL ARTICLE

Association Between Choroidal Thickness and Glycaemic Control in Type 2 Diabetes Without Retinopathy

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Abstract:

Objectives: To evaluate choroidal thickness in patients with type 2 diabetes without retinopathy, comparing it to nondiabetic individuals and investigating the relationship between choroidal thickness and HbA1c levels.

Methods: A prospective, cross-sectional analytical study was conducted at the ophthalmology department of Fauji Foundation Hospital, Rawalpindi. The study included 110 diabetic patients without retinopathy and 55 age- and sex-matched nondiabetic controls. Choroidal thickness was measured at five locations (subfoveal, 1500 µm and 3000 µm nasal, and 1500 µm and 3000 µm temporal) using spectral domain-optical coherence tomography (SD-OCT). Sub-foveal choroidal thickness at 1500 µm nasal, 3000 µm nasal, followed by 1500 µm temporal, and 3000 µm temporal, followed by central macular thickness, was taken. Correlation analysis between HbA1c levels and choroidal thickness in diabetics was performed using SPSS version 24.

Results: Diabetic participants demonstrated significantly reduced choroidal thickness in subfoveal and nasal regions compared to nondiabetics ($p < 0.01$). No significant difference was found in temporal regions or central macular thickness. A strong negative correlation was observed between HbA1c levels and choroidal thickness ($R = -0.783$), whereas disease duration showed minimal association ($R < 0.1$).

Conclusion: The findings suggest that elevated HbA1c levels adversely impact choroidal thickness in diabetics, even in the absence of diabetic retinopathy. This highlights the potential role of choroidal alterations in the early stages of diabetic ocular changes. Further longitudinal studies are warranted to explore the prognostic significance of choroidal thickness in diabetic retinopathy progression. *Al-Shifa Journal of Ophthalmology 2025; 21(3): 184-191. © Al-Shifa Trust Eye Hospital, Rawalpindi, Pakistan.*

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Introduction:

Diabetes is the leading cause of visual impairment in middle age individuals and is also a major cause of diabetic retinopathy (DR) [1, 2]. AS reported in literaure, the diabetic retinopathy (DR) is caused by the destruction of the blood-retinal barrier due to disruption in the structure of the retinal vascular wall [3,4]. The choroid being a five layered structure is located between the retina and sclera ; its inner layer has abundant interstices for the metabolism of the retina. Situated between the retina and sclera, the choroid comprises five discrete structural layers; the innermost of these layers is extensively fenestrated, facilitating metabolic exchange with the

retina. Choroidal lobular capillaries expand into a fan-shaped network, interlinking to modulate the hydraulic resistance and regulate blood flow within discrete lobules, thereby safeguarding overall retinal oxygenation. 5. It ultimately nourishes the outer retina, the layer of retinal pigment epithelium, and the photoreceptor cells. While the pathogenesis of DR is still not fully explained, studies with a possible role of choroidal alteration in the presence and progression of DR are available in the literature [6]. Abnormalities documented in choroid of diabetic patients include delayed vascular filling and photodynamic reduction of choroidal perfusion [7], diminished flow [8], and structural malformations such as luminal narrowing , choroidal aneurysms, neovascular membranes and regions of capillary nonperfusion. Choroid has been evaluated utilizing various modalities, e.g., indocyanine green angiography, laser Doppler flowmetry, and ultrasonography. The utility of optical coherence tomography (OCT) has facilitated as a non-invasive mode of evaluating posterior segment of eye. [9]Particularly, the addition of enhanced depth imaging (EDI) to OCT (Spectralis Heidelberg Engineering, Germany (SD-OCT) has made it possible to obtain high-resolution images of the retina and choroid [10,] 11. Evaluating the choroid is challenging because of a wide range of influencing factors responsible for varying information pertinent to the choroid in various diseases [12]. Choroidal thickness otherwise has prognostic significance for diagnosis and follow-up of various retinal and choroidal diseases [13].

In diabetes, chronically raised HbA1c levels are well known to influence ocular health, while some evidence also suggests that short-term spikes in blood glucose can affect the choroid.[14,15] The reported changes in choroidal thickness among diabetic patients remain inconsistent, largely because this parameter is influenced by a variety of systemic and local determinants.[16,17] These include patient age, fluctuations during the day, axial length, refractive status, intraocular pressure, and ocular perfusion pressure. Previous intraocular treatments—such as anti-VEGF therapy, corticosteroid injections, or laser procedures—may also alter choroidal measurements. In the diabetic population, factors like fasting glucose levels, long-term glycaemic control, and associated kidney disease have been linked to such alterations.

The purpose of the present study was to compare choroidal thickness in individuals with type 2 diabetes but without retinopathy to that of non-diabetic controls, using enhanced depth imaging spectral domain OCT (EDI SD-OCT) [18] Within the diabetic cohort, we further explored how choroidal thickness correlates with HbA1c levels. Establishing such a baseline may provide a reliable reference point for future work on proliferative and non-proliferative diabetic retinopathy.

Methodology:

This prospective, cross-sectional analytical study was conducted in the Department of Ophthalmology at Fauji Foundation Hospital (FFH), Rawalpindi, over a period of six months following ethical clearance (approval letter no. 842/RC/FFH/RWP, dated 01 January 2024). Written informed consent was obtained from all participants prior to enrolment.

The required sample size was estimated using the WHO sample size calculator, based on a 5% significance level, 90%

study power, and an expected mean central choroidal thickness of 247.8 ± 39.74 μm in diabetic patients.[19] This yielded a minimum of 109 diabetic participants. For comparison, a control group was recruited in a 2:1 ratio, comprising 55 non-diabetic volunteers from the refractive clinic.

A non-probability purposive sampling approach was adopted. Diabetic participants were included if they were between 18 and 70 years of age, had type 2 diabetes mellitus confirmed by a physician (HbA1c > 6.5% or fasting plasma glucose > 126 mg/dl), and met other eligibility criteria. Controls were recruited from the general ophthalmology clinic, provided they were ≥18 years old with no clinical diagnosis of diabetes. Patients were excluded if they had diabetic retinopathy (DR), diabetic macular edema (DME), a history of intraocular surgery (including cataract), intraocular injections (steroids or anti-VEGF), or laser photocoagulation. Exclusion also applied to pregnancy/puerperium, significant media opacities compromising OCT image quality, refractive error >5 dioptres, or axial length >25 mm. Non-diabetic candidates with HbA1c values >6.5% were reclassified into the diabetic cohort.

Diabetic participants were referred from medical OPDs for DR screening, while non-diabetic controls were enrolled from the refractive clinic. One eye was randomly selected per individual. After initial screening, each participant underwent a detailed assessment including history, visual acuity testing, slit-lamp examination, tonometry, indirect ophthalmoscopy, and dilated fundus evaluation. Best-corrected visual acuity (BCVA) was determined using Snellen's chart. Intraocular pressure was measured with Goldman's applanation tonometer before pharmacological dilation, after which the fundus was examined with a Volk 90D lens.

Participants were scheduled the following morning for fasting blood sugar and HbA1c testing, along with blood pressure measurement. Mean arterial pressure (MAP) was derived using the formula:

MAP=Diastolic+⅓(Systolic–Diastolic).

Patients continued their routine medications but were advised to avoid heavy, sugar-rich drinks before testing.Choroidal imaging was performed with the SOCT Copernicus REVO80® SD-OCT system (Optopol Technology, version 10.0.1, Poland; 840 nm diode source, 12 μm transverse resolution, 5 μm axial resolution, 80,000 A-scans/sec), using enhanced depth imaging (EDI) mode. All scans were obtained by the principal investigator (Naz A) and verified by a senior ophthalmologist (Intesar Ul Haq R). Examinations were scheduled between 12 pm and 2 pm to minimize diurnal variations. Choroidal thickness was measured from the hyperreflective outer border of the retinal pigment epithelium to the hyporeflective sclero-choroidal junction at the subfoveal region.20 Additional measurements were taken at 1500 μm and 3000 μm nasal and temporal to the fovea, as well as central macular thickness. No post-procedural interventions were required.

Data analysis was performed using IBM SPSS Statistics for Mac (version 24.0; IBM Corp., Armonk, NY). Descriptive statistics were reported as mean ± standard deviation or interquartile ranges (with minimum and maximum values) for quantitative variables, and frequencies with percentages for categorical data. Independent sample t-tests compared mean choroidal thickness between diabetic and non-diabetic groups. Within the diabetic cohort, associations with HbA1c and disease duration were

examined. Pearson's correlation coefficient was applied for correlation analysis, with statistical significance set at $p < 0.05$

Results:

A total of 165 patients were included in the study, 110 being diabetics and 55 non diabetic from the refractive clinic. The Mean Age was 48.24 ± 22.22 years, with a sex ratio being 1 male to 2.48 females. Mean IOP was 13.65 mm Hg ± 4.65. The demographics for the sample population are explained below in Table 1.

Table 1: Demographics of patients and group wise comparison between and diabetics and non-diabetics (n=165)

S. No	Characteristics	Total	Diabetic	Nondiabetic	P value*
1.	No of cases (n)	165	110	55	-
2.	Age Mean (+ SD)	48.24±22.22	47.36±1.52	50.12±1.61	-
3.	Sex (M:F)	1: 2.48	-	-	-
4.	HbA1C (Mean)	6.64 ± 1.13	7.3±0.76	5.34±0.32	-
5.	IOP	13.65±4.65	-	-	-
6.	Mean arterial pressure	83.00±12.11	-	-	-
7.	Choroidal thickness Nasal 1500 um	245.06±16.07	233.90±3.22	267.40±1.36	0.001*
8.	Choroidal thickness Nasal 3000 um	236.73±11.53	228.70±2.01	252.80±1.17	0.004*
9.	Choroidal thickness Temporal 1500 μm	245.73±15.18	235.10±1.92	267.00±1.42	0.094*
10.	Choroidal thickness Temporal 3000 um	250.93±13.98	241.10±0.94	270.60±1.02	0.136*
11.	Central choroidal thickness (Sub foveal)	252.86±10.28	245.70±1.42	267.20±1.84	0.001*
12.	Central macular thickness	248.20±4.33	251.10±1.52	242.40±1.02	0.631*

SD=Standard deviation, IOP=Intraocular pressures, HbA1C= Glycosylated hemoglobin
**Independent sample t-test*

The mean sub foveal central choroidal thickness, choroidal thickness at nasal 1500 and nasal 3000 were statistically significant in diabetic vs non diabetic group (p value=0.001) whereas the temporal 1500, (p value=0.094) temporal 3000(p value=0.136) and macular thickness (p value=0.631) were not found statistically significant between two groups. The detailed values are depicted as in table 1.

There was negative correlation found between HbA1C levels and mean choroidal

thickness. (R= -0.783) as shown in table 2. This suggested that higher HbA1c values are associated with thinner choroidal layers. Diabetic individuals showed significantly reduced choroidal thickness in central and nasal regions, which might indicate microvascular changes. HbA1c levels strongly correlate with choroidal thinning, highlighting the potential impact of glycemic control on ocular health.

Table 2: Association of mean choroidal thickness and HbA1C and disease duration

S. No			Pearson's corelation coefficient
1.	Mean Choroidal Thickness	Mean HbA1C	R= - 0.783
2.		Disease duration	R= < 0.1

Discussion:

This study explored the association between choroidal thickness and HbA1c levels in individuals with type 2 diabetes. When compared with non-diabetic controls, our findings suggest that higher HbA1c levels are linked with reduced choroidal thickness, indicating a possible detrimental impact of chronic hyperglycemia on choroidal structure.

Previous research on diabetic patients without retinopathy has produced mixed results. For example, Esmaeelpour et al.[21], Querques et al.[22], and Vujosevic et al.[23] each reported variable changes in choroidal thickness, although these investigations were limited by relatively small sample sizes. In our cohort, choroidal thickness was consistently reduced across all measured quadrants in the diabetic group compared to controls. This may reflect early structural alterations sometimes referred to as "diabetic choroidopathy" occurring before clinical evidence of retinopathy becomes apparent.

Lee et al.[24] assessed choroidal thickness in 203 diabetic eyes and 48 non-diabetic eyes, reporting no significant difference between diabetic patients without DR and healthy individuals. In contrast, Sudhalkar et al.[25] observed thinner choroids in diabetics (with or without DR) relative to controls, with progressive thinning noted from non-proliferative to proliferative stages of DR. Supporting the idea of early involvement, another study[26] also documented thinner choroid in diabetics without retinopathy compared with non-diabetic controls.

We found a negative correlation between HbA1C and choroidal thickness. This contrasts with findings from Yazgan et al.[27], who reported no such relationship when examining both macular and peripheral choroid using EDI-OCT, and even observed thicker choroids in diabetics compared to controls. Similarly, Shiba et al.[28] suggested that rising HbA1c might be associated with increased choroidal thickness, potentially reflecting hyperglycemia-induced vascular wall stress. The relationship between DR severity and choroidal thickness was not addressed in the present study, though prior work[29] has shown inconsistent patterns of change with disease progression.

Multiple systemic and ocular factors are known to influence choroidal thickness[30,31]. We attempted to minimize measurement variability by applying strict exclusion criteria, yet several limitations remain. This was a cross-sectional study, and examiners were aware of participants' group allocation, which could introduce observer bias. Additionally, the relative inexperience of the grader and incomplete control for potential confounders may have affected results. Future longitudinal studies with larger sample sizes, masked evaluation, and inclusion of patients across different DR stages would provide a more comprehensive understanding of how HbA1c levels influence choroidal morphology.

Conclusion:

The study concluded that elevated HbA1c levels adversely impact choroidal thickness in diabetics, even in the absence of diabetic retinopathy. Higher HbA1c values are associated with thinner choroidal layers. This highlights the potential role of choroidal alterations in the early stages of diabetic ocular changes even without retinopathy. Further longitudinal studies are warranted to explore the prognostic significance of choroidal thickness in diabetic retinopathy progression.

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