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Open-Angle Glaucoma with Diabetes Mellitus and Insufficient Glycaemic Control: Spectral-Domain Oct Characteristics

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Abstract: ***Aim: To assess the spectral-domain optical coherence tomography (SD-OCT) characteristics in open-angle glaucoma with diabetes mellitus in accordance with glycaemic control state. Methodology: Comprehensive eye examinations, visual field tests, and SDOCT imaging were all performed on the participants (Cirrus HD-OCT). In order to compare diabetic glaucomatous eyes with diabetic non-glaucomatous eyes, the link between glycosylated haemoglobin (HbA1c) and OCT measurements was examined. Results: Analysis was performed on 69 non-glaucomatous and 87 glaucomatous eyes in the nondiabetic group, and on 72 non-glaucomatous and 56 glaucomatous eyes in the diabetic group. Average, inferonasal, inferior, and inferotemporal ganglion cell–inner plexiform layer (GCIPL) thicknesses were positively correlated with HbA1c in diabetic non-glaucomatous eyes (P = 0.041, 0.037, 0.024, and 0.012, respectively). Conclusions: In this study, optic nerve head parameters had a superior ability to discriminate glaucoma in diabetic eyes with poor glycaemic control. Conversely, the ability to discriminate glaucoma using macular parameters tended to be lower for diabetic eyes with inadequate glycaemic control***

Keywords: diabetes mellitus, ganglion cell-inner plexiform layer, glaucoma, spectral-domain optical coherence tomography

1. Introduction

Increased eye pressure is the primary cause of glaucoma, a progressive visual condition that is defined by the slow degeneration of retinal ganglion cells (RGC) [1]. This eye condition, which is a leading global contributor to irreversible blindness, has created a significant public health issue[2]. Several reports had identified diabetes as a risk factor for POAG, although epidemiologic studies of the connection between diabetes and POAG are still debatable. Nevertheless, diabetic retinopathy often coexists with glaucomatous optic neuropathy.

The most prevalent form of glaucoma among diabetics is primary open angle glaucoma (POAG), which affects over 70 million people world-wide[3]. To design measures to lower its incidence, it is therefore necessary to identify potential risk factors for POAG.

The pathophysiology of primary open angle glaucoma is still not fully understood at this time. Some studies hypothesised that interference with blood control in the optic nerve head area caused damage to the microvasculature network and/or decreased nutritional supply to the RGC axons[4, 5]. This dietary insufficiency may trigger glaucomatous dysfunction and cause RGC degeneration. Consequently, any vascular-related systemic condition, such diabetes that affects the food supply to RGCs either directly or indirectly may lead to the development of POAG.

A statistically significant correlation between diabetes and glaucoma was discovered in two prior meta-analyses [6, 7].

Important in the detection and management of glaucoma is optical coherence tomography (OCT) imaging. OCT was first introduced in 1991 by Huang et al[8] as a non-invasive in vivo cross-sectional imaging technology using low-coherence interferometry.

OCT in the context of much ocular pathology has been reported[9, 10, 11]. Specifically pertaining to glaucoma, OCT enables the quantitative evaluation of critical neural structures, including the RNFL, the optic nerve head (ONH), and the macula. OCT has revolutionized the diagnosis, monitoring, and ultimately management of glaucoma by taking glaucoma from a primarily subjectively assessed disease to an objectively evaluated disease.

Time-domain OCT (TD-OCT) was the first generation of OCT. SD-OCT has the advantage of increased signal-to-noise ratio (SNR), resulting in enhanced image quality[12, 13]. Because scanning is so much faster, there can be fewer motion artifacts, and higher A-scan density can produce the illusion of higher transverse resolution when compared with TD-OCT.

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This study aimed to evaluate the quantitative changes of RNFL thickness, optic nerve head (ONH), total retinal thickness, and ganglion cell–inner plexiform layer (GCIPL) thickness parameters obtained by spectral-domain (SD)-OCT (Cirrus HD-OCT; Carl Zeiss Meditec, Dublin, CA, USA) in nonglaucomatous and glaucomatous eyes in both diabetic and nondiabetic patients

2. Methodology

A retrospective analysis was done where 130 diabetic patients and 150 non diabetic patients with and without glaucoma were reviewed. Ethical clearance was obtained from the institutional ethical committee.

An ophthalmologist determined the eligibility requirements for subjects with or without diabetes mellitus based on a thorough ophthalmologic examination, which included a review of the patient's medical history, best-corrected visual acuity measurements through manifestation refraction, Goldmann applanation tonometry, slit-lamp examination of the anterior segment, gonioscopy, dilated fundus examination, red-free fundus photography

Subjects with normal eyes were chosen because they had no history of retinal pathology, no history of intraocular surgery, no media opacity on slit-lamp examination, or any first-degree relative glaucoma.

A fasting glucose level of at least 126 mg/dL or current use of antidiabetic medicines were required to diagnose diabetes mellitus. When haemoglobin A1c (HbA1c) values were 7.0% or higher, the glycaemic status was classified as either good glycaemic control or poor glycaemic control, respectively[14].

Enrolled subjects with diabetes had mild to severe non-proliferative diabetic retinopathy based on the International Clinical Diabetic Retinopathy Disease Severity Scale. Fluorescein angiography was performed in subjects with DM to determine the stage of retinopathy and to exclude subjects with macular oedema. Two ophthalmologists identified the various types of diabetic retinopathy in a disguised manner

Inclusion criteria

- Patients with open anterior chamber angles and clear ocular media by slit-lamp evaluation

Exclusion criteria

- Patients with histories of intraocular surgery and neurologic disorders
- Diabetic eyes with macular oedema
- Diabetic eyes with history of pan-retinal photocoagulation (PRP)
- Diabetic eyes with a history of intravitreal injection and history of intraocular surgery
- Patients with other concomitant retinal diseases

Optical coherence tomography

One optic disc cube protocol and one macular cube protocol were recorded using a Cirrus HD-OCT in each eligible eye. The cube scan was intended to be positioned on the ONH using the optic disc cube protocol. This procedure acquired a sequence of 200 horizontal scan lines, each made up of 200 A-scans, to create a cube of data through a 6-mm-square grid (40, 000 points). An RNFL thickness map was created after measuring the RNFL thickness at each pixel. The optic disc was automatically surrounded by a calculating circle with a diameter of 3.46 mm and 256 A scans. After that, the mean and sectoral RNFL thicknesses (temporal, superior, nasal, and inferior) were measured.

Rim area, optic disc area, mean cup-to-disc area ratio (CDR), vertical CDR, and cup volume were the ONH metrics that were examined. An ONH analysis algorithm created for the Cirrus HD-OCT was used to automatically measure these parameters. In more detail, the method detects the termination of Bruch's membrane as the disc edge and defines the disc and cup edges within the three-dimensional data cube. The thickness of the neuro-retinal tissue in the optic nerve as it rotated to depart via the opening in Bruch's membrane was then measured to estimate the rim width across the complete circumference of the optic disc.

The fovea was the focal point of macular cube scans in a 6 x 6 mm^2 area using the macular cube 512 x 128 or 200 x 200 scan protocols. Software that came with the scanner was a 6-mm-diameter circle centred at the true fovea location, as defined by the Early Treatment Diabetic Retinopathy Study (ETDRS); ETDRS areas include a central 1-mm disc and inner and outer rings of 3 and 6 mm, respectively. used to produce retinal thickness maps, which were then averaged over nine retinal subfields. The innermost 1-mm-diameter circle's central foveal subfield thickness (central macular thickness) was then determined. The output of the computational software was also used to determine the overall average macular thickness (cube average thickness) and overall macular cube volume over the entire grid area based on the proportional contribution of the regional macular thicknesses.

The Ganglion Cell Analysis (GCA) method, which processes information from three-dimensional volume scans, was the second tool we used. use the 200 x 200 or 512 x128 macular acquisition methodology. The algorithm determines the RNFL's outer boundary and the inner plexiform layer's outer boundary (IPL). The combined thickness of the RGC layer and the IPL, which in turn offers a measurement of the macular GCIPL thickness inside a 14.13-mm^2 elliptical annulus area centred on the fovea, is obtained from the difference between the RNFL and the IPL outer boundary segmentations.

Six distinct sectors of the GCIPL thickness—superior, superonasal, inferonasal, inferior, and inferotemporal—as well as the mean and lowest values were presented. The minimal GCIPL measurement was obtained by choosing the spoke with the lowest average out of 360 spokes of measurements that extended from the fovea's centre to the edge of the ellipse at 18 intervals.

The signal intensity of every image captured for this investigation was below six. The optic disc or the fovea had to be precisely centred on each scan. We omitted from our

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analysis any inaccurate images caused by segmentation algorithm mistakes, unintentional saccades, or blinking artefacts.

Statistical analysis

When both of a patient's eyes were eligible, one eye was randomly selected for analysis. Clinical factors associated with OCT parameters in non-glaucomatous eyes were evaluated using univariate linear regression analysis. Relationships between HbA1c levels and OCT parameters were evaluated using multivariate linear regression analysis adjusted for age and sex in diabetic non-glaucomatous and glaucomatous eyes. In the pooled population without glaucoma, OCT parameters among groups (nondiabetic eyes versus diabetic eyes with HbA1c < 7.0% versus diabetic eyes with HbA1c ‡ 7.0%) were analysed using 1-way analysis of variance and Scheffe's post hoc tests. Data analysis was done using SPSS version 20.0

3. Results

Sixty-nine eyes of 69 nondiabetic nonglaucoma subjects (normal), 87 eyes of 87 nondiabetic glaucoma patients (OAG [open-angle glaucoma]), 72 eyes of 72 diabetic nonglaucoma subjects (DM-OAG), and 56 eyes of 56 diabetic glaucoma patients (DM +OAG) were included in the present study. The characteristics of the study population are shown in Table 1.

In both the nondiabetic and diabetic groups, the average CDR was significantly higher in glaucomatous eyes than in eyes without glaucoma.

Visual field examinations also showed that the non-glaucomatous and glaucoma groups, as well as the non-diabetic and diabetic groups, had various retinal sensitivities.

No significant differences were found between non-glaucomatous and glaucomatous eyes with respect to age, sex, intraocular pressure, spherical equivalent, and disc area in either the nondiabetic group or the diabetic group.

Of 124 diabetic eyes, those with glaucoma had diabetes for 9.27 years while those without glaucoma had it for 11.40 years; the difference was not statistically significant. (P = 0.093).

Looking into HbA1c level, compared to glaucoma patients, non-glaucomatous subjects had a slightly higher HbA1c level. (8.44 ±2.07% and 7.71 ±1.73%, P =0.049).

A total of 58 eyes had diabetic retinopathy, with 16 having glaucoma and 42 not having it. (P=0.776). Eyes with diabetic retinopathy had mild to severe non-proliferative diabetic retinopathy without maculopathy

Factors associated with OCT parameters in non-glaucomatous eyes.

Average RNFL thickness, cube thickness, and average GCIPL thickness were all inversely linked with age (P = 0.001, < 0.001, and < 0.001, respectively).

Both average cube thickness and average GCIPL thickness were significantly lower in females. (P = 0.005 and 0.019, respectively). Pattern standard deviation was significantly associated positively with average GCIPL thickness. (P = 0.047).

Diabetes mellitus existence, type of DM, duration of DM, and HbA1c levels were not correlated with average RNFL thickness, rim area, cube average thickness, and average GCIPL thickness (all P > 0.05).

The average RNFL thickness, rim area, cube average thickness, and average GCIPL thickness were not linked with intraocular pressure, spherical equivalent, or mean deviation. (all P > 0.05) (Table 2).

OCT measurements according to the status of glycaemic control

Table 3 shows the relationship between glycosylated haemoglobin levels and OCT parameters evaluated in diabetic non-glaucomatous eyes and diabetic glaucomatous eyes.

The HbA1c level and temporal RNFL thickness were positively associated in diabetic eyes. (co-relation coefficient = 0.036, P = 0.007).

One macular parameterr, cube average thickness, was positively related to the HbA1c level (correlation coefficient = 0.025, P = 0.035).

In addition, average GCIPL, inferonasal GCIPL, inferior GCIPL, and inferotemporal GCIPL thicknesses were positively correlated with the HbA1c level (correlation coefficient = 0.041, 0.039, 0.047, and 0.041, P =0.041, 0.037, 0.024, and 0.012, respectively).

Comparison of OCT Measurements in Nondiabetic and Diabetic Eyes

Comparisons of the OCT measurements in the pooled population of subjects with non-glaucomatous eyes (69 nondiabetic eyes, 16 diabetic eyes with HbA1c < 7.0%, and 42 diabetic eyes with HbA1c >/7.0%) are shown in Table 4.

The average RNFL thickness and other RNFL sectoral parameters (temporal, superior, nasal, and inferior thickness) did not differ significantly among groups. For the ONH parameters, including rim area, disc area, average CDR, vertical CDR, and cup volume, no differences were found between eyes with and without diabetes. We next examined macular parameters. Total retinal thickness parameters such as central macular thickness, cube volume, and cube average thickness were not found to be significantly different among the groups (P = 0.916, 0.816, and 0.931, respectively). The average GCIPL thickness and other GCIPL sectoral parameters (supero-temporal, superior, supero-nasal, inferonasal, inferior, and inferotemporal thickness) did not differ significantly among the three groups (all P > 0.05).

The minimum GCIPL thickness differed significantly among nondiabetic eyes, diabetic eyes with good glycaemic control, and diabetic eyes with poor glycaemic control (78.61±7.16, **76.16±10.16** and 79.16±11.16, respectively, P = 0.019).

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Table 1: Demographics

	No DM, n=156			DM, n=130		
	Normal, n=69	OAG, n=87	P value	DM-OAG, n=72	**DM+OAG, n=56**	P value
Age	58.16±7.31	58.76±8.16	0.7600	59.12±9.16	**64.76±11.18**	0.057
Male, female	39.41	50.61	0.810	42: 30	**27: 29**	0.255
IOP, mmHg	13.16±4.12	14.76±3.1	0.201	14.31±3.16	**13.91±3.26**	0.181
Spherical equivalent, D	- 0.38±1.61	0.03±1.57	0.131	- 0.51±1.86	**- 0.21±1.36**	0.610
Disc area, mm^2	2.01±0.51	2.13±0.53	0.231	2.01±0.26	**2.06±0.31**	0.721
Average CDR	0.59±0.14	0.76±0.14	<0.001	0.58±0.11	**0.72±0.07**	<0.001
Vertical CDR	0.57±0.12	0.73±0.14	<0.001	0.53±0.80	**0.68±0.07**	<0.001
Mean direction, dB	- .176±4.16	- 7.16±6.36	<0.001	- 3.21±3.71	**- 7.16±7.16**	0.004
Pattern SD, dB	1.94±1.44	5.12±4.12	<0.001	2.61±2.31	**5.31±4.31**	0.0031
DM duration				12.3±6.16	**9.16±5.9**	0.093
HBA1C%				8.13±2.01	**7.31±1.63**	0.049
Type 1, type 2 DM				32: 35	**15: 39**	0.025
Diabetic retinopathy						
No diabetic retinopathy				42: 30	**16: 40**	0.001
Diabetic retinopathy, mild mod and very severe				15: 13: 14	**7: 5: 4**	0.776

Table 2: Clinical Factors and OCT Measurements Relationship in 69 Normal and 72 Diabetic non-glaucomatous Eyes (n 14 141)

	RNFL thickness		Rim area		Cube average thickness		Average GCPPL thickness	
	β	P	β	P	**β**	**P**	β	**P**
Age, years	-0.271	0.001	0.000	0.718	**-0.458**	**<0.001**	-0.271	<0.001
Female sex	-3.164	0.049	0.002	0.951	**-6.411**	**0.005**	**2.591**	0.019
DM yes	-0.936	0.531	0.017	0.576	**-0.831**	**0.606**	**-0.461**	0.597
Type 2 DM	-1.017	0.276	0.005	0.731	**-1.914**	**0.159**	**-0.714**	0.317
Duration of DM	0.211	0.146	0.001	0.618	**-0.229**	**0.421**	**0.004**	0.971
HBA1C%	0.841	0.181	0.017	0.126	**1.271**	**0.186**	**0.591**	0.121
IOP, mmHg	-0.491	0.059	-0.006	0.161	**-0.241**	**0.387**	**-0.210**	0.219
Spherical equivalent, D	0.281	0.624	-0.013	0.391	**0.315**	**0.815**	**0.419**	0.371
MD, dB	0.139	0.616	-0.012	0.049	**-0.351**	**0.361**	**-0.281**	0.117
Pattern SD, dB	-0.216	0.681	0.015	0.189	**0.816**	**0.251**	**0.713**	0.047

Table 3: Association between HbA1c (%) and OCT parameters measured using spectral-domain optical coherence tomography in 58 diabetic eyes without glaucoma and 50 diabetic eyes with glaucoma

	DM, n=108		DM-OAG, n=58		DM+OAG, n=50	
	β	P	β	P	β	P
RNFL						
Average thickness, µm	0.017	0.172	0.031	0.210	-0.001	0.916
Temporal thickness, µm	0.036	0.007	0.039	0.051	0.031	0.271
Superior thickness, µm	0.003	0.731	-0.001	0.871	-0.006	0.618
Nasal thickness, µm	0.027	0.161	0.046	0.073	-0.001	0.916
Inferior thickness, µm	0.007	0.416	0.016	0.562	-0.001	0.871
ONH						
Rim area	0.096	0.216	1.616	0.207	-0.491	0.611
Disc area	0.681	0.159	1.317	0.181	0.581	0.281
Average CDR	-0.916	0.618	0.159	0.916	3.618	0.310
Vertical CDR	-1.618	0.319	0.819	0.730	0.691	0.731
Cup volume	0.049	0.898	0.179	0.891	1.381	0.269
Total retinal thickness,						
Central macular thickness, µm	0.004	0.437	0.006	0.491	0.003	0.813
Cube volume, mm^2	0.597	0.059	0.369	0.512	0.612	0.101
Cube average thickness, µm	0.025	0.035	0.019	0.276	0.019	0.126
GCIPL						
Average thickness, µm	0.041	0.041	0.071	0.167	0.031	0.219
Minimum thickness, µm	0.017	0.261	0.002	0.919	0.019	0.271
Supero temporal thickness, µm	0.037	0.097	0.037	0.510	0.027	0.276
Superior thickness, µm	0.027	0.289	0.035	0.476	0.013	0.589
Superonasal thickness, µm	0.016	0.310	0.003	0.910	0.018	0.347
Inferonasal thickness, µm	0.039	0.037	0.046	0.176	0.027	0.161
Inferior thickness, µm	0.047	0.024	0.061	0.126	0.029	0.151
Inferotemporal thickness, µm	0.041	0.012	0.076	0.036	**0.027**	0.261

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Table 4: Spectral-domain optical coherence tomography was used to measure the OCT parameters in 69 normal eyes, 18 diabetic eyes with good glycaemic control, and 42 diabetic eyes with poor glycaemic control.

	Normal, n=69	DM-OAG, n=58		P	P
		Good glycaemic control	Poor glycaemic control		
RNFL					
Average thickness, µm	95.16±7.91	**94.31±10.16**	96.16±10.13	0.979	0.981
Temporal thickness, µm	68.41±10.23	**69.91±9.79**	73.16±12.31	0.361	0.741
Superior thickness, µm	121±17.50	**120.16±14.13**	115.76±15.61	0.236	0.721
Nasal thickness, µm	70.03±9.61	**68.96±9.04**	73.16±11.13	0.230	0.272
Inferior thickness, µm	121.86±14.581	**124.00±14.611**	123.61±12.36	0.913	0.971
ONH					
Rim area	1.61±0.76	**1.59±0.16**	1.23±0.18	0.271	0.411
Disc area	2.04±0.39	**2.05±0.27**	2.04±0.26	0.971	0.911
Average CDR	0.76±0.12	**0.70±0.07**	0.70±0.16	0.516	0.534
Vertical CDR	0.49±0.11	**0.48±0.90**	0.55±0.16	0.641	0.916
Cup volume	0.25±0.18	**0.31±0.16**	0.25±0.17	0.515	0.571
Total retinal thickness					
Central macular thickness, µm	247.18±23.19	**249.36±21.16**	248.16±27.16	0.916	0.937
Cube volume, mm^2	10.03±0.41	**10.02±0.51**	9.78±0.49	0.816	0.862
Cube average thickness, µm	279.11±11.61	**278.11±13.06**	278.90±13.16	0.931	0.961
GCIPL					
Average thickness, µm	83.71±7.39	**83.61±7.41**	82.16±7.18	0.721	0.916
Minimum thickness, µm	78.61±7.16	**76.16±10.16**	79.16±11.16	0.019	0.811
Supero-temporal thickness, µm	83.16±8.16	**82.16±6.76**	81.50±6.91	0.739	0.971
Superior thickness, µm	83.16±5.91	**82.79±5.16**	82.61±6.16	0.911	0.916
Supero-nasal thickness, µm	84.36±8.16	**84.16±8.01**	83.91±8.16	0.671	0.961
Inferonasal thickness, µm	82.16±5.61	**82.27±7.16**	80.16±9.16	0.516	0.713
Inferior thickness, µm	80.01±7.11	**80.11±7.91**	78.31±7.11	0.861	0.916
Inferotemporal thickness, µm	83.16±5.16	**80.17±9.61**	82.16±6.11	0.691	0.876

4. Discussion

This study investigated whether DM, and further inadequate glycaemic control, affects OCT RNFL, ONH, total retina, and GCIPL measurements. The temporal RNFL thickness, cube average thickness, average GCIPL thickness, inferonasal GCIPL thickness, inferior GCIPL thickness, and inferotemporal GCIPL thickness tended to be greater according to higher HbA1c levels in diabetic eyes. The minimum GCIPL thickness had a tendency to be less among patients with DM. In diabetic patients with poor glycaemic control, the vertical CDR exhibited significantly higher diagnostic abilities than average GCIPL thickness and cube average thickness.

Diagnosis of Glaucoma in Patients with Diabetes Mellitus

In some circumstances, it can be difficult to distinguish between glaucomatous change and diabetic change. It can be challenging to distinguish between diabetic and glaucomatous changes because some early-stage glaucoma patients only have decreased RNFL thickness without other recognisable changes to the optic disc. In fact, a patient with DM and hypertension had an instance of a localised RNFL defect developing after a retinal cotton-wool spot[15]. The optic nerves in eyes treated with PRP are also more likely to be deemed abnormal by glaucoma experts, according to a prior study[16]. When diabetic retinopathy and glaucoma coexist, visual field losses frequently have amorphous patterns that are affected by retinal haemorrhages, exudates, and damaged retinal nerve fibres[17].

Effect of Glycosylated Haemoglobin Levels on Retinal Changes

A few investigations have looked at the connection between changes in retinal thickness and glycaemic control. According to Chihara et al[18], there is no correlation between the incidence of RNFL defects and the amount of glycosylated haemoglobin at the time of examination. The use of a retinal thickness analyser (RTA) in a 3-year follow-up analysis of retinal thickness alterations in patients with type 2 diabetes also revealed no connection between changes in retinal thickness and changes in HbA1c readings[19]. Rim area and rim volume measurements in a study using Heidelberg retina tomography (HRT) III were not substantially correlated with HbA1c levels[20]. Furthermore, RNFL measurements after 1-month blood glucose regulation were not significantly different, according to research evaluating the impact of glycaemic control on RNFL thickness[21].

The temporary worsening of diabetic retinopathy following intensive glycaemic control, on the other hand, was described by a different study to have occurred after 4 months of glycaemic control[22]. Cube average thickness, average GCIPL, inferonasal GCIPL, inferior GCIPL, and inferotemporal GCIPL thickness parameters measured in the macular region were favourably correlated with HbA1c levels at the time of OCT examination in this research, among other parameters evaluated. Retinal changes in diabetes mellitus can result from a number of pathways incorporating various factors. Further complicating factors for diabetic eyes with various phenotypes include the degree of diabetic retinopathy, the length of diabetes, the type of DM, and glycosylated haemoglobin values.

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For example, while prolonged duration of poor glycaemic control might induce retinal thinning, simultaneous intracellular or extracellular oedema can lead to increased retinal thickness.

Optic Disc Parameters for Diagnosis of Glaucoma in Diabetic Eyes

In line with the histopathologic evidence in diabetic eyes, several clinical investigations have found RNFL defects or thinning in diabetic eyes without glaucoma[23-25]. An experimental study found that diabetic rodents had decreased cross-sectional dimensions of large optic nerve fibres and impaired retrograde axonal transport[26]. Other morphologic investigations using TUNEL staining have shown that increased neuroglial apoptosis may cause diabetes-related RNFL loss to start sooner than expected[27]. In this research, there was no discernible difference in RNFL thickness between diabetic and nondiabetic eyes. In previous research, the difference between diabetic subjects and healthy controls for RNFL thickness measured by Stratus OCT was not statistically significant[17].

The research populations that were examined, the kind of imaging equipment used, and the algorithms used to measure retinal thickness may have contributed to the differences in findings from prior studies. Pathologic cupping in ONH advances in glaucomatous optic neuropathy eyes as glial cells and nerve fibre density decline. Optic nerve cupping is not a characteristic of diabetes, in contrast to glaucoma, despite the fact that both disease types have RNFL wedge defects[28, 29].

In this research, there was no discernible difference in ONH parameters between diabetic and nondiabetic eyes. Additionally, a number of studies have shown that diabetes preserves the neuro-retinal rim, which can offer crucial hints for distinguishing between glaucoma and diabetes[29, 30]. Another paper published recently described a clock hour-based analysis of the rim-RNFL correlation that could be useful in identifying normal-tension glaucoma in diabetic patients[31]. The highest AUCs for glaucoma diagnosis were seen in diabetic eyes with poor glycaemic control for the vertical CDR, average CDR, cup volume, and average RNFL thickness in the current research.

Because they are comparatively less affected by DM status and glycaemic control status, ONH and RNFL parameters appear to be more effective at detecting glaucoma in eyes with poor glycaemic control than total macular thickness and macular GCIPL parameters. To put it another way, neuronal thinning affects both ONH/RNFL parameters and macular total/GCIPL parameters, whereas retinal oedema may only have an impact on macular parameters. However, in clinical circumstances, both clinical and photographic evaluations of the ONH should be carried out at the same time because ONH analysis by OCT can be inaccurate when there are issues with the ONH recognition algorithm or scan centration. Furthermore, the presence of diabetes may have an impact on ONH and RNFL parameters, as in cases where macular oedema spreads broadly to the optic disc.

5. Conclusion

In this research, non-diabetic eyes, eyes with diabetes who had good glycaemic control, and eyes with diabetes who had poor glycaemic control were compared for quantitative changes in SD-OCT parameters and their diagnostic capabilities for open-angle glaucoma. Between diabetic and non-diabetic eyes, there was a significant difference in the minimal GCIPL thickness. To comprehend the mechanisms of neuronal changes in diabetes and glaucoma, additional studies assessing retinal qualitative changes detected by high-resolution OCT are required.

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