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Original Article

The role of OCT versus OCTA in Diabetic Patients with and without diabetic retinopathy

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ABSTRACT

Objective: The aim of this study is to compare performance/utility of optical coherence tomography (OCT) versus OCT-Angiography (OCTA) in assessment of retina and retinal vessels of diabetic patients with and without diabetic retinopathy (DR). **Methods:** This case control study involved 64 eyes of 64 participants differentiated into 4 equal groups each of 16 eyes. Group (1); normal healthy individuals (control group), group (2): diabetic patients without DR, group (3): diabetic patients with non-proliferative DR, group (4): diabetic patients with proliferative DR. All included eyes had best-corrected visual acuity (BCVA) greater than 0.5 Log MAR in the studied eye at baseline (to ensure proper execution of examination). Both sexes with age between 30-60 years were included, while patients with elevated intra-ocular pressure (IOP), high myopia, and those with media opacity were excluded. **Results:** Comparison of foveal thickness between the four groups showed statistically high significant difference (p=0.002), while parafoveal areas showed non-significant difference. However, central and all paracentral quadrants choroidal thickness showed statistically significant difference. Comparison of vascular patterns by OCTA showed highly significant difference (p <0.001) between the four studied groups. Post-Hoc test displaying the difference between each group and the other and mostly significant differences regarding retinal or choroidal thickness and density. **Conclusions:** The results of this study suggested that OCTA can identify preclinical DR before the manifestations of clinically apparent retinopathy. They highlight the potential role of OCT-A in monitoring and quantifying retinal vascular alterations in diabetes.

Keywords: OCTA, Diabetic retinopathy, Foveal thickness.

INTRODUCTION

Diabetic retinopathy (DR) is one of the most frequent diabetic complications, which has become a chief cause for vision loss, mostly because of vitreous hemorrhage and macular edema **[1].**The increased duration of developing diabetes disease increases the possibility of developing DR **[2].** The precise mechanism by which diabetes causes retinopathy is still unclear, but numerous studies based on imaging and histopathology revealed that DR is a consequence of microvascular changes including capillary remodeling, regression and decreased density **[3].**

Optical coherence tomography angiography (OCTA), a dye-free imaging technique is beneficial for visualizing retinal and choroidal vasculature and has permitted detection of angiographic features of DR and changes in the macular capillary network, even before disease onset. Areas of non-perfusion and their localization in the superficial and deep plexuses, irregular capillaries, and micro aneurysms have been clearly analyzed in DR patients using OCTA **[4].**

The superficial retinal capillary plexus can be accurately mapped and quantified by OCTA, and pathologic alterations in the foveal microvascular networks can be detected **[5].**Recent research using OCTA has reported quantifying the density of vessels in the macula**[6].**

Swept-source OCT allows for faster scanning and its longer wavelength enables deeper penetration in the choroid revealing more details and a clearer sclero-choroidal interface **[7].**

The aim of this study is to compare performance/utility of OCT versus OCTA in assessment of retina and retinal vessels of diabetic patients with and without DR.

PATIENTS AND METHODS

This case control study was conducted between January 2022 and August2023 at Bahgat Eye Clinic, Zagazig, Egypt. The Institutional Review Board approved the study protocol, which adhered to the tenets of the Declaration of Helsinki, andwritteninformedconsentswereobtainedfromallparticipantsbeforeparticipation.

It involved 64eyes of 64 participants differentiated into 4 equal groups each of 16 eyes. Group (1); normal healthy individuals (control group), group (2): diabetic patients without DR, group (3): diabetic patients with non-proliferative DR, group (4): diabetic patients with proliferative DR. All included eyes had BCVA greater than 0.5 Log MAR in the studied eye at baseline (to ensure proper execution of examination). Both sexes with age between 30-60 years were included, while patients with high myopia, elevated IOP, and those with media opacity were excluded.

Data Collection Tools:

History and clinical ophthalmic examination including visual acuity, refraction, IOP measurement, slit lampbio microscopy and BCVA were done.

SS-OCT and OCT angiography image acquisition was done. During the same visit, all study subjects underwent swept-source (SS)-OCT examination (DRI Triton, Topcon, Tokyo, Japan), which contains a 1,050-nm-wavelength swept light source and has a scanning speed of 100,000 A-scans/second.

OCT was done to acquire:

a- Retinal thickness at the fovea and para foveal area using a six-line radial pattern scan (1,024 A-scans) centered on the fovea from each eye.

b- Choroidal thickness measured (nasal, temporal, superior and inferior) at 2 mm from the fovea.

We obtained a six-line radial pattern scan (1,024 A-scans) centered on the fovea from each eye. The definition of choroidal thickness was the vertical distance between the posterior edge of the hyper-reflective retinal pigment epithelium and the choroid/sclera junction. The choroidal thickness was manually measured using a built-in caliper in the OCT software.

OCT –Angiography was done to study:

a) Quantitative measuring of Foveal Avascular Zone (FAZ) area at SCP using the (3x3 mm scan) We outlined the FAZ area and perimeter manually along the innermost capillaries on OCT angiography images at the SCP.

b) Superficial capillary plexus (SCP) and Deep capillary plexus (DCP) (qualitative analysis) at the parafoveal area in (4.5x4.5 mm scan).

c) Quantitative measuring the retinal vessels density map at the SCP in the

(4.5x4.5 mm scan) (measured automatically by the device).

d) Measuring the choroidal vessels density map in the (4.5x4.5 mm scan) (Measured manually by the operator by applying a superior line at the level of Bruch's membrane and an inferior line at the sclera –choroidal interface (SCI).

The OCT device automatically segments the layers using a built-in segmentation algorithm for the superficial plexus (2.6 µm below the internal limiting membrane to 15.6 µm below the junction between the inner plexiform and inner nuclear layers (IPL/INL) and deep plexus (15.6 µm below the IPL/INL to 70.2 µm below the IPL/INL). En-face projections of volumetric scans allow for the visualization of structural and vascular details within segmented retinal layer boundaries. We only used OCT images with a signal strength index >60 and excluded scans with poor image quality. Scans with poor image quality met these criteria: (1)weak local signal or poor clarity, (2) poor fixation resulting in a double vessel pattern and motion artifacts, (3) macular edema, and (4) macular segmentation errors **[8]**.

STATICAL ANALYSIS

Statistical analysis was performed using SPSSv23 statistical software (SPSS, Inc, Chicago, Illinois). Quantitative data were represented as (mean and standard deviation).Two-sided Chi-square, student-t and ANOVA test were used for parametric data, and Mann-Whitney U and Kruskal Wallis tests were employed for non-parametric variables. The significance level was calculated and $P \leq 0.05$ was considered statistically significant, while $P > 0.05$was considered statistically non-significant.

RESULTS

Thisstudyinvolved64 eyes of 64 patients: 29 males (45.3%) and 35 females (54.7%). The mean ages were 38.3±8.93 y, 42.2±14 y, 49.4±8.59 y, and 50.8±7.37 y in the control group, non-DR group, NPDR group and PDR group, respectively. The mean Log MAR BCVAs were 0.01±0.03, 0.03±0.06, 0.04±0.07, and 0.18±0.16 and the mean IOP was 12.0±1.46, 12.2±1.11, 12.7±1.62 and 13.1±1.34 mmHg, respectively **(table1)**.

Comparison of central retinal (foveal) thickness by OCT between the four groups showed statistically high significant difference (p = 0.002), while parafoveal areas showed non-significant difference. On the other hand, central and all paracentral quadrants choroidal thickness showed statistically significant difference **(table2)**.

Comparison of vascular patterns by OCTA showed statistically high significant difference (p <0.001) between the four studied groups**(table3)**. Post-Hoc test displaying the difference between each group and the other and mostly significant differences regarding retinal or choroidal thickness and density **(table 4)**.

On studying the correlation between retinal thickness and retinal density, and between choroidal thickness and choroidal density showed non-significant differences **(table5)**.

Correlation coefficient(r) between FAZ area and central retinal (foveal) thickness **(Fig.1)** as well as the receiver operating characteristics (ROC) curve showed considerable diagnostic value of foveal thickness in differentiation between patients with proliferative diabetic patients and the rest of studied patients and controls. The area under the curve (AUC) was 0.837 with significance <0.001 and cut-off value of 252 µm **(table 6)**. This means that foveal thickness of more than 252 µm indicating PDR in this study.

Table (1): Patients' characteristics of the two studied groups.

Variable	Control	Non-DR	NPDR	PDR	ANOVA	P
Age (y)	38.3±8.93	42.2±14	49.4±8.59	50.8±7.37	5.53	0.002*
Log MAR BCVA	0.01±0.03	0.03±0.06	0.04±0.07	0.18±0.16	11.07	0.000*
IOP (mmHg)	12.0±1.46	12.2±1.11	12.7±1.62	13.1±1.34	1.913	0.137

*P= statistically significant.

Table (2): Comparison of retinal and choroidal thickness between the four studied groups

Retinal thickness	Control		Non-DR		NPDR		PDR		Significance	
	Mean	±SD	Mean	±SD	Mean	±SD	Mean	±SD	ANOVA	P
CRT (µm)	238.5	2.97	238.06	1.61	233.75	0.77	230.25	1.48	68.6	0.000*
SPFT (µm)	310.7	9.39	309.38	11.22	308.88	16.24	299.44	15.28	2.397	0.077
IPFT (µm)	296.4	11.86	294.13	36.4	292.06	31.38	291.75	25.11	0.09	0.962
NPFT (µm)	300.8	24.02	300.50	9.61	296.63	28.35	282.62	32.95	1.83	0.151
TPFT (µm)	293.6	12.27	292.44	16.6	292.19	13.18	290.50	12.09	0.14	0.938
Choroidal thickness	**Control**		**Non-DR**		**NPDR**		**PDR**		**Significance**	
	Mean	±SD	Mean	±SD	Mean	±SD	Mean	±SD	t	P
CCT (µm)	265.5	14.65	229.50	2.10	225.31	1.70	196.56	21.65	74.1	0.000*
SCT (µm)	266.8	40.45	236.31	26.6	234.25	2.91	173.88	13.92	38.1	0.000*
ICT (µm)	255.0	23.74	226.31	3.34	218.88	3.34	186.56	31.21	32.5	0.000*
NCT (µm)	251.1	50.10	234.13	35.9	231.88	73.54	191.31	70.43	2.90	0.042*
TCT (µm)	265.5	14.65	229.50	2.10	225.31	1.70	196.56	21.65	74.1	0.000*

*p <0.05: significant, CRT: central retinal thickness, SPFT: superior parafoveal thickness, IPFT: inferior parafoveal thickness, NPFT: nasal parafoveal thickness, TPFT: temporal parafoveal thickness, CCT: central choroidal thickness, SCT: superior choroidal thickness, ICT: inferior choroidal thickness, NCT: nasal choroidal thickness, TCT: temporal choroidal thickness.

Table (3): Comparison of vascular patterns between the four studied groups

Area	Control		Non-DR		NPDR		PDR		Significance	
	Mean	±SD	Mean	±SD	Mean	±SD	Mean	±SD	ANOVA	P
FAZ (mm)	0.29	0.02	0.29	0.04	0.40	0.05	0.48	0.06	57.92	0.000*
SRD (µm)	55.74	.57	53.36	1.83	52.10	1.31	42.57	4.34	88.18	0.000*
IRD (µm)	56.87	1.89	54.19	1.24	50.71	1.97	43.28	3.77	95.52	0.000*
NRD (µm)	48.43	1.29	46.03	1.11	45.01	1.36	41.55	1.25	82.86	0.000*
TRD (µm)	53.68	2.05	52.92	1.36	50.29	1.20	40.77	2.87	143.5	0.000*
SCD (µm)	50.68	0.68	48.23	2.32	44.19	2.34	40.28	5.60	31.41	0.000*
ICD (µm)	53.77	4.16	49.18	0.93	46.58	0.80	38.35	4.20	73.58	0.000*
NCD (µm)	49.46	4.57	45.73	2.41	45.04	2.04	42.31	1.08	17.33	0.000*
TCD (µm)	57.43	5.88	47.35	1.79	44.46	2.91	40.67	3.13	58.86	0.000*

ANOVA test, *p <0.001: highly significant, FAZ: foveal avascular zone, SRD: superior retinal density, IRD: inferior retinal density, NRD: nasal retinal density, TRD: temporal retinal density, SCD: superior choroidal density, ICD: inferior choroidal density, NCD: nasal choroidal density, TCD: temporal choroidal density.

Table (4): Post Hoc test displaying multiple comparisons within groups.

	P1	P2	P3	P4	P5	P6
FAZ Thickness	0.514	0.000*	0.000*	0.000*	0.000*	0.000*
FAZ Density	0.681	0.000*	0.000*	0.000*	0.000*	0.000*
Superior retinal density	0.008*	0.000*	0.000*	0.153	0.000*	0.153
Inferior retinal density	0.003*	0.000*	0.000*	0.000*	0.000*	0.000*
Nasal retinal density	0.000*	0.000*	0.000*	0.025*	0.000*	0.000*
Temporal retinal density	0.281	0.000*	0.000*	0.000*	0.000*	0.000*
Superior choroidal thickness	0.000*	0.000*	0.000*	0.371	0.000*	0.000*
Inferior choroidal thickness	0.001*	0.001*	0.000*	0.818	0.000*	0.000*
Nasal choroidal thickness	0.000*	0.000*	0.000*	0.291	0.000*	0.000*
Temporal choroidal thickness	0.424	0.366	0.006*	0.915	0.046*	0.059
Superior choroidal density	0.038*	0.000*	0.000*	0.001*	0.000*	0.001*
Inferior choroidal density	0.000*	0.000*	0.000*	0.018*	0.000*	0.000*
Nasal choroidal density	0.000*	0.000*	0.000*	0.494	0.001*	0.008*
Temporal choroidal density	0.000*	0.000*	0.000*	0.033*	0.000*	0.006*

*p <0.05: significant, FAZ: foveal avascular zone, P1: Comparison between control vs non-diabetic retinopathy, P2: Control vs non proliferative DR, P3: Control vs proliferative DR, P4: Non diabetic retinopathy vs non proliferative DR, P5: Non diabetic retinopathy vs proliferative DR, P6: Non proliferative DR vs proliferative DR.

Table (5): Correlation between retinal thickness and retinal density, and between choroidal thickness and choroidal density.

Retinal	**SPFT**		**IPFT**		**NPFT**		**TPFT**	
	r	**P**	**r**	**P**	**r**	**P**	**r**	**P**
SRD	-0.101	0.494	0.045	0.759	-0.145	0.325	-0.171	0.245
IRD	0.093	0.531	0.095	0.519	-0.23	0.116	-0.111	0.452
NRD	-0.03	0.842	0.231	0.114	-0.237	0.104	0.003	0.986
TRD	-0.057	0.702	0.226	0.122	-0.163	0.269	-0.074	0.616
Choroidal	**SCT**		**ICT**		**NCT**		**TCT**	
	r	**P**	**r**	**P**	**r**	**P**	**r**	**P**
SCD	0.207	0.157	0.017	0.909	-0.144	0.328	-0.034	0.818
ICD	-0.145	0.325	-0.002	0.991	0.031	0.832	0.164	0.264
NCD	-0.113	0.443	0.16	0.276	0.118	0.423	0.243	0.096
TCD	-0.236	0.106	-0.055	0.71	-0.004	0.978	0.193	0.189

r: correlation coefficient, p >0.05: non-significant, SPFT: superior parafoveal thickness, IPFT: inferior parafoveal thickness, NPFT: nasal parafoveal thickness, TPFT: temporal parafoveal thickness, SRT: superior retinal density, IRD: inferior retinal density, NRD: nasal retinal density, TRD: temporal retinal density, SCT: superior choroidal thickness, ICT: inferior choroidal thickness, NCT: nasal choroidal thickness, TCT: temporal choroidal thickness, SCD: superior choroidal density, ICD: inferior choroidal density, NCD: nasal choroidal density, TCD: temporal choroidal density.

Table (6): Diagnostic accuracy and cut-off value of foveal thickness in differentiation between patients with proliferative diabetic patients and the rest of studied patients and controls.

AUC	SE	P	95% CI	Sensitivity	Specificity	Accuracy	Cut-off
0.837	0.06	0.000	0.72-0.954	87.5%	31.3%	74.2%	252 µm

AUC: area under the curve, SE: standard error, p <0.001: highly significant. The smallest cutoff value is the minimum observed test value minus 1, and the largest cutoff value is the maximum observed test value plus 1. All the other cutoff values are the averages of two consecutive ordered observed test values. The test result variable(s): Central Retinal Thickness has at least one tie between the positive actual state group and the negative actual state group.

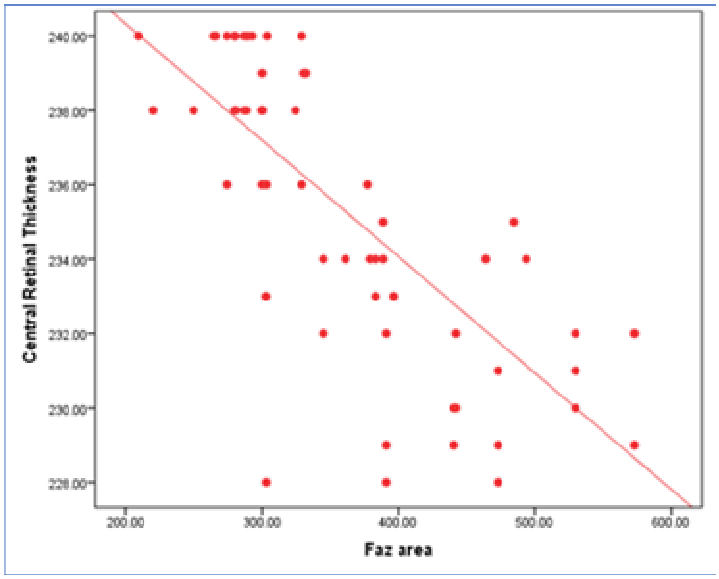

Fig. (1): Correlation coefficient (r) between FAZ area and central retinal (foveal) thickness.

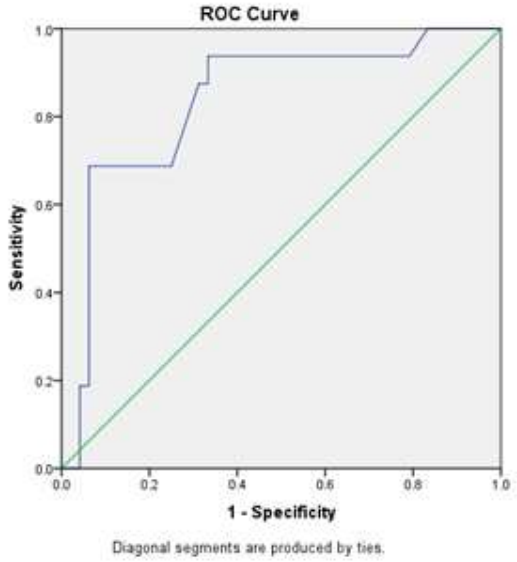

Fig. (2): ROC Curve displaying Diagnostic accuracy of central thickness to differentiate between patients with proliferative diabetic patients and the rest of studied patients and controls.

DISCUSSION

DR is a progressive microvascular disease and OCTA has the potential to increase our understanding of DR by giving high-resolution images of retinal and choroidal microvasculature blood flow and structure **[4]**. By using OCT and OCTA measurement for macular, choroidal thickness, FAZ, vessel density at SCP and choroidal density, our study noticed that there is decrease in choroidal thickness (CT) in diabetic patients.

In agreement with our study **Querques et al. [9]** identified choroidal thinning regardless of the disease stage, even in diabetic patients without DR. Also, **Sudhalkar et al. [10]** described an advanced thinning of CT with increasing severity of DR. **Regatieri et al. [11]** informed that CT decreased in eyes of PDR.

Most research, like ours, describes a steadily diminishing CT with increasing severity of retinopathy**[12,13].** A study by **Regatieri et al. [11]**reported that it is uncertain whether the choroidal thinning is primary or secondary to the retinal ischemia. According to this study, choroidal thinning predicts the beginning of retinal disease, and the thinning worsens as the retinopathy progresses.

Contrary to these findings, a hospital-based study from Korea by **Kim et al. [13]** reported an increased CT in patients with increasing severity of DR, and while the precise mechanism is unknown, there is conflicting evidence on the change in retinal blood flow and pulsatile ocular blood flow in subjects with diabetes **[14].**

In our study there is increase in FAZ area in diabetic patients, which agreed with **de Carlo et al. [15]; Takase et al. [16]**who reported increase FAZ in NDR. Also, **Kim et al. [17]; Hwang et al. [18]** reported statistically significant enlargement in patients with DR.

In contrast to our study, **Scarinci et al. [19]** did not find differences between type1DM patients with NDR and normal controls in FAZ area of both superficial and deep capillary plexus.

In our study there was decrease in vessel density at the superficial capillary plexus. **Kim et al. [20]**had noticed progressively decreasing capillary density, branching complexity, and progressively increasing average vascular caliber in eyes with different stages of DR which was similar to our findings, however, they were unable to detect a significant difference in these variables between healthy subjects and patients with mild NPDR and significantly reduced density in the superficial vascular plexus in mild NPDR in comparison to controls, as **Agemy et al. [2]** observed.

Also, in a cross-sectional study done by **Dimitrova et al. [22]**, in which 33 control subjects and 29 patients with NDR were joined, diabetic eyes were noted to have lower parafoveal superficial and deep retinal vascular density than healthy patients.

In our study there is decrease in choroidal vascular density which was similar to **Nagaoka et al. [2004]** that demonstrated a decreased choroidal blood flow, even before visible DR was present. Furthermore, a prior study reported that patients with background DR had considerably lower choroidal circulation, as estimated by colour Doppler imaging of the posterior ciliary arteries**[23].**

Schocket et al. [24]and **Nagaoka et al. [14]**suggested that due to retinal tissue hypoxia and over expression of vascular endothelial growth factor (VEGF), choroidal hypoperfusion may trigger the development of DR. They informed that choroidal volume and choroidal blood flow were significantly decreased in patients with PDR.

In our study there was a reduction in retinal thickness in diabetic patients and there was no significant differences in the retinal thickness between control subjects and patients with NDR. Consistent with our study, **Di et al. [25]** reported that there were no significant differences in the retinal thickness between control subjects and NDR

patients, recommending that retinal vascular changes occur before retinal structural changes.

Reduced retinal thicknesses in diabetic patients reflected neuro-degenerative changes such as reactive gliosis, decreased retinal neuronal function and neural-cell apoptosis, which have been observed to occur before obvious microangiopathy in experimental models of diabetic retinopathy and in diabetic donors' retina **[26, 27]**. Furthermore, in murine experimental models, there was progressive thinning of the inner retina over time (as assessed by OCT) **[28]**.

Variations in retinal thickness produced by diabetes are not totally understood. Former studies have observed that diabetics with limited or no DR had diminished retinal thickness as compared to non-diabetic persons**[29,30]**. In contrast, other researchers have discovered a rise in retinal thickness in those with advanced DR**[31,32].**

There were some limitations to our study:the16 eyes per group is a relatively small number. Because we measured the choroidal thickness using the manual method, the results might contain slight errors and this was the best clinical method currently available with the current OCT equipment, we tried to mitigate this by taking 2 choroidal measurements for the same choroidal point.

Because choroidal imaging was not performed at a specified time of day, we cannot rule out the effect of diurnal variation on CT as previously reported.**[33]**.OCT angiography, as is well known, has concerns with numerous artifacts, and artifacts emerge more commonly in eyes with impaired vision and retinal disorders. **[34].**We excluded images of OCT angiography with low image quality or diabetic macular edema, which may have introduced selection bias.

CONCLUSION

The results of this study recommended that OCTA could identify preclinical DR before the manifestation of clinically apparent retinopathy. They emphasize the potential role of OCTA in examining and quantifying retinal vascular alterations in diabetes.

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