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Influence of Diabetes on Macular Thickness Measured Using Optical Coherence Tomography: Iraqi Study

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ABSTRACT

Background*: DMO refers to the thickening of the central retina and is linked to long-term vision loss of individuals suffering from DR. Newly, the use of intravitreal injections of antiangiogenic medications has been explored as a potential method to enhance vision in individuals with MO resulting from DR. OCT utilizes optical reflectivity to visualize retinal thickness and structure, producing both cross-sectional and three-dimensional images of the macula. It is already extensively utilized due to its ability to offer an objective and quantitative evaluation of MO, in contrast to the subjective nature of fundus biomicroscopic assessment, which is commonly employed by ophthalmologists as an alternative to photography. OCT is additionally utilized for the quantitative monitoring of the treatment effects on CSMO. The operation of OCT is analogous to ultrasound B-mode imaging, except that light is used instead of acoustic waves. In this study, we used OCT to quantify retinal thickness in DM patients who do not exhibit maculopathy & patients with various degrees of DR with MO. We evaluated the validity of OCT measurements of macular thickness for evaluating the existence of MO. We also evaluated the relationship between the retinal thickness at the central fovea and BCVA.* ***Purpose:*** *To assess impact of DM on macular thickness as evaluated through OCT in a population-based sample.* ***Methods:*** *In this cross-sectional study, 205 diabetic patients participated; 110 with MO & 95 have no MO, all underwent full ophthalmic examination, with VA, refraction, fundus examination and then send for OCT examination& HbA1C.* ***Results:*** *The age of diabetic participants was range from 20-79 years, with 53.7% females and 46.3% males. Diabetic participants with moderate or severe DR with MO had greater macular thickness compared with those with no DR.* ***Conclusion:*** *In summary, our findings offer population-based data that will aid in interpreting macular thickness values in individuals with diabetes. We have demonstrated that measurements of macular thickness are increased in cases of DMO.*

Keywords: DM, DMO, OCT, HbA1C.

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INTRODUCTION

Diabetes mellitus (DM) is classified according to its pathogenic mechanisms that lead to hyperglycemia. There are two main classifications of DM, referred to as type 1 and type 2 DM. Type 1 DM arises from an autoimmune response targeting the insulin-producing beta cells, which results in a complete or nearly complete deficiency of insulin. In contrast, type 2 DM includes a wide array of disorders characterized by different degrees of insulin resistance, impaired insulin secretion, and elevated hepatic glucose production.[1]

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DM is a global pandemic disease. As of 2010, more than 200 million individuals had been diagnosed with diabetes, and this number is predicted to increase by 62% by 2025 [2].

This rise can be attributed to a surge in obesity, coupled with heightened life expectancy of the global population. DM complications include macroangiopathy (myocardial infarction or vasculocerebral stroke) and microangiopathy (diabetic nephropathy, neuropathy, and retinopathy). DR is the primary reason for blindness in Europe [3], impacting 1.9% of patients with DM [4].

Furthermore, 2.64% of diabetic patients have visual-threatening DR. The major cause of visual impairment in DM patients is DMO, with an annual incidence of 2.19%. DMO is a consequence of DR in the macular area and is secondary to retinal barrier rupture, which is in turn secondary to a range of metabolic changes brought about by hyperglycemia [5].

DR is a significant complication linked to DM, which continues to be a primary cause of vision loss among working-age individuals. The diagnosis of DR is established through the clinical presentation of vascular irregularities in the retina. Clinically, DR is categorized into two stages: NPDR and PDR. NPDR signifies the initial phase of DR, where increased vascular permeability and capillary occlusion are the two principal findings in the retinal vasculature during this period. Retinal pathologies such as microaneurysms, hemorrhages, and hard exudates can be identified through fundus photography, even though patients may remain asymptomatic.

PDR, the more advanced stage of DR, is marked by neovascularization. In this stage, patients may suffer from significant vision impairment when the newly formed abnormal vessels bleed into the vitreous, leading to VH, or when TRD occurs.

The predominant cause of vision loss in individuals with DR is DMO which is characterized by the swelling or thickening of the macula due to the collection of fluid both sub-retinally & intra-retinally, which is triggered by the disruption of BRB. [**6**]. DMO can occur at any stage of DR and cause distortion of visual images and decrease in VA. Current therapeutic modalities for DR aim at managing the microvascular complications, including intravitreal pharmacologic agents, laser photocoagulation and vitreal surgery. Intravitreal administration of anti-VEGF agents is currently the mainstay of therapy for both early and advanced stages of DR. [**7**].

OCT enables obtaining the high resolution (few micrometres) cross-sectional images (tomograms) of the human retina in a noninvasive manner [**8**]. Retinal morphology is reconstructed based on the analysis of backscattered or reflected light. In contrast to classic fundus photography taken with fundus camera, OCT also provides information on the depth that the scattered light comes from. If light is reflected by the deeper retinal layers, it has to go a longer way to return to the detector compared to the light reflected from more superficial layers. That is why it takes the light longer to return from deeper layers. This feature makes it possible to precisely determine what retinal depth (i.e., layer) the particular signal comes from. Therefore, OCT resembles ultrasound imaging with the only difference consisting in utilizing light instead of sound. The use of light gives OCT higher axial resolution compared to any other imaging techniques currently used in clinical medicine. The OCT technique described above is referred to as Time Domain OCT (TDOCT) due to the fact that the information on retinal morphology along the scanning beam is obtained by recording the optical signal when the mirror is moved. It was the first OCT technique described in 1991.

An alternative solution is Frequency Domain OCT (FDOCT). It differs from TDOCT in how the sample image is

constructed. There are two practical implementations of FDOCT. The first is Spectral Domain OCT (SDOCT) in which interferometric signal is detected using the spectrometer equipped with a line of light sensitive elements [**9**]. The other method is a Swept Source OCT (SSOCT) utilizing swept tunable lasers and a standard photodiode detector [**10**]. This makes it possible to achieve several hundred-fold increases in speed and sensitivity of scanning compared to TDOCT. As a result, significant motion artefacts are avoided and multiple measurements can be taken in a short time enabling the three-dimensional retinal scanning. The OCT images can be also acquired at the video-rate and the measured structures observed in real time. The first commercially available SDOCT device was launched in 2006. [11]

METHODS

A cross-sectional study was carried out at the Specialized Center for Endocrinology and Diabetes located in Baghdad-Iraq. The study was conducted from the period of October 2024 to August 2025. 205 patients participated; 110 with DR and MO & 95 have no MO.

All patients underwent a thorough ophthalmic examination by trained ophthalmologists and optometrists comprising ophthalmic history, UCVA and BCVA. The VA testing was done using Snellen's (E letter) chart. Fundoscopic examination were carried out after pupillary dilatation with topical tropicamide eyedrop. Then send for OCT examination& laboratory investigation (HbA1C).

The findings were recorded in a self-designed proforma consisting of the patient profile, VA, refractive errors of both eyes, fundus examination result &OCT findings.

STATISTICAL ANALYSES

Version 26 of IBM impact of various factors on study's parameter was assessed using statistics for the statistical package for the social sciences {SPSS}. Frequencies and percentage calculate for age, sex, macular status, VA, HbA1C and duration of diabetes. A chi-squared test was used for calculation coefficient correlation. That was consider significant when $p \leq 0.05$.

RESULTS

This study includes 205 diabetic patients, 110 with MO & 95 have no MO. The age of them was range from 20-79 years, with 53.7% females and 46.3% males.

Table 1: Shows the distribution of variables (Age, sex, macular status, VA, HbA1C& duration of diabetes) and their percent.

Variables		Frequency	Percent
Age	**20-29 years**	**7**	**3.4%**
	30-39 years	**13**	**6.3%**
	40-49 years	**27**	**13.2%**
	50-59 years	**65**	**31.7%**
	60-69 years	**66**	**32.2%**
	70-79 years	**27**	**13.2%**
Sex	**Male**	**95**	**46.3%**
	Female	**110**	**53.7%**

Variables		Frequency	Percent
Macula	DMO	110	53.7%
	Normal macula	95	46.3%
VA	UNVA	116	56.6%
	BCVA	89	43.4%
HbA1C	High	140	68.3%
	Normal	65	31.7%
Duration	1-10 years	72	35.1%
	>10 years	133	64.9%

In table 2 shows Chi-Square Test of correlation between macular thickness and other variables (age, VA, HbA1C& duration of DM), and reveals significant association between macular thickness and these variables.

Table 2: Chi-Square Test of correlation between macular thickness & other variables.

Variables		Macula			
		Diabetic Macular Oedema	Normal Macula	Total	Chi-Square Test
Age	20-29 years	3	4	7	.002
	30-39 years	2	11	13	
	40-49 years	8	19	27	
	50-59 years	39	26	65	
	60-69 years	40	26	66	
	70-79 years	18	9	27	
VA	UCVA	93	23	116	< .001
	BCVA	17	72	89	
HbA1C	High	93	47	140	< .001
	Normal	17	48	65	
Duration	1-10 years	18	54	72	.001<
	>10 years	92	41	133	

In table 3 shows Chi-Square Test correlation between VA and other variables (HbA1C &duration of DM) & reveals significant association between them.

Table 3: Chi-Square Test of correlation between BCVA& other variables.

Variables		VA			
		UCVA	BCVA	Total	Chi-Square Test
HbA1C	High	87	53	140	.018
	Normal	29	36	65	
Duration	1-10 years	30	42	72	.002
	10 years>	86	47	133	

DISCUSSION

In our study, we discovered that individuals of DMO exhibited greater thickness of macula in contrast to those without MO.

Table 2 illustrates a notable relationship between advancing age & macular thickness(Chi-Square Test is .002). In addition, there is significant correlation between VA status & DMO(Chi-Square Test is < .001) which shows increased in numbers of UCVA between DMO patients. There is increased in numbers of high HbA1C& longer duration of DM (>10 years) between DMO patients(Chi-Square Test is < .001).

In table 3 shows VA is significantly correlated with both HbA1C & duration of DM (Chi-Square Test is .018, .002) respectively. We showed that older age is associated with increased OCT measurements of the fovea as this supports the findings of Kashani et al,(12). In our study, HbA1C levels significantly affect macular thickness measurements which agreed with previous studies have reported increased macular thickness with higher HbA1C levels Chou et al,(13). Also, in our study duration of DM significantly affect macular thickness which disagreed with Chou et al.

CONCLUSION

In summary, our results provide population-based data that would be useful in the interpretation of macular thickness values in persons with diabetes. We have shown that macular thickness measurements are increased in DMO.

REFERENCES

1. Jameson JL, Fauci AS, Kasper DL, Hauser SL, Longo DL, Loscalzo J Harrison's Principles of Internal Medicine 20th ed New York (USA) McGraw Hill Education 2018 Alvin CP, Kevin DN, Carmella EM. Chapter No. 396 Diabetes Mellitus: Diagnosis, Classification, and Pathophysiology; 2850.

2. International Diabetes Federation. IDF Diabetes Atlas. 6th. Brussels, Belgium: International Diabetes Federation; 2013. http://www.idf.org/diabetesatlas. [Pub Med] [Google Scholar]

3. Bourne R. R. A., Stevens G. A., White R. A., et al. Causes of vision loss worldwide, 1990-2010: A systematic analysis. The Lancet Global Health. 2013;1(6):e339–e349. doi: 10.1016/S2214-109X(13)70113-X. [DOI] [PubMed] [Google Scholar]

4. Romero-Aroca P., De La Riva-Fernandez S., Valls-Mateu A., Sagarra-Alamo R., Moreno-Ribas A., Soler N. Changes observed in diabetic retinopathy: eight-year follow-up of a Spanish population. British Journal of

Ophthalmology. 2016;100(10):1366–1371. doi: 10.1136/bjophthalmol-2015-307689. [DOI] [PMC free article] [PubMed] [Google Scholar]

5. Romero-Aroca P. Targeting the pathophysiology of diabetic macular edema. Diabetes Care. 2010;33(11):2484–2485. doi: 10.2337/dc10-1580. [DOI] [PMC free article] [PubMed] [Google Scholar]

6. Romero-Aroca, P.; Baget-Bernaldiz, M.; Pareja-Rios, A.; Lopez-Galvez, M.; Navarro-Gil, R.; Verges, R. Diabetic macular edema pathophysiology: Vasogenic versus inflammatory. *J. Diabetes Res.* **2016**, *2016*, 2156273. [**Google Scholar**] [**CrossRef**] [**PubMed**]

7. Gonzalez, V.H.; Campbell, J.; Holekamp, N.M.; Kiss, S.; Loewenstein, A.; Augustin, A.J.; Ma, J.; Ho, A.C.; Patel, V.; Whitcup, S.M.; et al. Early and long-term responses to anti-vascular endothelial growth factor therapy in diabetic macular edema: Analysis of protocol I data. *Am. J. Ophthalmol.* **2016**, *172*, 72–79. [**Google Scholar**] [**CrossRef**] [**PubMed**]

8. Wojtkowski M., Srinivasan V., Fujimoto J. G., Ko T., Schuman J. S., Kowalczyk A., and Duker J. S., Three-dimensional retinal imaging with high-speed ultrahigh-resolution optical coherence tomography, *Ophthalmology*.(2005) 112,no.10, 1734–1746,2-s2.0-25844456000, **https://doi.org/10.1016/j.ophtha.2005.05.023**.

9. Wojtkowski M., Leitgeb R., Kowalczyk A., Bajraszewski T., and Fercher A. F., In vivo human retinal imaging by Fourier domain optical coherence tomography, *Journal of BiomedicalOptics*.(2002) **7**,no.3, 457–463,2-s2.0-0036629502, **https://doi.org/10.1117/1.1482379**.

10. Chinn S. R., Swanson E. A., and Fujimoto J. G., Optical coherence tomography using a frequency-tunable optical source, *Optics Letters*. (1997) **22**, no. 5, 340–342, 2-s2.0-0000063301.

11. Brar M., Bartsch D.-U. G., Nigam N., Mojana F., Gomez L., Cheng L., Hedaya J., and Freeman W. R., Colour versus grey-scale display of images on high- Resolution spectral OCT, *British Journal of Ophthalmology*.(2009) **93**,no.5, 597–602,2-s2.0-66149106748, **https://doi.org/10.1136/bjo.2008.146233**.

12. Kashani A, Zimmer-Galler I, Shah S, Dustin L, Do DV, Eliott D et al. Retinal thickness analysis by race, gender, and age using Stratus OCT. Am J Ophthalmol 2010; 149: 496–502.

13. Chou TH, Wu PC, Kuo JZ, Lai CH, Kuo CN. Relationship of diabetic macular oedema with glycosylated haemoglobin. Eye 2009; 23(6): 1360–1363.

