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Relationship of diabetic macular oedema with glycosylatedHaemoglobin-OCT based approach

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Abstract:***Purpose*** *To evaluate the correlation betweenglycosylated haemoglobin (HbA1c) andcentral foveal thickness as measured byoptical coherence tomography (OCT) in patients with diabetes.****Methods*** *Observational study of central foveal thicknessas measured by OCT and laboratory dataof glycosylated haemoglobin. HbA1cwas compared with foveal thicknessmeasured by OCT within the preceding 3months. Clinically significant macularoedema (CSME) was diagnosed if centralfoveal retinal thickness was greater than325 µm in OCT.****Results*** *One hundred and two eyes of 102patients were included in this cross-sectionalstudy. The analysis revealed that theCSME diagnosed by OCT in diabetes was notstatistically significant with sex, right or lefteye, DM duration > 10 years or not, and ACsugar level (> 140 or not). The HbA1C level(≥8) and age (≤50) showed a significant (P=0.005 and 0.006, respectively)and positive association with macularthickness in OCT. A trend towards higher riskwas seen for factors of age ≤50 and HbA1c≥8%.Conclusions Patients with HbA1c of ≥8 had an increase in macular thickness in type 2 diabetic eyes and there was a statistical significant correlation between younger age,shorter DM duration and thicker macularthickness. Strict sugar control decreased therisk of diabetic macular retinopathy, and OCTcould be an excellent detector of early diabeticmacular oedema.*
Keywords*:centralfoveal thickness,HbA1c, diabetic macular oedema,optical coherence tomography*

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I. Introduction

Diabetic Macular oedema is defined as retinal thickeningwithin 2 disc diameters of the centre of themacula, causing leakage of plasma constituents intothe surrounding retina due to microvascular changesin the blood retinal barrier and ultimately leading toretinal oedema[1]. CME is a cause of severe visual loss that occurs in a variety of pathologic conditions, such as age-related macular degeneration, diabetic retinopathy, branch or central retinal vein occlusion, epiretinal membrane or vitreomacular traction, and as a complication of intraocular surgery[2]. CME is a pathologic definition with two components: abnormal collection of extracellular fluid and cystoid-space formation[3]. Diabetic macular edema is classifiedin focal and diffuse types and this is importantbecause the treatments of the two types are different.Focal edema is caused by leakage from micro aneurysmsand is associated with hard exudates rings.Diffuse edema is caused by leakage from retinal capillariesand arterioles. Two types of laser treatmentfor DMO are focal and grid. Focal laser treatment isused to treat focal diabetic macular edema; the purposeis to close the leaking micro aneurysms. Gridlaser is used to treat diffuse macular edema and is applied in areas of retinal thickening with diffuse leakage.[1]The Wisconsin Epidemiology Study of Diabetic Retinopathy(WESDR) in 1995 showed that there is anincrease in diabetic macular edema in patients withincrease HbA1c[4].

Diabetic macular edema increases with the duration of diabetes, and the prevalence is 5% within the first 5 years after diagnosis and 15% at 15 years.[5]Flouresceinangiography is indicated in guiding treatment of macular oedema[6]. Withthe help of optical coherence tomography(OCT), it is now possible to measure themacular thickness objectively and to follow theprogression of DME quantitatively[7].With the advent of OCT, several investigators have classified DME on the basis of the retinal map and cross-sectional appearance of the retina on OCT[8].Spectral domain (SD) OCT allows for better characterization of the retinal morphology. The morphological patterns of DME on OCT are generally classified into diffuse retinal thickening, cystoid macular edema (CME), subretinal detachment, and vitreomacular interface abnormalities[9].OCT, first described by Huang et al[10] in 1991, is an imaging modality capable of providing high-resolution cross-sectional images of the neurosensory retina. As OCT is noninvasive, it was quickly adopted by clinicians for the assessment of patients with CME. In part, because of the relative ease with which they can detect CME, many clinicians now prefer OCT over FA for its detection[11].OCT has gained increasing popularity as an objective tool to measure retinal

thickness and other aspects associated with macular edema[12]. An advantage of using OCT is its quantitative assessment, rather than the qualitative evaluation performed with photography or biomicroscopy[7].
Periodicglycosylated haemoglobin (HbA1c)measurements can reflect the long-term controlof hyperglycaemia. Intensive glycemic controlhad been proved to be effective in decreasingincidence rate of development and progression of DR in type 1 and type 2 diabetic mellitus asdemonstrated by the diabetes control andcomplications trials[13] and the United KingdomProspective Diabetes Study[14].

II. Materials and methods

It is a observational study conducted in 102 patients attending the ophthalmology OPD between aug 16-May 18.Inclusion criteria: (1) received complete ophthalmicevaluation; (2) had HbA1c measured by specific highpressureliquid chromatography methods; and (3)received OCT examination (TOPCON) within 3 months precedingHbA1c measurement. OCT was performed in both eyes,but the eye with thicker macular oedema was used forstatistical analysis. Oct images of few patients of CSME are shown in fig1,2,3.Exclusion criteria included patientswho received intraocular surgery (cataract surgery, parsplanavitrectomy, intravitreal injection of triamicinoloneor Bevacizumab), subtenon injection, orphotocoagulation therapy within 1 year of evaluationand severe vitreous haemorrhage or vitreous opacity thatwould interfere with the OCT examination. Clinicallysignificant macular oedema (CSME) was diagnosedaccording to central macular retinal thickness >325 µm in OCT[15].
Statistical analysis- Pearson's correlation coefficient was used to find out the relationships betweenage, duration of diabetes, HbA1c level, AC sugar level,centralfoveal thickness. P value <0.05 was considered statisticallysignificant. All patients were divided into 2 groupswith no CSME and those with CSME.

III. Results

One hundred and two eyes of 102 patients were includedin this cross-sectional study. 63 patients weremale and 39 were female. The mean age±SD was62.3±8.1 years(range,40–77 years). The mean DMduration was 11.2±5.5 years (range, 1–30). Themean value of HbA1c was 7.8±1.4% (range, 5.1–12.1%).The mean central retinal thickness was 257.1±79.3 mm (range, 151–526 mm) shown by Table1

Table 1 Demographic & clinical data of study population

Parameters	observation
Total number of included	102
Male : female ratio	1.61:1
Mean age (years)	62.3±8.1
Mean DM duration (years)	11.2±5.5
Mean HbA1C (%)	7.8±1.4
Mean central retinal thickness (µm)	257.1±79.3

Table 2 shows the distribution of possible risk factorsfor CSME diagnosed by OCT among patients with diabetes and results of our study.

Table 2- Analysis of risk factors associated with OCT-based CSME in diabetic eyes

Factor	Definition	No CSME (OCT<325 µm) No. (%)	CSME (OCT≥325 µm) No. (%)	P value
Age	>50	81(95.3)	12(70.6)	0.006*
	≤50	4 (4.7)	5(29.4)	
Sex	Male	52(61.2)	11(64.7)	0.78
	Female	33(38.8)	6(35.3)	
Laterality	Left	49(57.6)	11(64.7)	0.59
	Right	36(42.4)	6(35.3)	
DM duration (years)	<10	26(30.6)	8(47.1)	0.18
	≥10	59(69.4)	9(52.9)	
AC sugar (mg/100 ml)	<140	28(40.6)	4(26.7)	0.315
	≥140	41(59.4)	11(73.3)	
HbAlc	<8	56(65.9)	5(29.4)	0.005*
	≥8	29(34.1)	12(70.6)	

This study revealed that the CSME diagnosed by OCT in diabetes was not statistically significant withsex (P=0.78), right or left eye (P=0.59), DM durationover 10 years or over (P=0.18), and AC sugar level (over140 or not) (P=0.315). The HbA1C level (8 or over) andage (50 or less) showed a significant (P=0.005 and 0.006,respectively) and positive association with macularthickness in OCT.

IV. Discussion

The inclusion of HbA1cinthescreeningprotocolshasgained significant importance, not only in the diagnosis and management of DM, but also in the on set and progression of DR [16]. It has been seen that compared to the measurement of glucoselevels,HbA1cassayisatleastasgoodindefining the level of hyperglycemiaat which the prevalence of DR increases [17,18]. Glycation of tissue proteins is a well-known patho-physiological mechanism in the complications related to diabetes leading to the formation of advanced glycation end products andHbA1c [19]. Tight glycemic control, as measured by these factors, is strongly associated with a decreased prevalence of micro-vascular complications related to DM like retinopathy [20-23]. Anitha et al. also found an association of advanced glycation index (AGI) with the severity of DR[24].

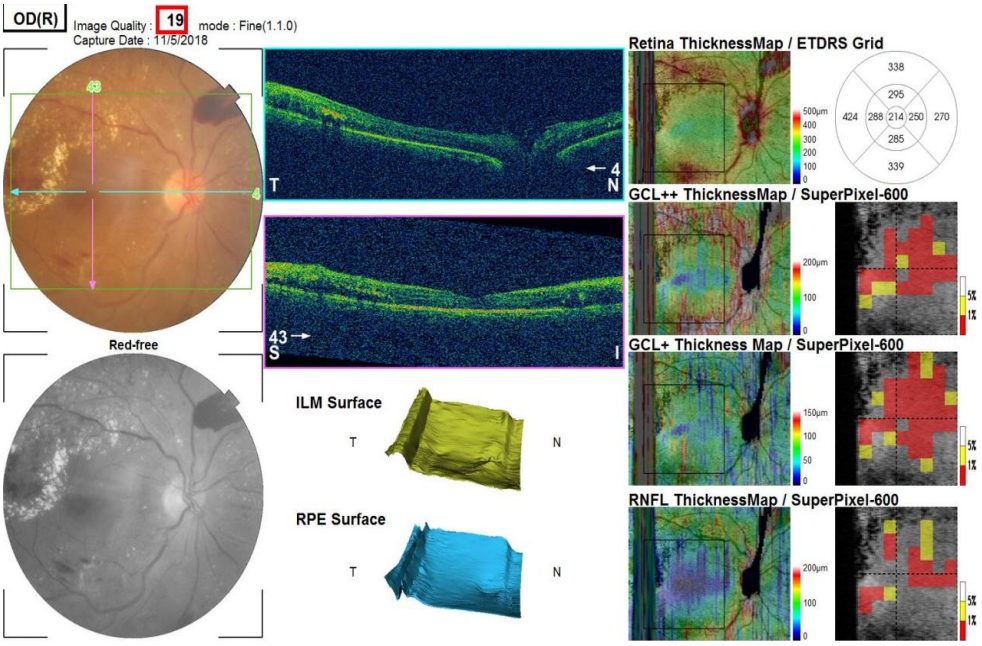

Figure 1- OCT 3D WIDE image showing subretinal fluid collection around macula with hard exudates (CME).

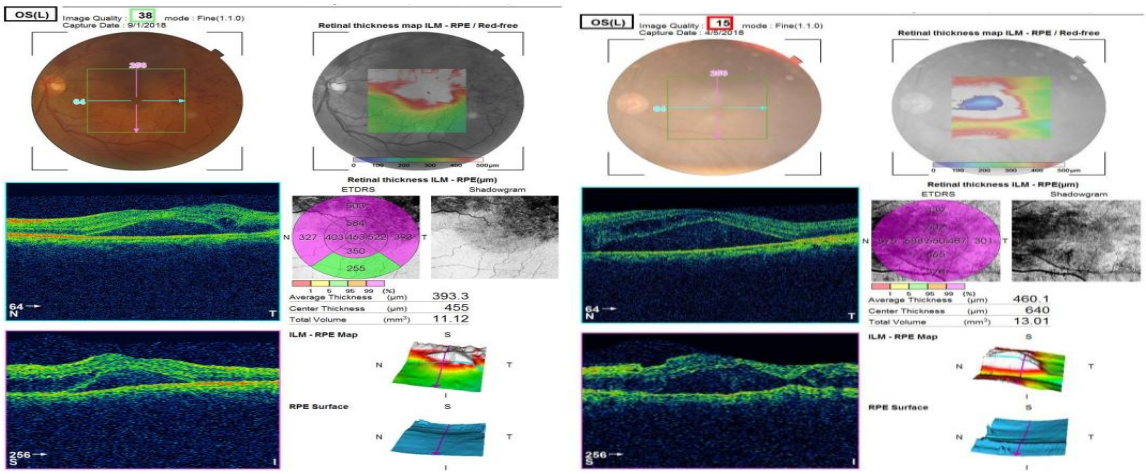

Figure 2 and 3 –OCT 3D MACULA showing increase in central macular thickness due to subretinal fluid collection (cystic spaces) around macula.

References

[1]. Antcliff RJ & Marshall J (1999): The pathogenesis of oedemain diabetic maculopathy. SeminOphthalmol 14: 223–232.
[2]. Johnson MW. Etiology and treatment of macular edema.Am J Ophthalmol. 2009;147:11–21.
[3]. Tso MO. Pathology of cystoid macular edema. Ophthalmology. 1982;89:902–915.
[4]. Klein R, Moss SE, Klein BE, et al. The Wisconsin EpidemiologicStudy of Diabetic Retinopathy. XI. The incidence ofmacular edema. Ophthalmology. 1989;96:1501–10.
[5]. Aiello LP, Gardner TW, King GL, et al. Diabetic retinopathy.Diabetes Care. 1998;21:143–156.
[6]. Diabetic retinopathy.American diabetes association.Diabetes Care 1998; 21: 157–159.
[7]. Hee MR, Puliafito CA, Wong C, Duker JS, ReichelE,Rutledge B et al. Quantitative assessment of macular edemawith optical coherence tomography. Arch Ophthalmol 1995;113: 1019–1029.
[8]. Kim NR, Kim YJ, Chin HS, Moon YS. Optical coherence tomographic patterns in diabetic macular oedema: prediction of visual outcome after focal laser photocoagulation. Br J Ophthalmol. 2009;93: 901–905.
[9]. Otani T, Kishi S, Maruyama Y. Patterns of diabetic macular oedema with optical coherence tomography. Am J Ophthalmol. 1999;127:88–93.
[10]. Huang D, Swanson EA, Lin CP, et al. Optical coherence tomography. Science. 1991;254:1178–1181.
[11]. Kozak I, Morrison VL, Clark TM, et al. Discrepancy between fluorescein angiography and optical coherence tomography in detection of macular disease. Retina. 2008;28:538–544.
[12]. . Diabetic Retinopathy Clinical Research Network [website on the Internet]. Available from: http://www.drcr.net. Accessed June 27, 2013.
[13]. The relationship of glycemicexposure (HbA1c) to the risk of development progression of retinopathy in the diabetes control: and complications trial. The Diabetes Control and Complications Trial Research Group.Diabetes 1995; 44: 968–983.
[14]. Intensive blood-glucosecontrol with sulphonylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes (UKPDS 33). UK prospective diabetes study (UKPDS) group. Lancet 1998; 352: 837–853.
[15]. Browning DJ, McOwen MD, Bowen Jr RM, O'MarahTL.Comparison of the clinical diagnosis of diabetic macular edema with diagnosis by optical coherence tomography.Ophthalmology 2004; 111: 712–715.
[16]. Krishnamurty U, Steffes MW. Glycohemoglobin: a primary predictor of the development or reversal of complications of diabetes mellitus. ClinChem 2001;47:1157–65.
[17]. Tapp RJ, Tikellis G, Wong TY, Harper C, Zimmet PZ, Shaw JE. Longitudinal association of glucose metabolism with retinopathy. Diabetes Care 2008;31:1349–54.
[18]. Sabanayagam C, Liew G, Tai ES, Shankar A, Lim SC, Subramaniam T, et al. Relationship between glycated haemoglobin and microvascular complications: is there a natural cut-off point for the diagnosis of diabetes. Diabetologia 2009 [Epub ahead of print].
[19]. Friedman Eli A. Advanced glycosylated end products and hyperglycemia in the pathogenesis of diabetic complications. Diabetes Care 1999;22(Suppl. 2):B65–71.
[20]. Nordwall M, Arnqvist HJ, Bojestig M, Ludvigsson J. Good glycemic control remains crucial in prevention of late diabetic complications – the Linko¨ ping Diabetes Complications Study. Pediatr Diabetes 2009;10:168–76.
[21]. McCance DR, Hadden DR, Atkinson AB, Archer DB, Kennedy L. Long term glycemic control and diabetic retinopathy. The Lancet 1989;2:824–8.
[22]. Schellhase KG, Koepsell TD, Weiss NS. Glycemic control and the risk of multiple micro-vascular diabetic complications.Fam Med 2005;37:125–30.
[23]. Wagener HP, Dry TJS, Wilder RM. Retinitis in diabetes. N Eng J Med 1934;211:1131–7.
[24]. Anitha B, Sampathkumar R, Balasubramanyam M, Rema M. Advanced glycation index and its association with severity of diabetic retinopathy in type 2 diabetic subjects. J Diabet Complications 2008;22:261–6.

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