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TO EVALUATE SUB FOVEAL CHOROIDAL THICKNESS (SFCT) IN UNCONTROLLED DM EYES USING ENHANCED DEPTH OCT (EDI-OCT).

Ophthalmology

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ABSTRACT

Purpose: To evaluate Sub Foveal Choroidal Thickness in uncontrolled DM eyes using Enhanced Depth OCT. **Method-** We study possible effects of sugar levels or HbA1c values with choroidal thickness(CT) in uncontrolled DM and its possible correlation to BCVA using EDI-OCT.Complete ophthalmic examinations were performed one or two days before EDI-OCT imaging,with BCVA documented using 4m ETDRS charts in Snellen fraction and fundus examination to grade DR. **Results-** 37 patients with HbA1c level between 7.1 - 8 (including 8) had mean SFCT of 257.05 with SD of 18.10 and 13 patients with HbA1c more than 8 had SFCT of 231.61 with SD of 11.38. Paired t-test showed statistically significant difference between SFCT of two groups, with p value <0.001.It was found that SFCT is inversely correlated with HbA1c levels,with Paersons' co-efficient being -0.68. 14 patients had CME of 300 or more in one or both eyes with mean average of 285.6.The correlation between BCVA and SFCT was 0.37 and 0.42 for RE and LE respectively,using Paerson's coefficient.13 patients had vision 6/60 or less in one or both eyes but role of choroidopathy couldn't be confirmed because of confounding factors like CME and senile cataract. **Conclusion-** Our study reveals an inverse correlation with significantly thinner SFCT in diabetic patients with moderate or poor glycemic control (HbA1c > 8%).Therefore,better diabetic control with HbA1c ≤ 7% may prevent choroidal vascular damage and choroidal thinning and finally prevent development and/ or progress of DR and vision loss.It is therefore mandatory to evaluate Choroid in all diabetics.

KEYWORDS

Diabetic Retinopathy, Sub Foveal Choroidal Thickness, Uncontrolled HB1AC.

INTRODUCTION

Diabetic retinopathy (DR) is one of the major causes of preventable visual loss.[1] DR is a multifactorial with many risk factors like blood sugar levels ,HbA1c ,Hypertension, and serum lipid levels. Pathophysiologically DR has primarily been considered a microvascular disorder leading to degeneration of neuronal and glial structures of inner retina as disease progresses however, choroidal vascular damage may have an important role. [2] Choroidal alterations also play a key role in pathophysiology of retinal diseases, including DR. [3,4,5,6] Choroidal thickness (CT) is considered a measure of choroidal blood flow,which supplies outer retinal layers(ORL).Delayed filling of choroidal vessels using ICG angiography has been reported in eyes with DR. [7,8,9] Moreover, reduction in choroidal blood flow and volume has been demonstrated in eyes with both NPDR and PDR using Doppler flowmetry. [10] Choroidal vascular changes, including choroidal aneurysms, vessel narrowing and tortuosity, CNV, and non-perfusion may occur in eyes with DR. These can alter Subfoveal Choroidal thickness (SFCT) which in turn might have effects on visual acuity. [11,12]

Optical coherence tomography (OCT) allows high resolution *in vivo* imaging of posterior segment.[13] Nowadays,improvement of enhanced depth imaging (EDI) software permits highly reliable measurement of SFCT.Being a non-invasive modality, is a useful modality of measuring SFCT. [14,15]

Certain studies have aimed to study changes in SFCT with type of retinopathy as well as compare differences between diabetics and non-diabetics.We study possible effects of sugar levels or HbA1c values with SFCT in uncontrolled diabetic patients and its possible correlation to visual acuity; using EDI-OCT.

Methods

Patients willing and co-operative with known DM,irrespective of type,were enrolled in our cross sectional observational study if they had uncontrolled sugar levels – FBS > 100 mg% or PLBS > 250 mg% and HbA1c > 7.1.

The exclusion criteria were patients with any pathology obscuring fundus visualization,those unable to focus well at OCT,nystagmus, etc, age > 65 years,any intraocular surgery or laser intervention and with any ocular pathology suspected to alter CT– glaucoma, high myopia, ARMD and variants, intraocular trauma, etc.

The current study was approved by Ethics Committee of our institute and was performed in agreement with ethical principles of Declaration of Helsinki. Informed consent was obtained.

All subjects included in study underwent an OCT scan on same day as blood investigations using Zeiss Cirrhus SD OCT, including macular cube scan, HD 21 line raster and EDI HD 21 line raster modes for BE. CT was defined from outer edge of hyperreflective line corresponding to RPE to inner surface of sclera.SFCT was manually measured and averaged by two independent observers from horizontal and vertical scans.Repeatability of SFCT measurements between scans and observers were analyzed.All OCT scans were performed by same experienced operator between 11 and 11:45 am to avoid potential CT changes with circadian rhythm.Complete ophthalmic examinations were performed one or two days before EDI-OCT imaging,with BCVA documented using 4m ETDRS charts in Snellen fraction and fundus examination for DR.

STATISTICAL ANALYSIS

Data were analyzed using Excel and Graph Calculator online tools.Mean ± SD was reported for quantitative variables.Percentage was reported for qualitative variables. A correlation test (Pearson coefficient when variables had normal distribution or Spearman coefficient when distribution of variables was not normal) was used to evaluate correlation of SFCT and quantitative variables, such as HbA1C, duration of diabetes, etc.Paired sample t-test was used to compare variables within each groups such as comparison of SFCT between RE and LE. P-value < 0.05 was considered significant.

RESULTS

A total of 50 Pts, 100 eyes, with uncontrolled diabetes (Hb1Ac >7.1 %) were enrolled in this study.There were 30 males and 20 females.Mean age of males and females were 55 and 54. Mean duration of diabetes was 13 years.Mean SFCT was 250.44 with SD of 19.76, males 248.75 and females 253.7. 5 pts with no retinopathy at presentation had a mean SFCT of 271.7. 16 pts with mild NPDR had a mean SFCT of 249.2 .13 pts with moderate NPDR had a mean SFCT of 248.3. 10 pts with severe NPDR had a mean SFCT of 235.75 . 6 pts with PDR at had a mean SFCT of 220.8

23 patient had visually significant cataract , out of those -

3 Had poor vision with Increased CMT, decrease SFCT , PDR
4 Had poor vision with Increased CMT, decrease SFCT,severe NPDR
8 Had poor vision decrease SFCT,moderate NPDR

5 Had poor vision decrease SFCT,mild NPDR
2 Had poor vision mild NPDR

14 patients were psuedophakic, Out of which ,
3 Had poor vision with Increased CMT, decrease SFCT, PDR .
3 Had poor vision, decrease SFCT,severe NPDR.
2 Had poor vision, increased CMT, decrease SFCT,severe NPDR.
4 Had poor vision, decrease SFCT,moderate NPDR.
2 Had poor vision, mild NPDR, out of which one patient had decrease SFCT and other one had normal SFCT

3 patients didn't have catarcact.
In this one patient had poor vision with CME , decrease SFCT with severe NPDR 2 others had decrease CMT with moderate NPDR ,near normal SFCT.

37 patients with HbA1c level between 7.1 - 8 (including 8) had mean SFCT of 257.05 with SD of 18.10 and 13 patients with HbA1c more than 8 had SFCT of 231.61 with SD of 11.38.

Paired t-test showed statistically significant difference between SFCT of two groups, with p value <0.001. It was found that SFCT is negatively correlated with HbA1c levels, with Pearson's co-efficient being -0.68.

14 patients had CME of 300 or more in one eye or both eyes with mean average of 285.6.The correlation between BCVA and SFCT was 0.37 and 0.42 for RE and LE respectively, using Pearson's coefficient. 13 patients had vision 6/60 or less in one or BE but role of choroidopathy couldn't be confirmed because of confounding factors like CME and senile cataract.

DISCUSSION

There is evidence to suggest that retinal neurodegeneration begins early in pathogenesis of DR [16]. The choroid provides oxygen and nutrients to ORL and is only blood supply of avascular fovea [17]. The association between CT and DR stage has been widely reported, Vusojevic et al. attributed this thinning to a relative vasoconstriction or decrease in perfusion pressure in choroidal vessels [18]. These findings suggest that changes in choroidal vasculature could be an early event in retina, even where there is no DR [19].The thinner CT observed in advanced stages of DR could be related to progressive damage of endothelium that results from this process,causing reduced flow and ischaemia.Poorly controlled HbA1c is associated with more severe stages of DR ,this may be due to lower choroidal blood flow and hypoxia in higher concentrations of blood sugar that ultimately may lead to choroidal thinning.Choroidopathy may play a major role in the pathogenesis of DR because the ORL are dependent on choroid for nutrition and oxygenation.

In this study, we found that SFCT was significantly low in patients with uncontrolled DM and choroid became thinner with increase in Hb1Ac level (Inverse correlation). We also found that patients with higher grade of retinopathy had thinner choroid, This finding is in agreement with Lee et al [15] who evaluated CT in diabetic patients using SD-OCT. They found that SFCT was less in eyes with NPDR or PDR than that in healthy non-diabetic eyes; however, there was no significant difference between diabetic eyes with no DR and normal control eyes. Sudhalkar et al [20] assessed SFCT changes in different types of DR. They showed that diabetic patients with or without DR had a significantly thinner choroid compared to non-diabetic control group. Moreover, they found that patients with PDR had a thinner sub-foveal choroid than patients with NPDR. The authors concluded that choroidal thinning continued with increasing stages of DR which is similar to our findings. A study Hamidreza Torabi1, MD et [21] all showed there was no difference in SFCT in diabetic with good glycemic control and non-diabetics.This study showed as Hb1Ac levels increased, SFCT decreased which is compatible with our results.High uncontrolled blood sugar levels in diabetics with inadequate treatment may lead to choroidal vascular damage and resultant thinning.Therefore, in patients with uncontrolled DM and high levels of HbA1c,choroid is expected to be thinner. Unsal et al reported a weak to moderate Inverse correlation between SFCT and HbA1c values, which is compatible with our results.[22]A previous study showed that abnormalities in choriocapillaris and reduction of choroidal blood flow occurred in early stages of DR,[23] which may lead to choroidal hypoxia,ischemia and finally choroidal thinning However, some other studies reported no significant difference in SFCT between diabetic and non-diabetic eyes.[24] Yazici and co-workers compared SFCT in diabetic patients with and without polyneuropathy and healthy control subjects.[25] They reported that central choroid is thicker in diabetic patients compared to normal subjects and also showed that there was additional choroidal thickening in diabetic patients with polyneuropathy. They concluded that choroidal thickening may be related to autonomic dysregulation secondary to diabetic neuropathy. Sahinoglu-Keskek et al classified 122 eyes of 122 diabetic patients into three study groups including diabetic patients without DR, with DR and no macular oedema (ME), and with DR and ME. [26] They reported that SFCT in patients with no DR was significantly thicker than that in patients with DR and without ME.Moreover, they reported that no significant differences in HbA1c levels were detected among three study groups and concluded that there was no correlation between SFCT and HbA1c values.

Study Strengths and Limitations

The present study has several limitations.Despite relatively large number of subjects with DM included in study, after stratification by degree of DR,numbers in each category were limited, especially in advanced stages.The refractive error of patients included in study was not recorded. Since myopic status could alter chorioretinal thickness, especially in high myopia,absence of data on refractive errors can also be added to confounding factors. Direct relation between visual acuity and SFCT could not be established as there were multiple confounding factors like senile cataract, CME, although CME could be an outcome of choroidopathy as the disease progresses.The possibility of measurement errors due to manual measurement and measurement by a single observer constitute other limitations.

CONCLUSION

In conclusion,our study reveals an inverse correlation with significantly thinner SFCT in diabetic patients with moderate or poor glycaemic control (HbA1c > 8%). Therefore,better diabetic control with HbA1c ≤ 7% may prevent choroidal vascular damage and thinning,and finally prevent development and/ or progress of DR and hence vision loss.However,future studies with larger sample size are required to establish correlation of SFCT with HbA1c and to determine optimal value of HbA1c that affects choroidal vascular system.It is therefore mandatory to evaluate Choroid in all diabetics.

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