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To study association of HBA1C and fasting serum lipids with central macular thickness in patients with type 2 diabetes mellitus

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Abstract
Purpose: To determine the relationship of serum HbA1c and fasting serum lipids with central macular thickness (CMT) in patients with Type 2 Diabetes Mellitus.
Methods: This cross-sectional descriptive study was conducted in 130 Type 2 diabetes mellitus patients attending the Ophthalmology OPD of Government Medical College, Kota. Patients underwent comprehensive ophthalmic evaluation including optical coherence tomography (OCT) for central macular thickness. Glycosylated hemoglobin (HbA1c) and fasting lipid profiles were recorded. Data were analyzed using SPSS-20 software; $p < 0.05$ was considered significant.
Results: Mean age of study subjects was 56.9 ± 8.3 years. Mean HbA1c was 8.28 ± 2.17%, total cholesterol 188.46 ± 60.64 mg/dl, triglycerides 184.07 ± 77.41 mg/dl, LDL 114.95 ± 43.29 mg/dl, and HDL 46.6 ± 9.84 mg/dl. Mean CMT in right and left eyes was 260.41 ± 101.66 µm and 255.69 ± 88.89 µm, respectively. CMT was significantly higher in patients with uncontrolled HbA1c (≥6.5%) compared to those with controlled HbA1c (<6.5%) (p=0.02). LDL cholesterol also showed a positive correlation with CMT, though not statistically significant.
Conclusion: Elevated HbA1c levels were significantly associated with increased central macular thickness in Type 2 diabetic patients, suggesting that strict glycemic control may prevent early macular changes before the onset of diabetic macular edema.

Keywords: HbA1c, Diabetic Retinopathy, Central Macular Thickness, OCT, Lipid Profile, Type 2 Diabetes Mellitus

Introduction

Diabetes mellitus (DM) represents a heterogeneous group of metabolic disorders characterized by chronic hyperglycemia due to defects in insulin secretion, insulin action, or both. The global prevalence of diabetes has reached epidemic proportions; the International Diabetes Federation (IDF) estimates more than 530 million adults currently live with diabetes, with India contributing one of the largest national burdens. Rapid urbanization, sedentary lifestyles, and increasing life expectancy have positioned India as the so-called "diabetic capital of the world."

Long-standing hyperglycemia damages microvascular beds throughout the body, leading to neuropathy, nephropathy, and retinopathy. Diabetic retinopathy (DR) is among the most significant microvascular complications and remains a major cause of preventable blindness in adults. The prevalence of DR among diabetics in India ranges between 18% and 28% depending on population studied. Structural retinal alterations—including capillary leakage, microaneurysm formation, and thickening of the macula—often precede symptomatic vision loss.

With the advent of optical coherence tomography (OCT), subtle morphologic changes in retinal architecture can now be quantified with micrometer precision. OCT enables non-invasive, cross-sectional imaging of retinal layers, providing objective measurement of central macular thickness (CMT). In patients with diabetes, CMT serves as an early indicator of macular involvement and can detect subclinical diabetic macular edema (DME) before ophthalmoscopic evidence appears.

Among biochemical markers, glycated hemoglobin (HbA1c)reflects average glycemic control over the preceding two to three months and is strongly correlated with the risk of developing microvascular complications. Several landmark trials—including the Diabetes Control and Complications Trial (DCCT) and the UK Prospective Diabetes Study (UKPDS)—have demonstrated that reducing HbA1c significantly decreases the incidence

and progression of DR. However, emerging evidence suggests that fluctuations and persistently elevated HbA1c levels may influence retinal thickness independently of clinical DR stage.

Additionally, dyslipidemia is increasingly recognized as a contributor to diabetic retinal pathology. Elevated serum cholesterol and triglyceride levels are associated with lipid exudation within the retina, breakdown of the blood-retinal barrier, and oxidative endothelial damage. High LDL-C and low HDL-C levels have been linked with formation of hard exudates and increased risk of clinically significant macular edema (CSME). The biochemical interplay between glycemic control, lipid metabolism, and retinal microvascular integrity remains an area of ongoing research.

Previous studies have yielded inconsistent results regarding the correlation between serum lipids, HbA1c, and macular thickness. Some investigators observed a positive relationship between HbA1c and CMT, while others reported no significant association between serum lipids and retinal thickness after adjusting for confounders. The paucity of data from the Indian population—where both diabetes prevalence and DR burden are high—necessitates further evaluation.

Materials and Methods

This was a hospital-based, cross-sectional observational study conducted in the Department of Ophthalmology, Government Medical College, Kota, Rajasthan. Data were collected prospectively over a period of 18 months after obtaining approval from the Institutional Ethics Committee.

A total of 130 patients with previously diagnosed type 2 diabetes mellitus attending the Ophthalmology outpatient department were recruited by consecutive sampling after informed written consent.

Inclusion Criteria are Patients aged 40 to 70 years with type 2 DM of at least one-year duration, Willingness to participate and provide informed consent, Clear ocular media allowing good-quality OCT imaging.

Exclusion Criteria are type 1 diabetes mellitus, co-existing retinal or choroidal disorders (e.g., vein occlusions, age-related macular degeneration), history of intraocular surgery or laser photocoagulation, ocular trauma, glaucoma, or significant cataract obscuring the fundus, systemic hypertension uncontrolled on medication, renal disease, or other systemic illnesses likely to affect retinal status.

Each participant underwent a detailed ophthalmic and systemic evaluation.

Best-corrected visual acuity (BCVA) was assessed using a Snellen chart. Performed slit-lamp biomicroscopy to rule out anterior segment pathology. After pupillary dilation with 1 % tropicamide and 5 % phenylephrine, fundus examination was done using indirect ophthalmoscopy and slit-lamp biomicroscopy with a +90 D lens. The stage of diabetic retinopathy was graded according to the ETDRS.

Macular imaging was performed using a spectral-domain OCT. The standard macular cube protocol was applied (6 × 6 mm area centered on the fovea). CMT was automatically calculated as the mean thickness in the central 1 mm ETDRS subfield. Scans with poor signal (< 7/10) were repeated.

Venous blood samples were drawn after overnight fasting. Glycated Hemoglobin (HbA1c): Measured by high-performance liquid chromatography. Fasting Lipid Profile: Included total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C). Standard enzymatic methods were used on an automated analyzer.

Patients were categorized into: Controlled glycemia-HbA1c < 6.5 %, Uncontrolled glycemia-HbA1c ≥ 6.5 %.

Data were entered into Microsoft Excel and analyzed using SPSS version 20.0 (IBM Corp., USA). Continuous variables were expressed as mean ± standard deviation (SD); categorical variables as percentages. Intergroup comparisons were made using Student's t-test or ANOVA. Correlations between CMT and biochemical parameters were assessed with Pearson's correlation coefficient (r). Multivariate linear regression was used to control for confounders such as age, sex, duration of diabetes, and DR grade. A p-value < 0.05 was considered statistically significant. Written informed consent was obtained from all participants. Confidentiality was maintained, and no identifying information was used in analyses or publications.

Results

Out of 130 patients, 87 (66.9%) were males and 43 (33.1%) females. The mean duration of diabetes was 8.87 ± 2.34 years. Based on ETDRS grading, 24.6% had no DR, 20.8% had very mild NPDR, 20% mild NPDR, 19.2% moderate NPDR, and 15.4% severe NPDR. Clinically significant macular edema (CSME) was present in 30% of subjects.

Patients with uncontrolled HbA1c (≥6.5%) had significantly greater mean CMT (266.24 ± 103.61 µm) compared to controlled HbA1c (<6.5%) (228.19 ± 45.02 µm, p=0.02). CMT increased with the severity of diabetic retinopathy (p<0.001). A weak positive correlation was observed between LDL and CMT, though not statistically significant.

A total of 130 patients with type 2 diabetes mellitus were included in the study. The mean age was 56.9 ± 8.3 years, ranging from 42 to 70 years. Of these, 87 (66.9%) were males and 43 (33.1%) were females, giving a male-to-female ratio of approximately 2:1.

The mean duration of diabetes was 8.87 ± 2.34 years. Most patients (68%) had diabetes for less than 10 years, while 32% had a longer duration. The mean HbA1c level was 8.28 ± 2.17%, indicating generally poor glycemic control among the study population. According to the ETDRS grading, the severity of diabetic retinopathy (DR) among the study subjects was distributed as follows:

Table 1: Total number of eyes in each stage of Diabetic Retinopathy

Stage of diabetic retinopathy	Number of Eyes (%)
No DR	32 (24.6%)
Very mild NPDR	27 (20.8%)
Mild NPDR	26 (20.0%)
Moderate NPDR	25 (19.2%)
Severe NPDR	20 (15.4%)
Proliferative DR	0 (0%)
DR= Diabetic retinopathy; NPDR= Non-Proliferative diabetic retinopathy	

Overall, 75.4% of patients had some degree of diabetic retinopathy, reflecting a high burden of retinal involvement in chronic diabetics. The mean CMT in the right eye was 260.41 ± 101.66 µm, and in the left eye 255.69 ± 88.89 µm. The values showed a symmetrical distribution between eyes (p > 0.05). Patients with no clinical DR had a mean CMT of 228.4 ± 45.6 µm, while those with severe NPDR had significantly higher mean CMT values (312.2 ± 121.5 µm, $p < 0.001$).

This demonstrates that CMT increased progressively with DR severity.

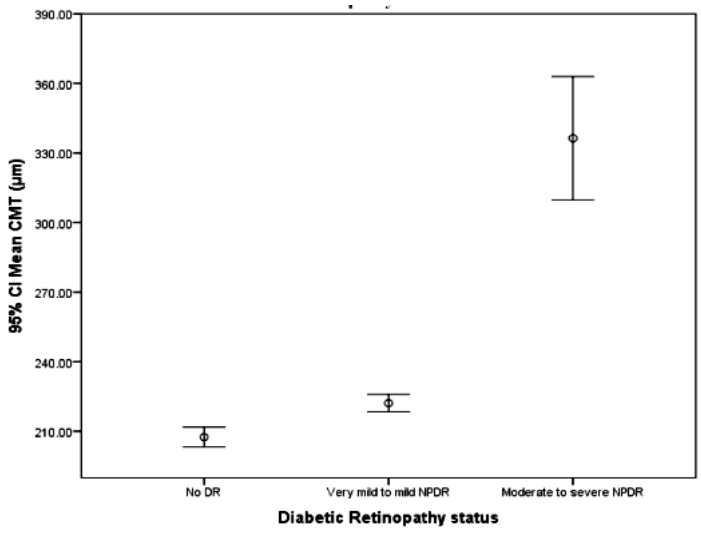

Fig 1: Relationship between central macular thickness and severity of diabetic retinopathy. A positive linear trend observed across DR stages

Table 2: Association of HbA1c with mean central macular thickness (CMT)

HbA1c Group	Mean CMT (µm) ± SD)	P- value
Controlled (<6.5%)	228.19±45.02	-
Uncontrolled (>6.5%)	266.24±103.61	0.02

Patients with uncontrolled HbA1c levels exhibited significantly higher macular thickness, indicating that poor glycemic control correlates with early structural retinal changes even in the absence of clinically significant macular edema. A positive correlation (r = +0.41, p = 0.02) was observed between HbA1c and CMT.

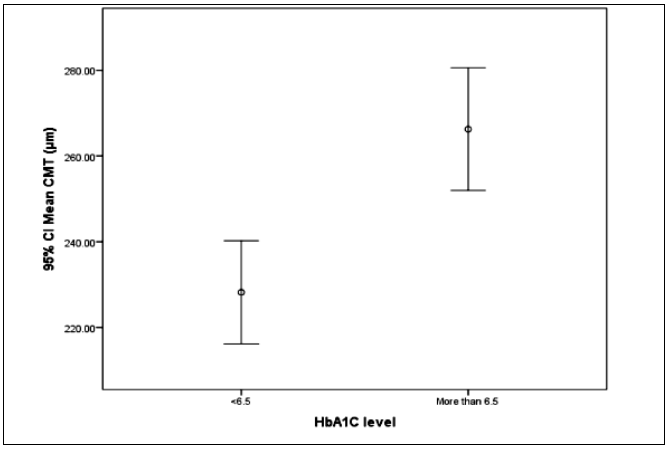

Fig 2: Scatter plot showing moderate positive correlation between HbA1c and central macular thickness.

The mean serum lipid levels were as follows:

- **Total Cholesterol:** 188.46 ± 60.64 mg/dl
- **Triglycerides:** 184.07 ± 77.41 mg/dl
- **LDL-C:** 114.95 ± 43.29 mg/dl
- **HDL-C:** 46.6 ± 9.84 mg/dl

Table 3: Correlations between lipid parameters and CMT

Lipid Parameter	Correlation Coefficient (r)	P-value	Interpretation
Total cholesterol	+0.17	0.12	Weak, not significant
Triglyceride	+0.14	0.18	Weak, not significant
LDL-C	+0.21	0.09	Positive trend,not significant
HDL-C	-0.08	0.32	Negative, not significant
CMT= central macular thickness; LDL= low lipoproteins; HDL= high lipoprotein			

Although no statistically significant relationship was found between lipid parameters and CMT, a positive trend was noted with LDL-C, suggesting a possible role in retinal thickening.

Multivariate regression analysis was performed with CMT as the dependent variable and HbA1c, LDL-C, HDL-C, age, sex, and duration of diabetes as independent predictors.

Only HbA1c showed a statistically significant association with CMT (β = 0.42, p = 0.02), confirming that glycemic control independently predicts macular thickness.

CMT increased significantly with DR severity ($p < 0.001$).

HbA1c positively correlated with CMT (r = 0.41, p = 0.02).

Lipid parameters showed nonsignificant correlations.

No gender difference was observed in CMT.

Longer duration of diabetes (>10 years) was associated with thicker maculae.

These results reinforce the hypothesis that poor metabolic control, especially elevated HbA1c, contributes to subclinical macular thickening detectable by OCT even before visual deterioration occurs.

Discussion

The present study explored the relationship between glycemic control (HbA1c), serum lipid profile, and central macular thickness (CMT) in patients with type 2 diabetes mellitus (T2DM). Using spectral-domain optical coherence

tomography (SD-OCT), we demonstrated that poor glycemic control is associated with increased macular thickness, even in the absence of clinically significant macular edema (CSME). Although serum lipid parameters showed only weak correlations, the overall findings emphasize the central role of metabolic control in preserving retinal structural integrity.

Our results revealed a statistically significant positive correlation between HbA1c and CMT ($r = 0.41$, $p = 0.02$). Patients with HbA1c ≥ 6.5 % had higher mean CMT than those with HbA1c < 6.5 %. This observation aligns with previous studies such as Chou *et al*. (2012) [1] and Parashar *et al*. (2018) [2], which demonstrated that elevated HbA1c levels correspond to increased macular thickness and early retinal neuro-glial dysfunction.

Persistent hyperglycemia damages retinal capillary endothelial cells and pericytes, causing basement-membrane thickening and microaneurysm formation. These microstructural alterations increase vascular permeability, leading to plasma leakage and intracellular edema within the macula. Chronic exposure to advanced glycation end products (AGEs) activates oxidative stress and inflammatory cascades that further disrupt the inner blood-retinal barrier. Consequently, even before clinical macular edema becomes apparent, OCT can detect subtle increases in retinal thickness that correlate with HbA1c levels.

The UKPDS and DCCT landmark trials established that each 1 % reduction in HbA1c decreases the risk of microvascular complications by 30-35 %. Our findings reinforce this, indicating that HbA1c control below 6.5 % may help delay macular changes in diabetic individuals.

Although not statistically significant, a weak positive trend was observed between LDL-cholesterol and CMT. Similar results have been reported by Benarous *et al*. (2011) [5] and Miljanović *et al*. (2004) [4], who found that higher LDL and triglyceride levels predispose to retinal exudation and macular edema. Lipid-induced oxidative stress, endothelial dysfunction, and altered vascular permeability may play a contributory role.

In our cohort, the absence of a strong lipid-CMT correlation may also reflect the relatively moderate dyslipidemia and concurrent use of lipid-lowering therapy in several patients. Future longitudinal studies controlling for statin use could clarify this relationship.

We found that CMT increased proportionally with DR severity ($p < 0.001$). This is consistent with Rema *et al*. (2016) [6] and Apte *et al*. (2020) [3], who reported that progressive capillary leakage and microaneurysm clustering in advanced NPDR lead to measurable thickening of the central macula. Interestingly, a subset of our patients without clinically evident DR already exhibited slightly increased CMT, suggesting that structural macular alterations may precede funduscopic signs of DR. This supports the concept of "subclinical diabetic macular changes," detectable only by OCT.

A number of studies have evaluated biochemical predictors of diabetic macular edema with varying outcomes: Chou *et al*. (2012) [1] reported a significant correlation between HbA1c and central foveal thickness in Taiwanese diabetics, supporting our results. Parashar *et al*. (2018) [2], in an Indian cohort, found a mean CMT of 273 µm in uncontrolled diabetics versus 231 µm in controlled cases ($p < 0.05$). Benarous *et al*. (2011) demonstrated that elevated serum lipids correlated with increased risk of retinal hard exudates. Yau *et al*. (2012), in a meta-analysis, emphasized that both duration of diabetes and HbA1c are major predictors of DR progression. Apte *et al*. (2020) [3] similarly noted an association between HbA1c and total macular volume using SD-OCT in Indian patients. Our findings thus corroborate global data while contributing additional evidence from an Indian population.

Conclusion

Elevated HbA1c levels are significantly associated with increased central macular thickness in Type 2 diabetic patients, even before the onset of clinically significant macular edema. Monitoring HbA1c and performing OCT screening can aid in early detection of subclinical macular changes, allowing timely intervention. Strict metabolic control is essential to prevent progression of diabetic retinopathy and associated vision loss.

Conflict of Interest

Not available

Financial Support

Not available

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