ARTICLE

Study of Macular Thickness between Patients with Type 2 Diabetes Mellitus without Diabetic Retinopathy versus Non-Diabetic Individuals

Reshma M.I.[1], M. Loganathan[1*]

[1]*Department of Ophthalmology, Sri Venkateshwaraa Medical College Hospital and Research Centre*

Corresponding author: M. Loganathan, log_smith@yahoo.com

ABSTRACT

Background: Type 2 diabetes mellitus (T2DM) is associated with early retinal neurodegeneration preceding clinical diabetic retinopathy (DR). This study compares macular thickness in T2DM patients without Diabetic Retinopathy versus non-diabetic controls to detect subclinical neuronal changes. Methods: A prospective cross-sectional study enrolled 120 participants (60 T2DM without DR, 60 healthy controls), aged 18-80 years in a tertiary care centre at Puducherry. Exclusion criteria included DR, hypertensive retinopathy, or significant media opacities. Macular thickness was measured using TOPCON 3D OCT-1 Maestro2. Groups were compared with Mann-Whitney U tests; gender effects via stratified analysis and ANCOVA. Results: Demographics were matched (mean age ~56 years; p-value >0.05 for age, gender, BCVA). Diabetics showed significant central foveal thinning (216.69 ± 1.33µm vs. 236.89 ± 0.13µm; p-value <0.001), temporal perifoveal (231.94 ± 4.36µm vs. 247.09 ± 2.80µm; p-value <0.001), temporal parafoveal (261.91 ± 2.77µm vs. 280.51 ± 3.52µm; p-value <0.001), and nasal perifoveal thinning (273.13 ± 2.38 µm vs. 280.87 ± 1.00µm; p-value <0.001). Superior and inferior sectors showed no differences (p-value >0.05). Patterns were consistent across genders; ANCOVA confirmed group effect (F=13,229.38, p-value <0.001) independent of gender (p-value >0.05). Conclusion: T2DM without DR demonstrates significant early macular thinning in central, temporal perifoveal and parafoveal and nasal perifoveal regions indicating neuronal loss before microangiopathy. Gender does not influence these changes, supporting proactive screening.

Keywords: Type 2 diabetes mellitus, diabetic retinopathy, subclinical neuronal changes

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INTRODUCTION

Type 2 diabetes mellitus (T2DM) represents a notable global health challenge among chronic noncommunicable diseases, contributing to significant morbidity, mortality and substantial economic burdens on healthcare system worldwide. Current epidemiological data indicate a rising prevalence affecting millions of adults. As a leading contributor to disability-adjusted life years (DALYs), diabetes results in multifaceted systemic complications involving the cardiovascular, renal, neurological and ocular systems.[1,2]

Diabetes mellitus is a wide range of metabolic diseases characterized by persistent hyperglycemia caused by decreased insulin secretion, reduced insulin sensitivity, or a combination of the two. The WHO diagnostic criteria state that certain glycemic thresholds, such as a fasting venous plasma glucose of ≥ 126 mg/dL, a 2-hour post-prandial glucose of ≥ 200 mg/dL, or a HbA1c level of ≥ 6.5%, are used to diagnose diabetes. Type 2 diabetes is generally caused by growing insulin resistance, relative insulin shortage, and metabolic changes often linked to obesity and aging, in contrast to type 1 diabetes, which is typified by autoimmune β-cell destruction.[3,4]

India contributes significantly to the global burden of diabetes and is frequently cited as a major epicentre due to its high prevalence, mortality and DALY rates. Projections suggest that India, China, and the United States had been collectively accounting for the number of about nearly one-third of the global diabetic population by 2050. This growing burden is attributed to a combination of genetic predisposition including the "thin-fat" phenotype, rapid epidemiological transitions, urbanization, sedentary lifestyle and fragmented primary healthcare systems that limit early screening and effective management.[1,5]

Globally, diabetic eye complications are the most common cause of vision impairment. These difficulties result from retinal neurodegeneration that affects several ocular tissues as well as microvascular damage. There are two traditional stages of diabetic retinopathy (DR): proliferative (PDR) and non-proliferative (NPDR). In individuals with severe disease, progressive retinal ischemia causes delicate new vessels to develop, putting them at risk for vitreous hemorrhage and tractional retinal detachment. Another significant cause of vision loss is diabetic macular edema (DME), which is brought on by fluid buildup in the macula and a breakdown of the blood retinal barrier. Anti-vascular endothelial growth factor (anti-VEGF) medication is crucial in the management of clinically severe macular edema, and OCT has emerged as a crucial imaging modality for the early detection, monitoring, and treatment of macular edema. Diabetes also increases the risk of secondary glaucoma, such as neovascular glaucoma brought on by retinal ischemia and iris neovascularization that results in an acute increase in intraocular pressure, and early cataract formation due to osmotic stress and sorbitol accumulation via the polyol pathway. To avoid irreparable vision loss, it is essential to identify these issues early and treat them appropriately.[6,7]

Retinal microangiopathy is the main underlying pathology of diabetic retinopathy. Chronic hyperglycemia causes structural changes in retinal capillaries, such as thickening of the basement membrane, pericyte loss, and malfunction of capillary endothelial cells. These alterations enhance vascular permeability, which causes fluid and lipoprotein leakage, intraretinal hemorrhages, and the development of microaneurysms. Macular edema and hard exudates are clinical manifestations of these alterations. In patients with proliferative diabetic retinopathy, capillary non-perfusion also causes retinal ischemia, which triggers the

*Author for Correspondence: log_smith@yahoo.com

production of VEGF and encourages pathological neovascularization in the disc, retina, and iris.[6,8]

Recent research suggests that retinal neurodegeneration can develop early in the course of diabetes, before clinically evident vascular abnormalities appear. The process known as diabetic retinal neurodegeneration (DRN) includes photoreceptor malfunction, gradual weakening of the inner retinal layer, and apoptosis of retinal ganglion cells. Neuronal injury has been linked to mechanisms such glutamate excitotoxicity, oxidative stress, and mitochondrial damage. By modifying neurovascular connection, these neuronal alterations may further exacerbate microvascular injury. Therefore, structural assessment of the retina using OCT, has gained importance in identifying early retinal changes such as macular thickness in diabetic patients. Integrating these longitudinal assessments into clinical practice enables precise risk stratification, allowing for intensive metabolic management and targeted intervention prior to the onset of irreversible visual impairment.[9,10]

Central macular thickness (CMT), measured using OCT, is widely used for the diagnosis and follow up of DME. Alterations in CMT are commonly used to assess the disease severity and response to treatment. However, changes in parafoveal retinal thickness may occur earlier than central involvement as this region contains a high density of retinal ganglion cells. Subtle thinning or thickening in these areas may indicate early neurodegenerative or microvascular changes before the development of clinically significant macular edema. Hence, evaluation of macular thickness parameters helps to detect early retinal alterations in diabetic patients without clinically evident diabetic retinopathy.[6,9,10]

There is limited literature evaluating macular thickness changes in type 2 diabetic patients without clinically detectable diabetic retinopathy, particularly in the Indian population. Therefore the present study was undertaken to compare the macular thickness between type 2 diabetic patients without diabetic retinopathy and healthy controls and to determine whether neuronal loss occurs prior to development of microangiopathy.

AIM

To assess the changes in macular thickness in type 2 diabetic patients without diabetic retinopathy.

OBJECTIVES

- To compare the macular thickness of type 2 Diabetes patient without diabetic retinopathy with non-diabetic individual.
- To determine whether neuronal changes occur before microangiopathy and vascular changes develop in retina.
- To assess gender differences in macular thickness among diabetes mellitus without retinopathy and non- diabetes mellitus patients.

MATERIALS AND METHODS

Study Design and Setting: Prospective cross-sectional study conducted in the Department of Ophthalmology in a tertiary care centre at Puducherry after approval from the Institutional Ethical Committee (Ref no: 22/SVMCH/IEC-Cert/February24) with study 120 study participants (60 in each group).

Calculation Basis: According to literature by Harwal et al. (2023), the central foveal thickness in Group 1 is 216.69 ± 2.06 µm and in Group 2 is 237.37 ± 1.26 µm (mean difference of -2.68). Using EPI INFO 3.0.1 with 80% statistical power and a 95% confidence level, the minimum calculated sample size was 1 per group. However, the study opted for 120 participants based on the previous years' census from the Ophthalmology Outpatient Department (OPD)[11].

Participant Timeline: Enrolment, history taking, general examination, and ocular examination including macular thickness measurement were done and it took approximately 2 hours per patient.

Inclusion criteria: Patients diagnosed with Type 2 Diabetes Mellitus, both male and female aged between 18 and 80 years and patients who have provided voluntary consent to participate in the study are included in the study.

Exclusion Criteria: Clinical evidence of Diabetic Retinopathy (DR) or Hypertensive Retinopathy and significant media opacity that interfered the imaging of Optical Coherence Tomography (OCT) were exclusion criteria.

Data collection: A standardized proforma that confidentially recorded demographic information (age, gender, occupation, and socioeconomic status) was used to gather participant data. Chronicity of symptoms, previous medical and surgical procedures, and ocular/systemic comorbidities were all covered in the comprehensive medical history. Visual acuity (Snellen's chart), intraocular pressure (non-contact tonometry), color vision (Ishihara charts), anterior segment evaluation (slit-lamp biomicroscopy), and fundus examination (indirect ophthalmoscopy) were all standardized ocular assessments that guaranteed diagnostic consistency. Quantitative and qualitative macular examination was made possible by advanced imaging using Optical Coherence Tomography (OCT). To facilitate precise statistical analysis and intergroup comparison, all results were methodically documented.

STATISTICAL ANALYSIS

Microsoft Excel was used to tabulate the data, while SPSS v24.0 was used for analysis. While categorical data were displayed as frequencies and percentages, continuous variables were given as mean ± SD. The Shapiro-Wilk test was used to determine normality. Chi-square or Fisher's exact tests were used to compare categorical variables, while the Mann-Whitney U test was used to compare non-parametric continuous data. Sex-specific patterns of macular thickness were investigated using gender-stratified Mann-Whitney U analysis. Central foveal thickness (CFT) was compared between the diabetic and control groups using ANCOVA, and group, gender, and interaction effects were evaluated using gender as a covariate. The threshold for statistical significance was fixed at $p < 0.05$.

RESULTS

The results of the study are presented in tables (1-7) and pictorial representation (1-3b).

Table 1: Gender distribution among the study population

Gender	Participants with T2DM	Healthy controls	Total	p
Male	26 (43.3)	25 (41.7)	51 (42.5)	1.000
Female	34 (56.7)	35 (58.3)	69 (57.5)	

Table 2: Age distribution and Assessment of Intraocular Pressure (IOP)

Age	Participants with T2DM	Healthy controls	p-value		
	Mean	SD	Mean	SD	0.517
	56.45	4.2042	56.95	4.5934	
Assessment of Intraocular Pressure (IOP)					
IOP (mmHg)	14.156	0.499	14.054	0.300	0.094

Table 3: Assessment of Macular Thickness

Macular thickness (microns)	Participants with T2DM	Healthy controls	p-value		
	Mean	SD	Mean	SD	
Temporal peri	231.937	4.3596	247.092	2.7994	<.001
Temporal para	261.908	2.7655	280.513	3.5247	<.001
Nasal peri	273.133	2.3808	280.865	0.9995	<.001
Nasal para	306.133	1.0611	306.505	1.167	0.065
Inferior peri	263.025	0.8736	262.983	0.9009	0.795
Inferior para	294.647	4.0784	296.003	3.7965	0.056
Superior peri	263	0.7642	263.062	0.8577	0.692
Superior para	281.852	3.9873	282.785	3.6716	0.172
Mann-Whtiney U test was applied					

Table 4: Gender stratified analysis of Age and IOP (mmHg)

Parameters	Participants with T2DM	Healthy controls	p-value		
	Mean	SD	Mean	SD	
Male					
Age	58.077	4.039	57.2	4.3301	0.473
IOP (mmHg)	14.202	0.331	14.085	0.308	0.243
Female					
Age	55.206	3.9449	56.771	4.8269	0.183
IOP (mmHg)	14.121	0.3183	14.033	0.2968	0.247
Mann-Whitney U test was applied					

Table 5: Gender stratified - Assessment of Macular thickness - Perifoveal zone

Parameters	Participants with T2DM		Healthy controls	p-value	
	Mean	SD	Mean	SD	
Male					
CFT (µm)	216.831	1.2973	236.9	0.1323	<.001
Tempo peri	232.415	4.5725	246.988	2.7936	<.001
Tempo para	262.304	2.8622	280.84	2.9625	<.001
Nasal peri	272.604	2.1908	280.776	0.9909	<.001
Nasal para	306.185	1.0271	306.568	1.1235	0.174
Inferior peri	263.031	0.8648	263.032	0.9272	0.94
Inferior para	294.062	3.7899	296.128	3.6255	0.062
Superior peri	262.962	0.8256	263.268	0.8615	0.196
Superior para	282.235	3.8603	283.568	3.7198	0.193
Female					
CFT (µm)	216.582	1.3675	236.889	0.1278	<.001
Tempo peri	231.571	4.2217	247.166	2.8419	<.001
Tempo para	261.606	2.6926	280.28	3.9024	<.001
Nasal peri	273.538	2.4714	280.929	1.0151	<.001
Nasal para	306.094	1.1001	306.46	1.2113	0.188
Inferior peri	263.021	0.8933	262.949	0.8936	0.727
Inferior para	295.094	4.2873	295.914	3.964	0.374
Superior peri	263.029	0.7251	262.914	0.836	0.528
Superior para	281.559	4.1148	282.226	3.585	0.457
Mann-Whitney U test was applied					

Table 6: Gender-Specific Trends and Consistency

Parameter	Male Trend (Participants with T2DM vs Control)	Female Trend (Participants with T2DM vs Control)	Significance
Central Foveal Thickness (CFT)	Significant Thinning	Significant Thinning	$p < 0.001$
Temporal Peri-foveal	Significant Thinning	Significant Thinning	$p < 0.001$
Temporal Para-foveal	Significant Thinning	Significant Thinning	$p < 0.001$
Superior and Inferior Sectors	No Difference	No Difference	$p > 0.05$

Table 7: Gender-Adjusted ANCOVA Comparison

	F	p
Overall model	4520.016	<.001
Gender	0.548	.461
Group	13229.381	<.001
Gender Group	0.456	.501
ANCOVA - CFT (µm)		

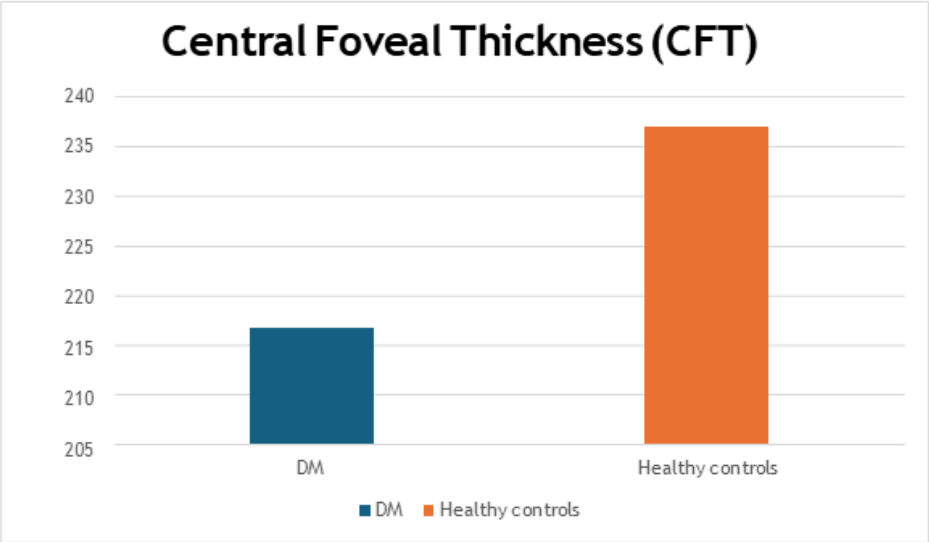

Figure 1: Central Foveal Thickness

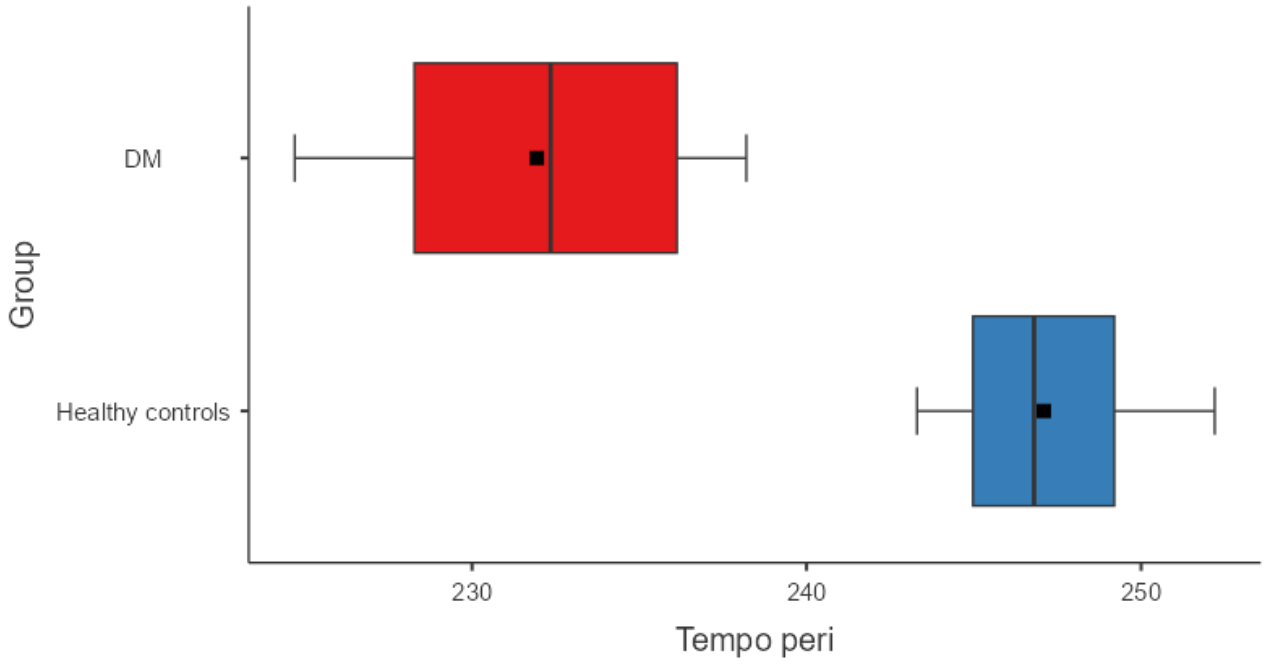

Figure 2a: Temporal Perifoveal zone

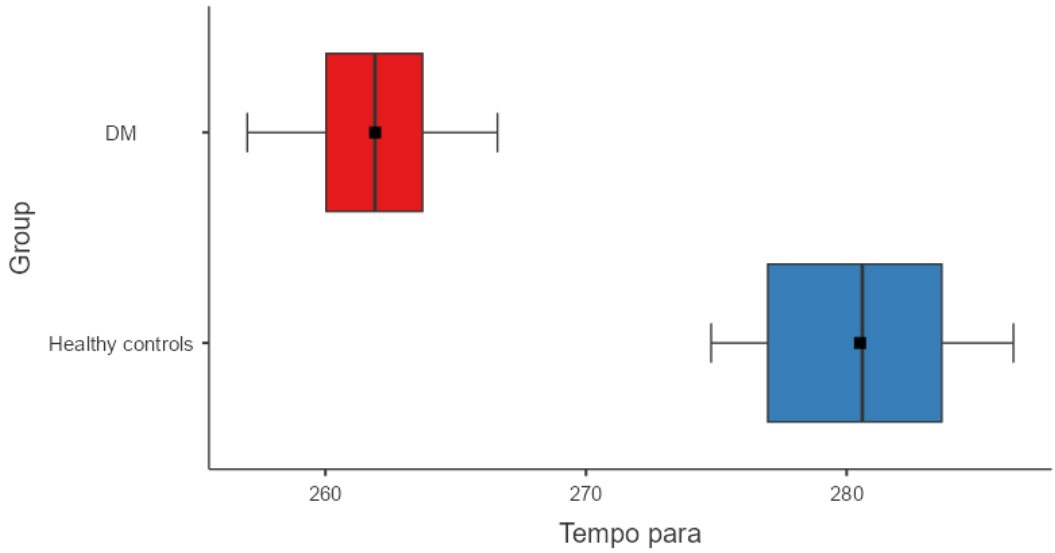

Figure 2b: Temporal Parafoveal zone

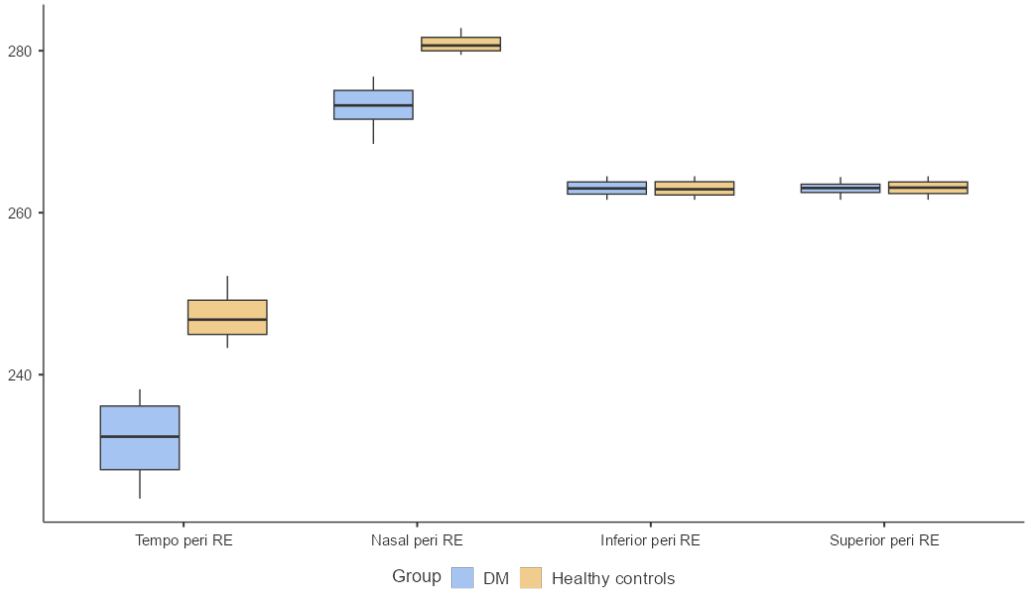

Figure 3a: Assessment of Macular thickness - Perifoveal zone

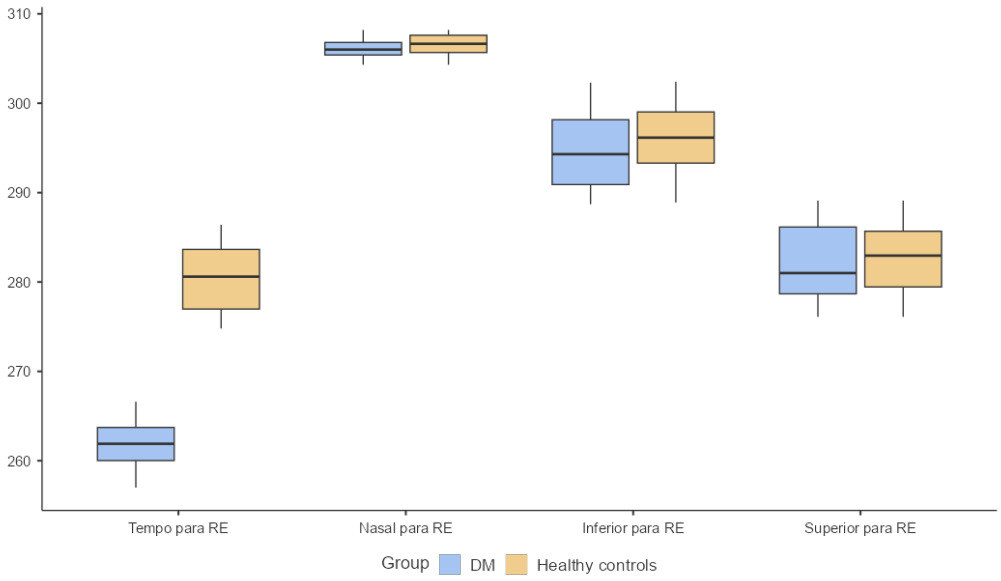

Figure 3b: Assessment of Macular thickness - Parafoveal zone

The gender distribution was consistent across both groups (p-value = 1.000); the diabetic cohort comprised of 43.3% males and 56.7% females, which is similar to the control group which consist of 41.7% males and 58.3% females.

The study participants in the diabetic group (DM) had a mean age of 56.45 ± 4.20 years, while the participants in the healthy control group had a nearly identical mean age of 56.95 ± 4.59 years. It shows no statistically significant difference between

diabetic and non-diabetic group (p-value = 0.517). Patients in the Diabetes Mellitus (DM) group showed a higher mean IOP of 14.156 ± 0.499 mmHg, whereas the healthy control group showed a mean value of 14.054 ± 0.300 mmHg, however there is no statistical significance.

Comparative analysis investigates the morphological changes in macular pattern among patients with Type 2 Diabetes Mellitus (T2DM) who have not yet developed clinical signs of Diabetic Retinopathy (DR), compared to a healthy control cohort. The data measured in microns (μm), reveals significant early-stage neurodegenerative thinning in specific macular zones prior to the onset of microvascular complications.

Thinning in Central and Temporal Zones

The study indicated significant reduction in Central Foveal Thickness (CFT) among diabetic patients. The mean CFT in the DM group was 216.69 ± 1.33 μm, compared to 236.89 ± 0.13 μm in healthy controls (p-value < 0.001).

Significant thinning was observed in the temporal regions. Both the Temporal Perifoveal zone (231.94 ± 4.36 μm vs 247.09 ±2.80 μm) and the Temporal Parafoveal zone (261.91 ± 2.77 μm vs 280.51 ±3.52 μm) showed a substantial decrease in thickness among diabetic patients (p -value < 0.001).

While the central, temporal, and nasal perifoveal zones showed significant differences, other regions of the macula appeared relatively spared at this early stage of the disease. The Nasal Parafoveal zone (p-value = 0.065) and the Inferior Parafoveal zone (p-value = 0.056) did not reach the statistical significance.

Furthermore, the Superior and Inferior perifoveal regions showed almost no difference between the groups (p-value = 0.692 and p -value= 0.795, respectively).

Gender stratified analysis

The results are stratified by gender to identify potential neurodegenerative changes in the retina that may precede clinical vascular signs.

Baseline Demographics and Clinical Parameters

In both the male and female population, there were no statistically significant differences between the diabetic and control groups regarding Age, Mean Best Corrected Visual Acuity (BCVA), or Intraocular Pressure (IOP) (p-value > 0.05). This indicates that the groups were well-matched at baseline, ensuring that any observed differences in macular thickness are likely attributable to the underlying diabetic metabolic state rather than age-related changes or variations in visual function.

Central Foveal and Temporal Macular Thinning

A highly significant reduction in central foveal thickness (CFT) was observed in diabetic patients across both genders (p-value < 0.001). In males, the diabetic group showed a mean CFT of 216.83 μm compared to 236.9 μm in controls; an identical trend was observed in females. Furthermore, the temporal regions (both peri-foveal and para-foveal) exhibited significant thinning in the diabetic groups (p-value < 0.001).

Nasal, Superior, and Inferior Sector Analysis

While the nasal peri-foveal region showed significant thinning in both males and females (p value < 0.001), other sectors appeared relatively resilient in the early stages of the disease. Specifically, the Superior (peri and para) and Inferior (peri and para) sectors did not show statistically significant differences between the diabetic and control groups (p-value > 0.05). The Nasal para-foveal region also remained stable.

Gender-Specific Trends and Consistency

The data reveals a remarkable consistency in the patterns of retinal thinning between males and females. This significant p-value (< 0.001) for CFT and temporal sectors in both cohorts reinforce the validity of these findings.

Evaluation of Central Foveal Thickness across Diabetic and Non-Diabetic Cohorts: A Gender-Adjusted ANCOVA Comparison

The provided ANCOVA results examine the differences in Central Foveal Thickness (CFT) between patients with Type 2 Diabetes Mellitus (without retinopathy) and a Non-diabetic control group, while controlling for the potential influence of gender.

Primary Comparative Analysis

The most significant finding of this study was the substantial difference in macular thickness between the two study groups. The Group effect yielded a remarkably high F-statistic (F = 13,229.38, p-value < 0.001), indicating that the presence of Type 2 Diabetes, even in the absence of clinical diabetic retinopathy significantly impacts Central Foveal Thickness.

Evaluation of Gender Influence and Interaction

The analysis confirms that gender does not play a significant role in determining Central Foveal Thickness within this specific cohort. The main effect for Gender was non-significant (F = 0.548, p = 0.461) and the Gender Group interaction was also statistically negligible (F = 0.456, p = 0.501). These results demonstrate that the impact of Type 2 Diabetes on CFT was consistent across both male and female participants, suggesting that the physiological thickening (or thinning) associated with the disease process was not gender-dependent.

DISCUSSION

This cross-sectional study conducted in a tertiary care teaching institute in South India utilized the SD-OCT to compare macular thickness between 2 groups .The first group was 60 patients diagnosed with T2DM without any clinically evident diabetic retinopathy and 2nd group consisted 60 healthy controls who were age and gender matched. The diabetic cohort demonstrated significant structural alterations, specifically a marked reduction in central foveal thickness (CFT- 216.69 ± 1.33 □m vs. 236.89 ± 0.13 □m) and thinning within the temporal perifoveal and parafoveal regions which is statistically significant with p-value of <0.001. There was no statistically significant thinning in superior and inferior regions in both diabetic and non-diabetic patients. Notably, ANCOVA revealed no significant group-by-gender interactions, confirming that these thinning patterns are similar across both sexes. Additionally, the diabetic group exhibited significantly higher intraocular pressure among the study population in diabetics when compared to non-diabetic though there was no significant difference in visual acuity between both groups (14.17 ± 0.31 mmHg vs. 14.05 ± 0.30 mmHg, p-value = 0.028). These findings provide clinical evidence of early onset of retinal neurodegeneration before the occurrence of microvascular diabetic complication and it was not associated with any difference among both genders.[12,13]

The diabetic group showed a significant reduction in central foveal thickness, which was the most remarkable finding of the present study. Similar findings were observed by Dumitrescu et al., who discovered that while most other quadrants remained unaffected, the central fovea was narrower in type 2 diabetes without retinopathy than in healthy persons.[12] Similarly, Jiang et al. found that the foveal, temporal parafoveal, and perifoveal regions showed the greatest signs of retinal thinning in diabetes without retinopathy.[13] They proposed that these early alterations are more indicative of neurological damage than vascular leakage based on the findings of their investigation. The current results are consistent with previous research, supporting the idea that

diabetes might affect the retina prior to the funduscopic manifestation of diabetic retinopathy.[14]

The temporal predominance of thinning in this study is also biologically plausible and has been repeatedly described in earlier literatures. For instance, Jiang et al. found that temporal areas were among the first to show reduced thickness, and findings from Clerk E et al. also reported thinner foveal and pericentral macula in type 2 diabetes without retinopathy.[13,15] This finding can be attributed to the mechanism that the temporal retina may be more vulnerable to metabolic stress because of its connection with the papillomacular bundle and the high density of inner retinal neurons in this region. This pattern supports the idea that early diabetes-relatedretinal injury may follow a neurodegenerative pathway, with structural loss occurring before obvious vascular signs.[16,17]

In contrast, several macular sectors in the present study, particularly the superior and inferior regions, did not show statistically significant thinning. This regional selectivity is not unusual and has been reported in previous OCT-based studies by Jiang J et al., Toprak I et al. and Netto M et al. where diabetes without retinopathy produced localized rather than diffuse macular changes.[13,18,19] Such sector-specific involvement may indicate that retinal damage begins in anatomically susceptible areas and then progresses gradually to broader retinal involvement as disease progress and the severity of disease increases. The relative preservation of some sectors may therefore reflect an early stage of retinal involvement rather than absence of disease effect.12,16,18

Another relevant finding in the present study was the absence of a significant association with gender. Although healthy-eye normative data by Adhi M et al have suggested that males may have slightly greater macular thickness than females, that difference does not appear to explain the diabetic-control contrast in this study population. The ANCOVA results here showed no significant gender effect and no group-by-gender interaction, indicating that the diabetic effect on central foveal thickness was stable across genders. This is useful clinically because it suggests that the observed thinning is primarily disease-related and not an artifact of sex distribution or gender-based anatomical variation.[20]

The higher intraocular pressure observed in the diabetic group, although small in absolute terms, may be clinically relevant. Existing literatures by Hymowitz M et al. and Hennis A et al. had documented that diabetes has been associated with a higher risk of ocular hypertension and glaucoma-related structural vulnerability, through altered trabecular outflow, osmotic effects, or vascular dysregulation.[21,22] The IOP difference may reflect a wider ocular impact of diabetes and requires attention in longitudinal follow-up, especially since retinal neurodegeneration and optic nerve vulnerability may coexist in diabetic eyes, even if the current study was not intended to show causation.

The findings of the present study support to the growing consensus that diabetic retinal disease is not solely vascular in origin. There is growing evidence that inner retinal thinning, ganglion cell damage, and retinal neuronal loss are early indicators of diabetes. This process is believed to be influenced by hyperglycemia, oxidative stress, excitotoxicity, mitochondrial dysfunction, and microvascular impairment.[13,17,18] By demonstrating that structural macular thinning can be identified even in the absence of clinically noticeable retinopathy, the current work contributes to the body of evidence by pointing to a possible opportunity for earlier identification and intervention.

The result of current study highlights the value of OCT as an adjunctive tool in diabetic eye assessment. Standard fundus examination may miss early neuroretinal changes, whereas OCT can detect subtle thinning before visible retinopathy appears. This has important implications for screening, risk stratification, early identification of the disease process and possible future role of neuroprotective agents as a part of management of diabetic retinopathy.

This study had comparable age and gender distribution between diabetic and control groups, minimizing confounding effects. The use of spectral-domain OCT (TOPCON 3D OCT-1 Maestro 2) provided high-resolution, quantitative macular thickness measurements across multiple sectors, enabling precise detection of subtle regional changes. Gender-stratified analyses and ANCOVA adjustment for gender further strengthened the findings, confirming consistent central foveal and temporal thinning independent of demographics. The sample size of 120 participants exceeded power calculations, enhancing statistical robustness for the primary outcome. Prospective data collection over the study period at a single center ensured standardized protocols and reduced variability from inter-observer or device differences.

However, the cross-sectional design limits the ability to establish causal relationships and does not allow longitudinal follow up of macular thinning over time. As a result, it remains unclear whether the observed changes can predict the development of future retinopathy. Key confounders such as diabetes duration, HbA1c levels, systemic hypertension, dyslipidaemia, and renal function were not reported or adjusted for, potentially influencing retinal neurodegeneration and thickness measurements. Exclusion of patients with significant media opacities or any retinopathy may limit generalizability to broader diabetic populations with comorbidities. Small absolute IOP difference (0.12 mmHg), despite statistical significance, may have limited clinical relevance without longitudinal glaucoma outcome data.

CONCLUSION

This cross-sectional study with 60 study participants and 60 control group reveals that type 2 diabetes mellitus, even without visible diabetic retinopathy, leads to early and selective thinning of the macular region, particularly in the central fovea and temporal regions, as detected through high-resolution Spectral-Domain Optical Coherence Tomography. These consistent neurodegenerative alterations across both male and female participants, unaffected by age or visual acuity differences underscore the presence of preclinical retinal changes that precede traditional microvascular complications. The findings of the current study support OCT as a powerful, non-invasive tool for identifying the certain findings in diabetic eyes before the appearance of clinically detectable diabetic retinopathy changes.

From a clinical perspective, incorporating routine macular OCT scan as screening in diabetic eye care protocols could facilitate earlier risk stratification, timely interventions, and potential neuroprotective strategies to safeguard long-term visual health and avert progression to vision threatening diabetic eye disease.

Further, prospective longitudinal research that accounts for glycemic control, disease duration, and systemic comorbidities are essential to detect the natural progression of these changes and define optimal therapeutic options for preserving retinal integrity and its function.

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