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Original Research Article

Assessment of Retinal Nerve Fiber Layer and Inner Macular Layer Thickness in Patients with Type 2 Diabetes Mellitus Using Spectral-Domain Optical Coherence Tomography

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Abstract

Introduction: Diabetic retinopathy (DR) is a leading microvascular complication of type 2 diabetes mellitus (T2DM). Neurodegenerative thinning of the retinal nerve fiber layer (RNFL) and ganglion cell layer–inner plexiform layer (GCL-IPL) complex may precede clinically visible retinopathy.

Purpose: The purpose of the study is to compare peripapillary RNFL (pRNFL), macular RNFL (mRNFL), GCL-IPL, and average macular thickness on spectral-domain optical coherence tomography (SD-OCT) among normal individuals, T2DM patients without DR, and T2DM patients with mild non-proliferative DR.

Methods: In this cross-sectional, hospital-based comparative study, 60 participants (20/group) underwent comprehensive ocular examination and SD-OCT (Topcon 3D OCT-1 Maestro) using 3D-wide, 3D-disc, 3D-macula(v), and 3D-macula scans. Group means were compared using one-way analysis of variance; $P < 0.05$ was significant.

Results: Mean age was 60.93 ± 10.06 years; 55% were women. GCL-IPL thickness (3D-wide) was significantly reduced in all six sectors in both diabetic groups versus controls ($P < 0.001$). Average pRNFL thickness was 101.96 ± 9.39, 98.89 ± 7.39, and 92.01 ± 9.64 μm in normal, DM-without-DR, and DM-with-DR eyes, respectively ($P = 0.001$), with significant thinning in all four quadrants. mRNFL thickness was significantly reduced ($P \leq 0.03$), whereas macular GCL-IPL ($P = 0.09–0.23$) and average macular thickness ($P = 0.06$) were not significantly different.

Conclusion: RNFL and GCL-IPL thinning occurs even before clinically detectable DR and may serve as an early structural biomarker of diabetic retinal neurodegeneration.

Key words: Diabetic retinopathy, Ganglion cell–inner plexiform layer, Macular thickness, Optical coherence tomography, Retinal nerve fiber layer, Type 2 diabetes mellitus

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a major global disease burden, and India accounts for a substantial share of the diabetic population.[1] Diabetic retinopathy (DR), its common microvascular complication, remains a leading cause of preventable blindness.[2] DR is traditionally classified by grading microvascular lesions – microaneurysms, hemorrhages, exudates and neovascularization – and its natural history and risk factors have been well characterized.[3]

Accumulating evidence suggests, however, that diabetes affects the neural retina before any vascular lesion becomes clinically visible, through ganglion cell apoptosis, glial activation and inner-retinal thinning – implying that diabetic retinal disease is, in part, a neurodegenerative process that may precede microvascular change.[4]

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Optical coherence tomography (OCT) permits non-invasive, *in vivo*, micrometer-scale quantification of retinal layers.[5] Since its introduction, OCT technology has evolved from time-domain to spectral-domain and swept-source platforms, markedly improving axial resolution and scan speed and enabling reproducible segmentation of individual retinal layers.[6-8] Spectral-domain OCT (SD-OCT) allows separate segmentation of the peripapillary RNFL (pRNFL) and, in the macula, the macular RNFL (mRNFL) and ganglion cell layer–inner plexiform layer (GCL-IPL) complex – structures composed of retinal ganglion cell axons and cell bodies – and reproducibility of these retinal nerve fiber layer (RNFL) measurements with SD-OCT has been well validated.[9] Quantifying these layers may therefore offer a potential early, objective biomarker of diabetic neurodegeneration.

RNFL and GCL-IPL analysis on OCT has proved useful beyond glaucoma, in conditions as varied as pediatric and gestational diabetes, epilepsy, multiple sclerosis and compressive or afferent pupillary pathway disease, reflecting the broad applicability of inner-retinal imaging as a neuro-structural biomarker.[10-14] Several studies have specifically examined RNFL and GCL-IPL thickness in diabetic eyes, with variable results depending on scan protocol, population and stage of retinopathy studied.[15-19] Indian normative OCT data comparing diabetics without retinopathy and those with early (mild non-proliferative) retinopathy against age-matched controls, using multiple scan protocols in the same cohort, remain limited.[20]

We therefore undertook this study to quantitatively measure, using SD-OCT, the pRNFL, mRNFL, and GCL-IPL thickness in patients with T2DM with and without DR and to compare these with an age-matched non-diabetic population, with the secondary aim of assessing average macular thickness across the three groups.

MATERIALS AND METHODS

Study Design and Setting

This was an observational, cross-sectional, comparative, hospital-based study conducted in the Department of Ophthalmology, R.D. Gardi Medical College and Hospital, Ujjain, Madhya Pradesh, India, after Institutional Ethics Committee approval and written informed consent from all participants.

Participants

Sixty participants attending the ophthalmology outpatient department were divided into three groups of 20 each: age-matched non-diabetic normal individuals; T2DM patients without DR; and T2DM patients with mild non-proliferative DR (NPDR), defined as microaneurysms, retinal hemorrhages, hard exudates or cotton-wool spots without intraretinal microvascular abnormalities or significant venous beading.

Inclusion criteria were T2DM patients aged ≥35 years with disease duration ≥1 year and non-diabetic controls aged ≥35 years. Exclusion criteria were type 1 diabetes, high refractive error (>+3D or –3D spherical equivalent), diabetic macular edema, glaucoma or other optic neuropathy, pre-existing retinal or choroidal disease, and significant media opacity. Sample size (20/group) was calculated using a two-mean hypothesis test (expected mean RNFL difference 6.0 μm, standard deviation (SD) 5.44, $\alpha = 0.01$, power 80%).

Examination Protocol

All participants underwent visual acuity assessment, slit-lamp biomicroscopy, non-contact tonometry (Topcon CT-800), and dilated fundus examination with a 90D lens. SD-OCT (Topcon 3D OCT-1 Maestro, Topcon Corp., Tokyo, Japan) was performed by a single examiner after pupillary dilatation; only scans with signal strength >20 were analyzed.

Four scan protocols were used: (1) 3D-Disc (6 × 6 mm) for pRNFL thickness in four peripapillary quadrants – superior, inferior, nasal and temporal; (2) 3D-Wide (12 × 9 mm) for GCL-IPL thickness in six macular sectors – superior, supero-temporal, supero-nasal, inferior, infero-temporal and infero-nasal; (3) 3D-Macula(v) (7 × 7 mm) for superior, inferior and total mRNFL and GCL-IPL thickness; and (4) 3D-Macula (6 × 6 mm) for average macular thickness.

Statistical Analysis

Data were recorded on a pre-tested pro forma and analyzed using SPSS software. Continuous variables are presented as mean ± SD and compared among groups using one-way analysis of variance; categorical variables are presented as frequencies and percentages. A $P < 0.05$ was considered statistically significant.

RESULTS

A total of 60 participants (20/group) completed the study. Mean age was 60.93 ± 10.06 years (range 40–80), with 36.7% aged 51–60 years. Women comprised 55% ($n = 33$) [Table 1].

On the 3D-Wide scan, GCL-IPL thickness was significantly reduced across all six macular sectors in a stepwise fashion – highest in controls, intermediate in diabetics without DR, and lowest in diabetics with mild NPDR ($P < 0.001$ for every sector), with the largest absolute

reduction in the infero-temporal and superior sectors [Table 2 and Figure 1].

On the 3D-Disc scan, mean average pRNFL thickness differed significantly among groups (101.96 ± 9.39 vs. 98.89 ± 7.39 vs. 92.01±9.64 μm; $P = 0.001$), with significant thinning in all four quadrants – temporal ($P = 0.01$), superior ($P = 0.02$), nasal ($P = 0.003$), and inferior ($P = 0.03$) [Table 2 and Figure 2].

On the 3D-Macula(v) scan, superior, inferior, and total mRNFL thickness were each significantly reduced across groups ($P = 0.01$, 0.02, and 0.03, respectively), whereas the corresponding macular GCL-IPL measurements did not reach significance ($P = 0.09$, 0.09, and 0.23), although normal eyes still showed numerically higher values than either diabetic group [Table 3 and Figure 3].

On the 3D-Macula scan, average macular thickness was numerically highest in controls (232.10 ± 9.93 μm) versus DM-without-DR (228.75 ± 19.51 μm) and DM-with-DR (220.65 ± 14.60 μm) eyes, without reaching significance ($P = 0.06$) [Table 3 and Figure 4].

DISCUSSION

In this study, we quantitatively compared pRNFL, mRNFL, GCL-IPL, and average macular thickness using SD-OCT among age-matched normal individuals, T2DM patients without retinopathy, and T2DM patients with mild NPDR. The principal finding was a consistent, stepwise reduction in neural retinal thickness with increasing diabetic severity, evident even in patients without any ophthalmoscopically visible retinopathy – supporting the concept that diabetic

Table 1: Baseline demographic characteristics of the study population (*n*=60)

Characteristic	Category	*n* (%)
Age (years)	Mean±Standard deviation: 60.93±10.06 (range 40–80)	
	≤50 yr/51–60 yr/61–70 yr/>70 yr	11 (18.3)/22 (36.7)/17 (28.3)/10 (16.7)
Gender	Male/Female	27 (45.0)/33 (55.0)
Residence	Rural/Urban	36 (60.0)/24 (40.0)
Education	Literate/Illiterate	34 (56.7)/26 (43.3)
Occupation	Housewife/Field worker/Office worker/Student	20 (33.3)/15 (25.0)/14 (23.3)/11 (18.3)
Socioeconomic status	Middle/Lower/Higher class	26 (43.3)/24 (40.0)/10 (16.7)

Table 2: Comparison of GCL-IPL thickness (3D-Wide scan) and pRNFL thickness (3D-Disc scan) among study groups (μm)

Scan/Parameter	Normal	DM without DR	DM with mild NPDR	p-value
3D-Wide GCL-IPL – Superior	104±4.75	95±3.19	84±2.5	<0.001
3D-Wide GCL-IPL – Supero-temporal	104±3.45	96±3.8	85±2.58	<0.001
3D-Wide GCL-IPL – Supero-nasal	107±3.36	94±2.70	86±1.51	<0.001
3D-Wide GCL-IPL – Inferior	103±3.67	94±3.01	84±2.68	<0.001
3D-Wide GCL-IPL – Infero-nasal	105±4.64	93±2.86	83±2.62	<0.001
3D-Wide GCL-IPL – Infero-temporal	107±2.90	98±3.34	88±1.97	<0.001
3D-Disc pRNFL – Average	101.96±9.39	98.89±7.39	92.01±9.64	0.001
3D-Disc pRNFL – Temporal	86.05±7.93	82.60±11.04	77.05±5.23	0.01
3D-Disc pRNFL – Superior	121.85±11.32	117.55±15.79	111.10±7.63	0.02
3D-Disc pRNFL – Nasal	87.60±8.62	85.25±5.72	78.45±8.03	0.003
3D-Disc pRNFL – Inferior	129.60±13.37	123.85±13.93	118.30±12.55	0.03

pRNFL: Peripapillary retinal nerve fiber layer, GCL-IPL: Ganglion cell layer–inner plexiform layer, DM: Diabetes mellitus, DR: Diabetic retinopathy, NPDR: Non-proliferative diabetic retinopathy

Table 3: Comparison of macular mRNFL, GCL-IPL (3D-Macula[v] scan), and average macular thickness (3D-Macula scan) among study groups (μm)

Scan/Parameter	Normal	DM without DR	DM with mild NPDR	p-value
3D-Macula (v) mRNFL – Superior	35.35±3.95	34.35±3.63	31.80±3.59	0.01
3D-Macula (v) mRNFL – Inferior	38.40±5.29	36.20±4.01	34.10±3.81	0.02
3D-Macula (v) mRNFL – Total	36.35±3.87	35.27±4.55	33.05±2.31	0.03
3D-Macula (v) GCL-IPL – Superior	74.45±13.08	64.25±14.09	68.35±15.14	0.09
3D-Macula (v) GCL-IPL – Inferior	71.45±13.08	62.05±12.93	66.05±13.99	0.09
3D-Macula (v) GCL-IPL – Total	69.80±7.50	64.30±12.75	64.55±12.74	0.23
3D-Macula – Average macular thickness	232.10±9.93	228.75±19.51	220.65±14.60	0.06

mRNFL: Macular retinal nerve fiber layer, GCL-IPL: Ganglion cell layer–inner plexiform layer, DM: Diabetes mellitus, DR: Diabetic retinopathy, NPDR: Non-proliferative diabetic retinopathy

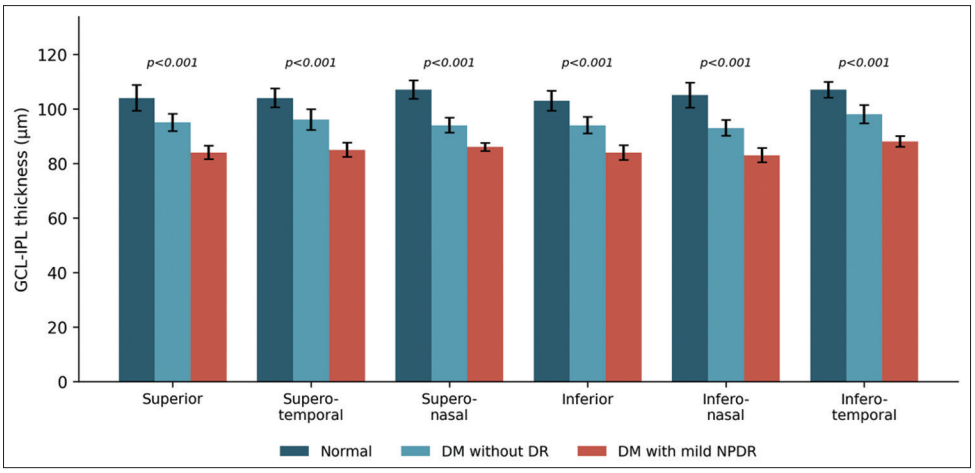

Figure 1: Sectoral ganglion cell layer–inner plexiform layer thickness (3D-Wide scan) across study groups. Error bars represent standard deviation; *P*<0.001 for all sectors

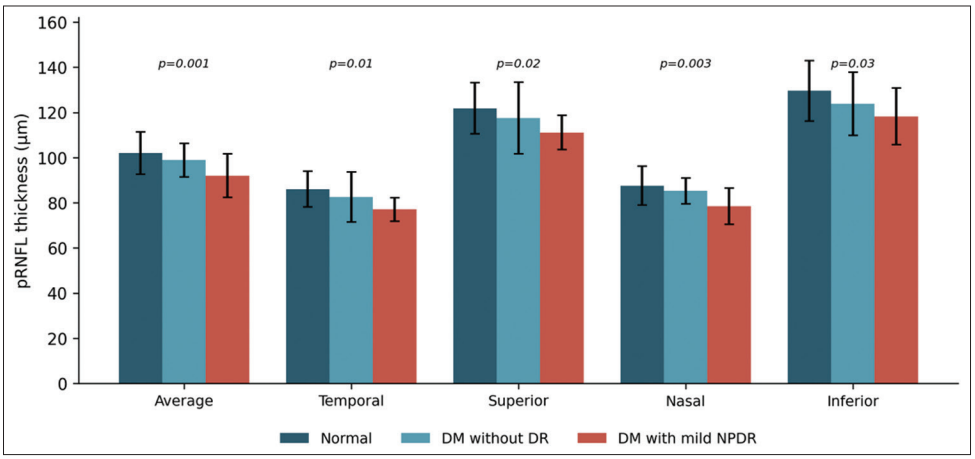

Figure 2: Peripapillary retinal nerve fiber layer thickness (3D-Disc scan) across study groups. Error bars represent standard deviation

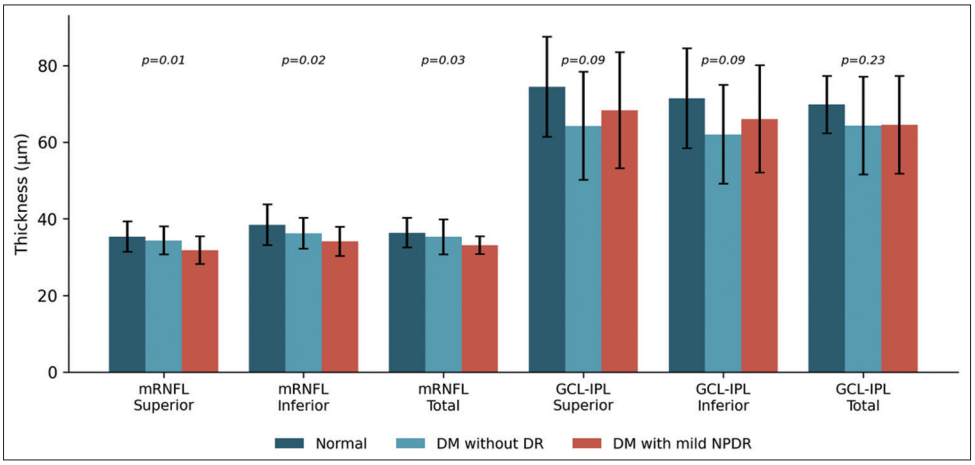

Figure 3: Macular retinal nerve fiber layer and ganglion cell layer–inner plexiform layer thickness (3D-Macula[v] scan) across study groups. Error bars represent standard deviation

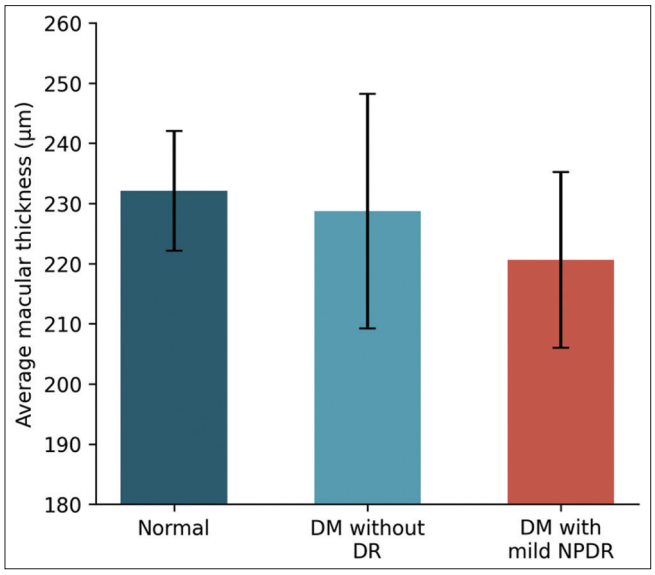

Figure 4: Average macular thickness (3D-Macula scan) across study groups (*P*=0.06)

retinal neurodegeneration precedes clinically detectable microvascular disease.

GCL-IPL Thickness (3D-Wide Scan)

The 3D-Wide scan showed significant GCL-IPL thinning in all six sectors in both diabetic groups versus controls, consistent with Carpineto *et al.*[21] Jia *et al.*, using a central-plus-four-quadrant protocol rather than a wide sectorial map, found no significant loss – highlighting how scan protocol affects detection sensitivity.[22] Chhablani *et al.* found greatest loss in the supero-nasal and infero-nasal quadrants, postulating that thicker nasal fibers allow more measurable change.[23] Ghita *et al.* found GCL-IPL parameters correlate closely with pRNFL findings, supporting combined analysis as a more sensitive marker than either alone.[24]

pRNFL Thickness (3D-Disc Scan)

Average pRNFL thickness and all four quadrants were significantly reduced in our diabetic cohort. This is broader than Shi *et al.*, who found quadrant-specific thinning (superior and inferior only) that intensified with DR severity, with superior thinning linked to diabetes duration.[25] Our pan-quadrant thinning even in mild NPDR may reflect an older cohort or protocol differences. Jia *et al.* localized early thinning to the inferior/temporal regions and papillomacular bundle, suggesting fiber-density-dependent vulnerability.[22] Demir *et al.* and Dhasmana *et al.*, using different OCT platforms in Indian cohorts, similarly found significant RNFL thinning in diabetics, supporting generalizability across platforms and populations.[26,27]

Macular mRNFL and GCL-IPL (3D-Macula[v] Scan)

In the macular region, mRNFL thinning was significant in the superior, inferior, and overall measurements, whereas GCL–IPL thinning on the 7 × 7 mm macular scan did not reach significance. This differs from Carpineto *et al.*, who reported significant macular GCL–IPL thinning in both no-DR and mild NPDR eyes.[21] A possible explanation is that averaging across broad superior and inferior regions may mask spatially localized loss detectable only with finer sectorial or quadrant-based analyses, as reported by Chhablani *et al.* and Jia *et al.*[22,23] These findings suggest that, with the scan protocol used, mRNFL may be a more sensitive early macular marker than GCL–IPL thickness.

Average Macular Thickness (3D-Macula Scan)

Average macular thickness did not differ significantly among our groups, though the direction of change (progressively thinner with worsening diabetic status) was consistent with the neurodegenerative pattern seen in the other scans. This is in keeping with literature suggesting that global macular thickness is a relatively insensitive early marker compared with layer-specific segmentation, since early subclinical fluid shifts or compensatory changes in other retinal layers may transiently offset neuronal thinning before overt diabetic macular edema develops – edema itself being a well-recognized, distinct OCT-detectable entity in more advanced diabetic eye disease that was deliberately excluded from our cohort to isolate pure neurodegenerative change.[28-30]

Clinical Implications

Taken together, our findings support the concept that diabetic retinal disease has an early neurodegenerative component detectable before clinically apparent retinopathy, particularly on wide-field peripapillary and mRNFL scans. Advances in SD-OCT and swept-source OCT have enhanced the detection and monitoring of these early structural changes.[31-33] Incorporating RNFL and GCL-IPL assessment into routine diabetic eye screening, especially in patients with longer disease duration, may facilitate earlier risk stratification, although this requires validation in longitudinal studies.

CONCLUSION

RNFL and GCL-IPL thinning occur in a stepwise fashion with increasing severity of diabetic retinal disease and are measurable even before clinically visible DR develops. Thinning was most consistently and significantly demonstrated on the wide-field peripapillary (3D-Disc) and mRNFL (3D-Macula[v]) scans, while macular GCL-IPL and average macular thickness showed a similar directional trend without reaching statistical significance. These findings support the role of SD-OCT-based RNFL and GCL-IPL measurement as a potential early, quantifiable structural biomarker for identifying diabetic retinal

neurodegeneration in patients with T2DM, even in the absence of overt microvascular retinopathy, and may aid earlier risk stratification and monitoring in clinical practice.

ETHICS

Approved by the Institutional Ethics Committee, R.D. Gardi Medical College, Ujjain; written informed consent obtained from all participants.

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