

Macular thickness in patients with type 2 diabetes without clinical diabetic retinopathy: a cross-sectional study

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Товщина макули у пацієнтів з цукровим діабетом 2 типу без клінічної діабетичної ретинопатії: крос-секційне дослідження

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Abstract

Background. Diabetic retinopathy (DR) is a major cause of visual impairment. Optical coherence tomography (OCT) enables the detection of early retinal structural changes before clinically evident retinopathy; however, findings on macular thickness in diabetes without retinopathy remain inconsistent, and data from Iraqi populations are limited. This study aimed to evaluate macular thickness in Iraqi patients with diabetes mellitus (DM) without clinical DR.

Methods. This observational, analytical cross-sectional study was conducted from January 2025 to January 2026 and included 53 participants (26 patients with DM without DR and 27 healthy controls). All participants underwent a standardized ophthalmic examination. Macular thickness was measured using spectral-domain OCT (Topcon 3D OCT). Only the right eye was analyzed to avoid inter-eye correlation. Central subfield macular thickness (CSMT) and ET-DRS-based regional thickness were recorded.

Results. The diabetes group was significantly older than controls ($p < 0.001$). CSMT differed significantly between

groups in unadjusted analysis ($p < 0.0001$). However, after adjustment for age, the direction of this association was reversed, indicating a strong confounding effect. No significant differences were observed in the parafoveal region. In the perifoveal region, the nasal subfield was significantly thicker ($p = 0.004$), and this difference remained significant after age adjustment ($p = 0.009$), while the temporal difference was not significant after adjustment. ROC analysis showed high discriminative ability of CSMT ($AUC = 0.965$), with a cutoff of $\leq 236 \mu\text{m}$ yielding high sensitivity (96.2%) and specificity (88.9%).

Conclusions. Patients with DM without clinical DR demonstrated alterations in macular thickness, including localized nasal perifoveal thickening, suggesting early retinal structural changes detectable by OCT. While CSMT demonstrated strong discriminative performance, its sensitivity to age-related effects limits its reliability as a standalone biomarker. These findings are exploratory and require confirmation in larger, well-controlled studies.

Keywords: diabetic retinopathy; diabetes mellitus; macula; optical coherence tomography; retinal thickness.

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Резюме

Діабетична ретинопатія (ДР) є однією з основних причин порушення зору. Оптична когерентна томографія (ОКТ) дозволяє виявляти ранні структурні зміни сітківки ще до появи клінічних ознак ретинопатії. Однак дані щодо товщини макули у пацієнтів з цукровим діабетом без ДР залишаються суперечливими, а інформація щодо іракської популяції обмежена. Метою цього дослідження було оцінити товщину макули у пацієнтів з цукровим діабетом (ЦД) без клінічної ДР.

Методи. Це обсерваційне аналітичне поперечне дослідження було проведено з січня 2025 року по січень 2026 року та включало 53 учасники (26 пацієнтів з ЦД

без ДР та 27 здорових осіб контрольної групи). Усі учасники пройшли стандартизоване офтальмологічне обстеження. Товщину макули вимірювали за допомогою спектрально-доменної ОКТ (Торсон 3D ОСТ). Для уникнення міжочного кореляційного впливу аналізували лише праве око. Оцінювали центральну товщину макули (CSMT) та регіональну товщину відповідно до сітки ETDRS.

Результати. Група пацієнтів з ЦД була статистично значуще старшою за контрольну групу ($p < 0,001$). CSMT достовірно відрізнялася між групами в некоригованому аналізі ($p < 0,0001$). Однак після корекції за віком напрямків цього зв'язку змінився на протилежний, що свідчить про значний вплив конфаундингу. У парафовеальній зоні статистично значущих відмінностей не виявлено. У перифовеальній зоні товщина в носовому підполі була значно більшою ($p = 0,004$), і ця різниця залишалася статистично значущою після корекції за віком ($p = 0,009$), тоді як різниця в скроневому підполі втратила статистичну

значущість після корекції. ROC-аналіз показав високу дискримінаційну здатність CSMT ($AUC = 0,965$) з пороговим значенням ≤ 236 мкм, що забезпечує високу чутливість (96,2%) та специфічність (88,9%).

Висновки. У пацієнтів з ЦД без клінічних ознак ДР виявлено зміни товщини макули, зокрема локалізоване потовщення в носовому перифовеальному підполі, що свідчить про ранні структурні зміни сітківки, які можуть бути виявлені за допомогою ОКТ. Хоча CSMT продемонструвала високу дискримінаційну здатність, її чутливість до вікових впливів обмежує надійність як самостійного біомаркера. Ці результати є попередніми та потребують підтвердження у більших, добре контрольованих дослідженнях.

Ключові слова: діабетична ретинопатія; цукровий діабет; макула; оптична когерентна томографія; товщина сітківки

Introduction

Diabetic retinopathy (DR) and diabetic macular edema are common vision-threatening complications of diabetes mellitus (DM) [1]. Macular involvement is a leading cause of blindness, and assessing retinal thickness is clinically important [2]. Clinically, macular edema detection depends on the biomicroscopic examination of the fundus [2, 3]. Retinal thickness can be objectively and reproducibly measured using Optical Coherence Tomography (OCT), which is widely used to assess macular thickness abnormalities [4]. OCT distinguishes between two groups of thickness disorders: macular thickening and thinning [4, 5].

The latest IDF Diabetes Atlas (2025) reports that approximately 11% of the adult population lives with DM, and this number is projected to increase to more than 850 million by 2050 [6]. Statistically, all type 1 DM and greater than 50% of type 2 DM patients develop at least one form of DR [1]. Visual impairment is caused by proliferative DR and diabetic macular disease (edema and ischemia) [1], mostly in type-2 DM. Traditionally, macular disease has been diagnosed by slit-lamp biomicroscopy and fundus fluorescein angiography [4, 5]. However, new non-invasive imaging techniques provide great detail inside-the-eye layer-by-layer pictures of the retina, allowing consistent checking of thickness and size in the macula [5]. On the other hand, spectral-domain optical coherence tomography (SD-OCT) is a new third-generation high-resolution OCT instrument that provides many advantages, including quick data acquisition time, three-dimensional reconstruction capabilities for retinal imaging, enhanced visualization of fine details within retinal microstructures, as well as the ability to detect very subtle changes within the foveal region not visible clinically. It plays an important role in research studies as well as in clinical practice regarding diagnosis, management, and follow-up assessment concerning foveal edema [5, 7].

SD-OCT-based studies have shown that diabetes can cause significant changes in the structure of retinal layers [8]. Inner retinal layer thinning has been noted as an early neurodegenerative change, possibly even before DR clinically manifests. Conversely, outer retinal layer thickening is seen in eyes with established diabetic retinopathy when compared to diabetic eyes without retinopathy. Published findings are not fully consistent [3, 5]. Some reports describe retinal thinning that may reflect early neurodegeneration; others show localized thickening or altered perfusion in selected macular regions [9, 10]. In addition, evidence from Middle Eastern populations remains limited, and data from Iraqi patients are scarce. Accordingly, the present study aimed to assess macular thickness in Iraqi patients with early diabetes and to determine whether structural changes are present before clinical retinopathy develops.

Methods

Study design and population

This was an observational, analytical cross-sectional study conducted at Al-Kindy Teaching Hospital (Baghdad, Iraq) from January 2025 to January 2026. Ethical approval was obtained from the Institutional Review Board of the Department of Ophthalmology, Al-Kindy Teaching Hospital (No. 2033, 2024). Written informed consent was obtained from all participants in accordance with the principles of the Declaration of Helsinki.

No formal a priori sample size calculation was performed. Although previous studies have evaluated macular thickness in patients with DM without DR, their findings are heterogeneous and do not provide a consistent basis for reliable effect size estimation. Therefore, given the exploratory nature of the study and the lack of reliable preliminary data for this specific population, all eligible partici-

pants who presented during the study period and met the inclusion criteria were consecutively included.

Inclusion criteria

1. Patients diagnosed with type 2 DM without clinical signs of DR

2. Age ≥ 18 years

3. Ability to undergo an OCT examination, defined as adequate fixation, sufficient patient cooperation, and absence of significant media opacity affecting image quality.

Exclusion criteria

1. Macular diseases (macular edema, macular holes, age-related macular degeneration)

2. Uveitis

3. Severe myopia (spherical equivalent ≤ -6.00 diopters)

4. Glaucoma

5. Vascular occlusive diseases

6. Cataract affecting the OCT image quality

7. Previous ocular interventions (intravitreal therapy, retinal laser treatment, or vitrectomy)

Exclusion criteria were assessed based on clinical ophthalmic examination, OCT findings, and review of medical records.

DM was defined as a prior clinical diagnosis of type 2 DM and/or current use of antidiabetic therapy, as documented in the medical records.

The absence of DR was confirmed by a comprehensive fundus examination performed by an ophthalmologist. OCT was additionally used to assess macular structure and exclude macular pathology.

The control group consisted of individuals without DM who attended the same clinic for routine ophthalmic examination and had no history of retinal disease. The absence of diabetes was based on medical history and patient report. Participants were not age-matched; therefore, age was included as a covariate in the statistical analysis. Demographic data (age and sex) were obtained through direct patient interviews.

Ocular examination and OCT assessment

All participants underwent a standardized ophthalmic evaluation to determine eligibility for inclusion in the study. Fundus examination was performed by an ophthalmologist to confirm the absence of clinical DR in the diabetic group and to exclude other retinal or macular pathologies in both groups. Examiners were not blinded to group allocation, as assessments were performed in a routine clinical setting.

Macular thickness was assessed using an SD-OCT device (TOPCON 3D OCT, Topcon Corporation, Tokyo, Japan) [16-18]. All OCT scans were performed under standardized conditions by a trained operator.

Only high-quality scans with correct segmentation were included based on signal strength and segmentation quality provided by the device software. Scans with motion artifacts, poor fixation, or segmentation errors were excluded to ensure measurement accuracy.

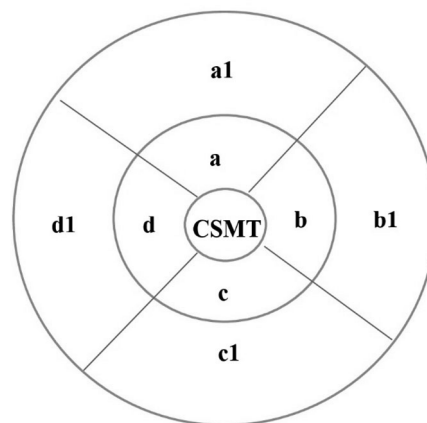


Figure 1. ETDRS macular grid showing CSMT, perifoveal (outer ring), and parafoveal (inner ring) subfields.

Macular thickness measurements were obtained using the built-in macular mapping software based on the Early Treatment Diabetic Retinopathy Study (ETDRS) grid. The ETDRS grid divides the macula into central, perifoveal (outer ring), and parafoveal (inner ring) regions.

Central subfield macular thickness (CSMT) was defined as the mean retinal thickness in the area of a circle with a 1-mm diameter centered on the macula. Besides the subfield central, macular thickness was also assessed in eight contiguous regions: four parafoveal subfields – superior, nasal, inferior, and temporal, and four perifoveal subfields – superior, nasal, inferior, and temporal. All measurements were taken with the same OCT device and scan protocol to maintain consistency between subjects. Figure 1 shows the ETDRS macular grid applied for thickness analysis.

Statistical analysis

Statistical analysis was performed using IBM SPSS Statistics (version 26.0; IBM Corp., Armonk, NY, USA). Normality of data distribution was assessed using the Shapiro–Wilk test. Continuous variables are presented as mean and the standard deviation ($M \pm SD$) or median and interquartile range [Me (Q25-Q75)], according to data distribution. Categorical variables are presented as frequencies and percentages.

To ensure independence of observations and avoid inter-eye correlation, only one eye per participant (the right eye) was included in the analysis. Between-group comparisons were performed using the independent-samples t-test for normally distributed variables and the Mann–Whitney U test for non-normally distributed variables. Effect sizes are reported as mean differences or Hodges–Lehmann median differences with corresponding 95% confidence intervals (95% CI). For variables showing statistically significant differences in univariate analysis, analysis of covariance (ANCOVA) was performed to evaluate group differences after adjustment for age. Assumptions of ANCOVA, including homogeneity of variances and homoge-

neity of regression slopes, were assessed using Levene's test ($p > 0.05$ for all models).

Correlation between age and macular thickness parameters was evaluated using Spearman's rank correlation coefficient.

Receiver operating characteristic (ROC) curve analysis was performed as an exploratory assessment of the discriminative ability of CSMT. A p -value < 0.05 was considered statistically significant.

A post hoc power analysis was conducted based on the observed difference in CSMT between groups to provide additional context for the observed effect size. Statistical power was estimated using G*Power software (version 3.1.9.7; Heinrich Heine University, Düsseldorf, Germany) for a two-tailed independent-samples t -test at a significance level of 0.05.

Results

A total of 53 participants were included in the study, comprising 26 patients with type 2 DM without DR and 27 healthy controls. The diabetes group was significantly older than the control group ($p < 0.001$), while sex distribution, visual acuity, refractive error, and OCT signal strength were similar between the groups (Table 1).

The overall CSMT of the right eye was significantly greater in the DM without DR group compared with controls ($p < 0.0001$). A post hoc power analysis demonstrated a large effect size (Cohen's $d = 2.11$). Using a two-tailed independent-samples t -test with an alpha level of 0.05 and the present sample sizes ($n = 26$ and $n = 27$), the estimated statistical power ($1 - \beta$) was > 0.99 , indicating high statistical power for the observed difference.

Detailed macular thickness measurements across ETDRS subfields are presented in Table 2.

Table 1. Baseline characteristics of the study participants.

Variable	DM without DR (n=26)	Control (n=27)	Mean difference (95% CI)	p-value
Age (years)	56.73 \pm 8.04	46.56 \pm 9.15	10.17 (5.44; 14.90)	<0.001
Sex, n (%)				0.68
• Male	13 (50.0)	15 (55.6)	—	
• Female	13 (50.0)	12 (44.4)	—	
Visual acuity (LogMAR)	0.19 \pm 0.14	0.15 \pm 0.12	0.04 (-0.03; 0.11)	0.27
Spherical equivalent (D)	-0.31 \pm 2.11	-1.12 \pm 2.32	0.81 (-0.39; 2.01)	0.18
OCT signal strength	7.6 \pm 1.2	7.8 \pm 0.92	-0.20 (-0.78; 0.38)	0.49
CSMT (μ m), right eye	250.26 \pm 14.97	218.92 \pm 14.70	31.34 (23.32; 39.36)	<0.001

Data are presented as $M \pm SD$. Between-group differences are expressed as mean differences with 95% CI. P-values were calculated using the independent-samples t -test. Abbreviations: D, diopters; μ m, micrometers; LogMAR, logarithm of the minimum angle of resolution; CSMT, central subfield macular thickness; OCT, optical coherence tomography.

Table 2. Macular thickness measurements in ETDRS subfields (right eye).

Parameter (μ m)	Control (n=27)	DM without DR (n=26)	Difference (95% CI)	p-value
Parafoveal				
Superior	277.89 \pm 11.46	278.18 \pm 11.54	0.29 (-4.77; 5.37)	0.904
Nasal	294.33 \pm 8.87	288.11 \pm 9.77	-6.22 (-11.11; -2.33)	0.003
Inferior	276.41 \pm 8.18	273.66 \pm 10.25	-2.75 (-7.62; 2.12)	0.261
Temporal	301 (290–309.75)	299.5 (290–310)	-1.0 (-8.0; 7.0)	1.000
Perifoveal				
Superior	280 (274–287.75)	276.5 (275–283)	-3.0 (-9.0; 4.0)	0.748
Nasal	287.62 \pm 6.96	294.33 \pm 8.87	6.71 (2.40; 11.02)	0.004
Inferior	277 (270.5–282)	274 (269–279)	-3.0 (-8.0; 4.0)	0.407
Temporal	264 (260–270)	259.5 (257–264)	-4.0 (-8.0; -1.0)	0.047

Data are presented as $M \pm SD$ and Me (Q25–Q75), according to data distribution. Between-group differences are expressed as mean differences with 95% CI for normally distributed variables and as Hodges–Lehmann median differences with 95% CI for non-normally distributed variables. P-values were calculated using the independent-samples t -test or Mann–Whitney U test, as appropriate. Abbreviations: DM, diabetes mellitus; DR, diabetic retinopathy.

Table 3. Adjusted analysis of macular thickness (ANCOVA, right eye).

Parameter (μm)	Adjusted Mean (Control)	Adjusted Mean (DM without DR)	F-value	p-value	Partial η^2
CSMT	246.07	223.27	29.53	<0.001	0.37
Perifoveal nasal	294.32	287.63	6.68	0.013	0.12
Perifoveal temporal	263.13	268.63	0.64	0.428	0.01

Abbreviations: CSMT, central subfield macular thickness; DM, diabetes mellitus; DR, diabetic retinopathy.

In the parafoveal region, no statistically significant differences were observed between groups in any of the subfields (superior, nasal, inferior, or temporal). In contrast, within the perifoveal region, a statistically significant increase in thickness was observed in the nasal subfield in the DM without DR group compared with controls ($p = 0.004$). A statistically significant difference was also noted in the temporal perifoveal subfield ($p = 0.047$). No significant differences were found in the superior or inferior parafoveal subfields.

To account for the effect of age, analysis of covariance (ANCOVA) was performed for variables that were significant in univariate analysis (Table 3).

After adjustment for age, the difference in CSMT between groups remained statistically significant ($F = 29.53$, $p < 0.001$), with a large effect size (partial $\eta^2 = 0.37$). The adjusted mean CSMT was lower in the DM without DR group compared with controls. Similarly, the perifoveal nasal subfield remained significantly different between groups ($F = 6.68$, $p = 0.013$), with a moderate effect size (partial $\eta^2 = 0.12$). In contrast, no significant difference was observed in the perifoveal temporal subfield ($p = 0.428$), with a negligible effect size (partial $\eta^2 = 0.01$).

Correlation analysis demonstrated a significant negative association between age and CSMT, indicating that macular thickness decreases with increasing age (Spearman $\rho = -0.64$, $p < 0.001$; Figure 2).

ROC analysis demonstrated high discriminative performance of CSMT between groups, with an area under the curve (AUC) of 0.965 ($p < 0.001$). In this cohort, a cut-off value of $\leq 236 \mu\text{m}$ yielded high sensitivity (96.2%) and specificity (88.9%) (Figure 3). However, this analysis was performed as an exploratory assessment and should be interpreted with caution, as the model was derived and tested on the same dataset, has not been externally validated, and does not imply clinical or diagnostic applicability.

Representative OCT images are shown in Figure 4 (see cover page 2). Control subjects demonstrated normal foveal contour and thickness distribution, whereas patients with diabetes showed subtle structural irregularities and localized variations in macular thickness.

Discussion

The present study provides new data on macular thickness in Iraqi patients with early DM before the development of clinical DR, a population that is underrepresented in current literature. Using spectral-domain OCT, it focus-

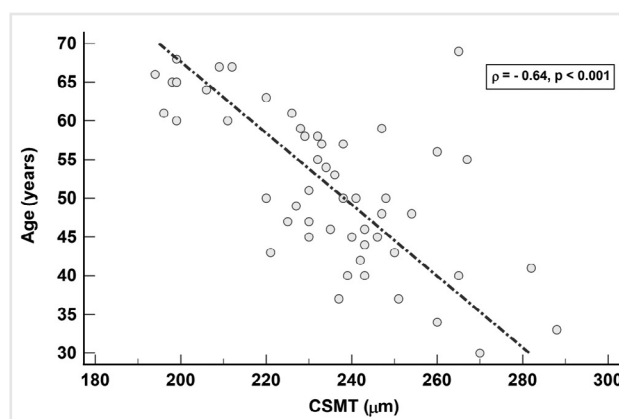


Figure 2. Scatter plot showing the relationship between age and CSMT.

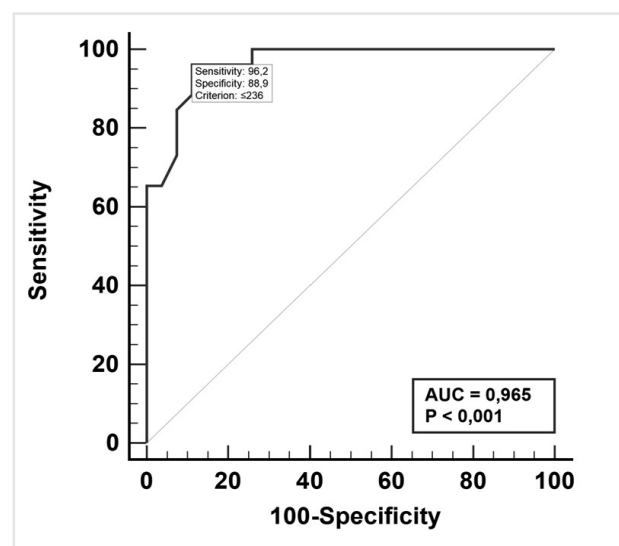


Figure 3. ROC curve analysis of CSMT showing its discriminative performance between patients with DM without DR and control subjects.

es on detecting early structural retinal changes that are not visible on routine examination.

The main finding of our study is that CSMT was significantly higher in patients with DM without DR compared with healthy controls. In addition, localized thickening was observed in the perifoveal nasal subfield, while no significant differences were found in the parafoveal re-

gion. After adjustment for age, the direction of the association for CSMT was attenuated and reversed. This finding suggests that age exerts a substantial confounding effect on macular thickness measurements. Importantly, this reversal suggests that the initial unadjusted finding may be misleading and that age substantially influences both the magnitude and interpretation of CSMT differences.

ROC analysis showed that CSMT had high discriminative ability between groups (AUC = 0.965), with high sensitivity and specificity at the identified cutoff value. However, this finding should be interpreted as exploratory, as the analysis was performed on the same dataset and does not imply clinical applicability. A negative association between age and CSMT was also observed, indicating that macular thickness tends to decrease with increasing age, which is consistent with known age-related retinal thinning patterns [11, 12].

Taken together, our results indicate that early diabetic retinal changes are localized and region-specific. The presence of differences in the perifoveal region, with no corresponding changes in the parafoveal region, suggests that early structural alterations may initially affect more peripheral macular zones rather than the central inner ring.

Previous studies have reported inconsistent findings regarding macular thickness in patients with DM without DR. Some studies have demonstrated increased macular thickness, suggesting early subclinical edema or vascular dysfunction [13, 14], while others have reported thinning of the macula, which has been attributed to early neurodegenerative changes in the retina [15, 16]. For example, the Maastricht study found central macular thinning in patients with DM without DR, due to early neurodegeneration [16, 17]. Karti et al. also showed reduced ganglion cell layer thickness in early DM without DR [17]. In contrast, Wei et al. reported increased macular thickness linked to risk factors like disease duration and lipids in early diabetic retinopathy [13]. Shah et al. found thicker inner retinal layers over 7 years in young type 2 DM patients, supporting early subclinical edema [14]. These differences may be explained by variations in study design, OCT technology, patient characteristics, glycemic control, and the retinal regions analyzed. In addition, some studies assess total retinal thickness, whereas others evaluate individual retinal layers, which may show different patterns of change [5, 18].

Overall, these findings can be interpreted within two main mechanisms: microvascular dysfunction leading to retinal thickening, and neurodegenerative processes leading to thinning [13, 19]. The variation between studies likely reflects differences in study populations, disease stage, OCT methodology, and whether total or layer-specific measurements were analyzed.

In this context, the findings of the present study are more consistent with studies reporting localized thickening [14, 20]. However, they also demonstrate that this pattern is not generalized across the macula and is influenced

by age. This helps explain part of the inconsistency in the literature, as age adjustment is not uniformly applied in previous studies. Therefore, the results of this study support a combined mechanism, where early microvascular alterations and neurodegeneration may coexist and produce heterogeneous structural changes, including localized areas of both thickening and thinning. The observed nasal perifoveal thickening may represent a region of increased susceptibility to early microvascular dysfunction.

There are several limitations in the study. The number of participants was relatively small; therefore, the results may not be conclusive for a larger population. Since our study is cross-sectional, it does not allow us to conclude causation or progression of DR. The DM without DR group was significantly older than the control group, and although statistical adjustment was performed, residual confounding by age cannot be fully excluded. In addition, the study was conducted at a single center, and the lack of examiner blinding may introduce potential assessment bias. Important clinical variables, including duration of diabetes, glycemic control (HbA1c), and treatment status, were not collected. These factors may influence macular thickness and represent potential sources of residual confounding. Functional visual assessments were also not performed, and thus, structural findings could not be correlated with visual function. The ROC analysis results should be interpreted with caution since the cutoff value was derived from a relatively small sample and was not externally validated. Therefore, its clinical applicability may be limited. Although one eye per participant was analyzed to avoid inter-eye correlation, this approach may not fully reflect bilateral retinal involvement. In addition, measurements were obtained using a single OCT device, and results may vary with different instruments or segmentation algorithms. Future longitudinal studies with larger sample sizes, external validation cohorts, and inclusion of metabolic and functional parameters are needed to confirm these findings and determine their role in predicting the development of diabetic retinopathy.

Conclusion

Patients with DM without clinical DR demonstrated significant alterations in macular thickness, suggesting early retinal structural changes in diabetes. Although CSMT differed significantly between groups, the change in its direction after age adjustment indicates that this finding is influenced by confounding factors and should be interpreted with caution. These results suggest that CSMT alone may not be a stable marker of early diabetic retinal changes, and localized alterations, particularly in the perifoveal region, may provide additional insight.

Overall, these findings support the ability of OCT to detect early retinal changes in diabetes. However, their clinical relevance remains uncertain and requires confirmation in larger, well-controlled, and longitudinal studies.

Author Contributions

Sura LK: conceptualization; data curation; investigations; methods; project administration; resource; software; writing – original draft and writing – reviews and editions.

Disclaimers

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Declaration of Generative AI and AI-assisted technologies in the writing process

During the preparation of this work, the author used the InstaText Word extension to improve the style and grammar of the manuscript. After using this tool, the author reviewed and edited the content as needed and takes full responsibility for the content of the publication.

Conflict of Interest

The author declare that they have no conflicts of interest related to this work.

Data Availability Statement

The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

Abbreviations

DM – diabetes mellitus, DR – diabetic retinopathy, OCT – Optical Coherence Tomography, SD-OCT – spectral-domain optical coherence tomography, CSMT – Central subfield macular thickness.

References

- Zhang J, Zhang J, Zhang C, Zhang J, Gu L, Luo D, et al. Diabetic Macular Edema: Current Understanding, Molecular Mechanisms and Therapeutic Implications. *Cells*. 2022;11(21):3362. doi: 10.3390/cells11213362.
- Kalaw FGP, Sharma P, Walker E, Borooah S. Differences in macular thickness associated with peripheral retinal vessel whitening in diabetic patients. *Sci Rep*. 2024;14(1):19881. doi: 10.1038/s41598-024-68839-0.
- Chondrozoumakis G, Chatzimichail E, Habra O, Vounotrypidis E, Papanas N, Gatziofufas Z, et al. Retinal Biomarkers in Diabetic Retinopathy: From Early Detection to Personalized Treatment. *J Clin Med*. 2025;14(4):1343. doi: 10.3390/jcm14041343.
- Badaró E, Novais E, Prodócimo LM, Sallum JM. Spectral-domain optical coherence tomography for macular edema. *ScientificWorldJournal*. 2014;2014:191847. doi: 10.1155/2014/191847.
- Szeto SK, Lai TY, Vujosevic S, Sun JK, Sadda SR, Tan G, et al. Optical coherence tomography in the management of diabetic macular oedema. *Prog Retin Eye Res*. 2024;98:101220. doi: 10.1016/j.preteyeres.2023.101220. Erratum in: *Prog Retin Eye Res*. 2025;104:101319. doi: 10.1016/j.preteyeres.2024.101319.
- Duncan BB, Magliano DJ, Boyko EJ. IDF Diabetes Atlas 11th edition 2025: global prevalence and projections for 2050. *Nephrol Dial Transplant*. 2025;41(1):7-9. doi: 10.1093/ndt/gfaf177.
- Mandloi G, Jain A, Sharma A, Tirkey ER, Jain S. Evaluation of Spectral Domain OCT Changes Following Anti-VEGF Therapy in Diabetic Macular Edema. *J Pharm Bioallied Sci*. 2026;18(Suppl 1):S212-S214. doi: 10.4103/jpbs.jpbs_1238_25.
- Tang Z, Chan MY, Leung WY, Wong HY, Ng CM, Chan VTT, et al. Assessment of retinal neurodegeneration with spectral-domain optical coherence tomography: a systematic review and meta-analysis. *Eye (Lond)*. 2021;35(5):1317-1325. doi: 10.1038/s41433-020-1020-z.
- Vaughan M, Denmead P, Tay N, Rajendram R, Michaelides M, Patterson E. How early can we detect diabetic retinopathy? A narrative review of imaging tools for structural assessment of the retina. *Graefes Arch Clin Exp Ophthalmol*. 2025;263(9):2413-2425. doi: 10.1007/s00417-025-06828-3.
- Dai Y, Zheng D, Zhao J, Wang K, Fu B, Xu Z, et al. Macular Neural and Microvascular Alterations in Type 2 Diabetes Without Retinopathy: A SS-OCT Study. *Am J Ophthalmol*. 2024;262:229-236. doi: 10.1016/j.ajo.2024.02.034.
- Peto T, Evans RN, Reeves BC, Harding S, Madhusudhan S, Lotery A, et al. Long-term Retinal Morphology and Functional Associations in Treated Neovascular Age-Related Macular Degeneration: Findings from the Inhibition of VEGF in Age-Related Choroidal Neovascularisation Trial. *Ophthalmol Retina*. 2022;6(8):664-675. doi: 10.1016/j.oret.2022.03.010.
- Toprak I, Fenkci SM, Fidan Yaylali G, Martin C, Yaylali V. Early retinal neurodegeneration in preclinical diabetic retinopathy: a multifactorial investigation. *Eye (Lond)*. 2020;34(6):1100-1107. doi: 10.1038/s41433-019-0646-1.
- Wei Q, Qiu W, Liu Q, Jiang Y. Relationship Between Risk Factors and Macular Thickness in Patients with Early Diabetic Retinopathy. *Int J Gen Med*. 2022;15:6021-6029. doi: 10.2147/IJGM.S366348.
- Shah J, Tan B, Wong D, Abdul Gani NFB, Hu Q, Liu X, et al. Evaluation of thickness of individual macular retinal layers in diabetic eyes from optical coherence tomography. *Sci Rep*. 2024;14(1):17909. doi: 10.1038/s41598-024-68552-y.
- Boned-Murillo A, Fernández-Espinosa G, Orduna-Hospital E, Díaz-Barreda MD, Sánchez-Cano A, Sopeña-Pinilla M, et al. Changes in Inner Retina Thickness and Macular Sensitivity in Patients with Type 2 Diabetes with Moderate Diabetic Retinopathy. *Biomedicines*. 2023;11(11):2972. doi: 10.3390/biomedicines11112972.
- De Clerck EEB, Schouten JSAG, Berendschot TTJM, Goezinne F, Dagnelie PC, Schaper NC, et al. Macular thinning in prediabetes or type 2 diabetes without diabetic retinopathy: the Maastricht Study. *Acta Ophthalmol*. 2018;96(2):174-182. doi: 10.1111/aos.13570.
- Karti O, Nalbantoglu O, Abali S, Ayhan Z, Tunc S, Kusbeci T, et al. Retinal Ganglion Cell Loss in Children With Type 1 Diabetes Mellitus Without Diabetic Retinopathy. *Ophthalmic Surg Lasers Imaging Retina*. 2017;48(6):473-477. doi: 10.3928/23258160-20170601-05. Erratum in: *Ophthalmic Surg Lasers Imaging Retina*. 2017;48(7):530. doi: 10.3928/23258160-20170630-02.
- Boned-Murillo A, Diaz-Barreda MD, Ferreras A, Bartolomé-Sesé I, Orduna-Hospital E, Montes-Rodríguez P, et al.

- Structural and functional findings in patients with moderate diabetic retinopathy. *Graefes Arch Clin Exp Ophthalmol.* 2021;259(12):3625-3635. doi: 10.1007/s00417-021-05277-y.
19. Viganò I, Galbiati S, Aragona E, Gabellini D, Lattanzio R, Pedon V, et al. Diabetes-Driven Retinal Neurodegeneration: Its Role in the Pathogenesis of Diabetic Retinopathy. *Biomedicines.* 2025;13(6):1328. doi: 10.3390/biomedicines13061328.
20. Lin CY, Sheen YJ, Chen HM, Lu YA, Chen JP, Huang HE, et al. Vulnerable parafoveal microcirculation quadrant in patients with type 2 diabetes mellitus. *Sci Rep.* 2025;15(1):1237. doi: 10.1038/s41598-024-85021-8.

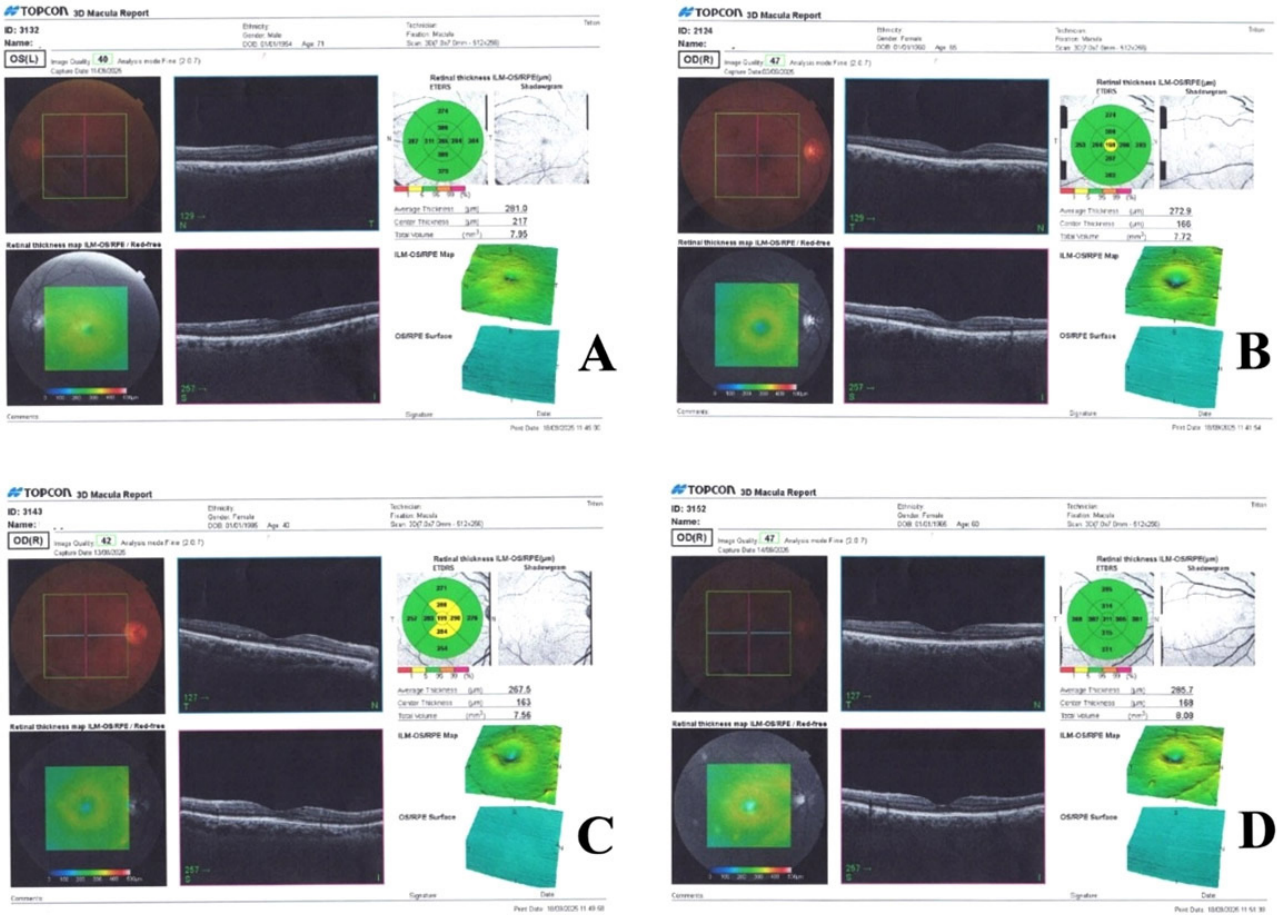


Figure 4. Representative Topcon 3D macular OCT images. (A, C) Normal macular morphology in control subjects, showing preserved foveal contour and normal retinal thickness maps. (B, D) Subtle structural alterations in patients with DM without DR, including mild irregularity of the foveal contour and localized changes in thickness distribution.

Фото до статті Канцер К. С. з співавт. «Клінічні особливості травматичних розривів макули при контузії очного яблука та при мінно-вибуховій травмї ока»

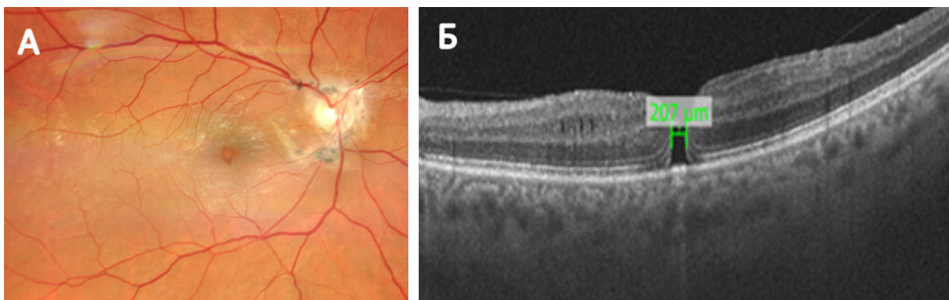


Рис. 1. Травматичний розрив макули внаслідок контузії очного яблука.
А – фундус-фото центрального відділу;
Б – ОКТ макули.

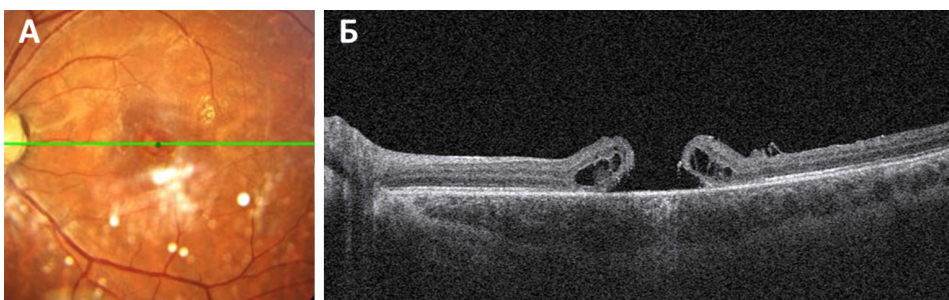


Рис. 2. Травматичний розрив макули внаслідок мінно-вибухової травми.
А – фундус-фото центрального відділу;
Б – ОКТ макули: базальний розмір розриву – 1575 мкм, мінімальний розмір розриву – 905 мкм.