Biomedicine Advances

Biomed adv 2026;3(3):111-116
doi:10.34172/bma.72
https://biomedad.ae

Original Article

Unveiling Retinal Effects of SGLT2 Inhibition: A Retrospective Analysis of Empagliflozin on Macular Thickness in Type 2 Diabetes with Macular Edema

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Summary

Introduction: Diabetic macular edema (DME) continues to be a primary cause of visual impairment in non-proliferative diabetic retinopathy. Sodium–glucose cotransporter-2 inhibitors (SGLT2i) are suggested to provide vasculoprotective benefits and may have pleiotropic effects that could improve macular edema in patients with DME. This study aimed to determine whether empagliflozin use in routine clinical care is associated with differences in central macular thickness at a ~8-week follow-up, through retrospective analysis of medical records.

Methods: This retrospective observational before-and-after study included patients aged 45–75 years with type 2 diabetes mellitus and OCT-confirmed DME in whom empagliflozin therapy was initiated as part of routine clinical care. Central macular thickness (CMT) and visual acuity were extracted from medical records at baseline and approximately 8 weeks after treatment initiation.

Results: Forty-seven eyes from 30 patients (mean age: 66.9 ± 8.4 years) were analyzed. Mean central subfield thickness (CST) decreased from 400.4 ± 84.8 µm at baseline to 344.3 ± 76.7 µm at follow-up, representing a mean reduction of 14.0% (56.06 µm; $P = 0.003$). Mean visual acuity improved from 0.323 ± 0.209 to 0.266 ± 0.276 logMAR (20/41 to 20/36 Snellen) (mean change in LogMAR (−0.057 ± 0.098)), although this change was not statistically significant ($P = 0.57$).

Conclusion: Empagliflozin initiation was associated with a significant short-term reduction in CST in patients with type 2 diabetes and DME. These preliminary findings suggest a potential therapeutic benefit in DME that warrants investigation in prospective, controlled trials.

Keywords: Diabetes, Sodium-glucose 2 cotransport inhibitors, Diabetic macular edema, Neovascularization

Received: April 11, 2026, **Revised:** June 1, 2026, **Accepted:** June 28, 2026, **ePublished:** July 20, 2026

Introduction

The diabetes epidemic and its complications have emerged as a global health concern. The International Diabetes Federation (IDF) atlas reports that 537 million people globally are affected by diabetes.[1] Diabetic retinopathy (DR) is a common microvascular complication of diabetes and the principal etiology of vision impairment among the population in numerous nations.[2] One in three individuals with type 2 diabetes mellitus (T2DM) has diabetic retinopathy (DR), which is the leading and continuously increasing cause of blindness globally.[3] Proliferative diabetic retinopathy (PDR) and diabetic macular oedema (DME) are the advanced stages of DR. In diabetic macular edema, fluid collects in the central retina due to capillary leakage, resulting in visual loss. Rather, PDR entails neovascularization and fibrous retinal lesions.[4] The worldwide occurrence of DME is estimated at 6.8%. Approximately 27 million people globally are impacted by DME. DME exhibits a complex pathophysiology, typically associated with increased vascular permeability, angiogenesis, and inflammation. [4,5] Given that hyperglycaemia is acknowledged as the catalyst for disease progression in DR and other ocular problems, efficient management of glycaemia and blood pressure is crucial for minimizing disease advancement. [6,7] Current therapy approaches for DME mostly involve intravitreal injections of anti-vascular endothelial growth factor (anti-VEGF) medicines or corticosteroids, which directly address inflammation and pathological retinal neovascularization. Nonetheless, these treatments are intrusive, expensive, and not universally efficacious, necessitating the investigation of systemic medications that may augment or diminish the need for ocular procedures. [8,9]

Sodium–glucose cotransporter 2 inhibitor (SGLT2i) is a novel antidiabetic agent that provides a pleiotropic improvement in glycemic control in the management of T2DM.[10] In addition to the general effects of SGLT2 inhibitors, it was demonstrated that SGLT2 gene expression was elevated in the retina and surrounding retinal blood vessels of db/db mice, but dapagliflozin reduced glucose

uptake in human retinal endothelial cells.[11] Additionally, research demonstrated that early administration of empagliflozin in type 1 diabetes reduced the incidence of vascular leakage and mitigated the expression of the pathogenic vascular endothelial growth factor (VEGF) in the retina, which is significantly increased, altering the retinal genetic profile in the Akimba mouse model.[12] Despite the study not being performed in T2DM, the analogous hyperglycemic conditions in the retina suggest that SGLT2 inhibitors may also provide ocular advantages in T2DM. Moreover, another trial that assessed the alteration in retinal thickness, treatment with dapagliflozin resulted in a greater increase in central retinal thickness than glibenclamide treatment.[13] Also, Dapagliflozin was observed to enhance retinal capillary flow, promote arteriole remodeling, and reduce arterial stiffness indicators, demonstrating efficacy in decelerating the course of diabetic retinopathy.[14] However, the post hoc analysis of the Empagliflozin Cardiovascular Outcome Event Trial in Type 2 Diabetes Mellitus (EMPA-REG) did not reveal any significant difference in the incidence of retinopathy between empagliflozin and placebo treatment. .[15] Considering the discrepancies among the existing evidence, it appears that the association between the use of empagliflozin and macular thickness in patients with T2DM remains still unclear.

Consequently, the objective of this study was to ascertain whether the utilization of empagliflozin in routine clinical care is associated with differences in central macular thickness at an ~8-week follow-up, through retrospective analysis of medical records. Understanding these effects could have important implications for clinical practice, introducing a new aspect to the therapy of diabetic eye disease that transcends glucose regulation.

Methods and Materials

Study Design and Participant Selection

This was a retrospective observational before-and-after study. We examined the medical records of individuals diagnosed with T2DM and verified diabetic macular edema (DME) who commenced treatment with empagliflozin in standard clinical practice. Patients were identified at the ophthalmology clinic of Nikokari Hospital in Tabriz from 2021 to 2022. The baseline visit (Visit 0) was designated as the clinic appointment immediately preceding the commencement of empagliflozin, during which spectral-domain optical coherence tomography (SD-OCT) validated the presence of DME. The subsequent appointment (Visit 1) was the next planned ophthalmological assessment, occurring about 8 weeks later.

Inclusion criteria were: (1) Age 45-75 years; (2) Diagnosis of T2DM for ≥ 10 years; (3) Most recent HbA1c < 8%; (4) New initiation of empagliflozin therapy; and (5) Availability of SD-OCT data at both baseline and ~8-week follow-up.[16] The exclusion criteria encompassed the use of medications inducing retinal edema within the preceding year, a history of intravitreal Anti-VEGF injections within the past year, prior retinal laser treatments within the past year, a history of retinal disorders such as Retinal Vein Occlusion (RVO) papillopathy, Central Serous Chorioretinopathy (CSCR), Epiretinal Membrane (ERM), Age-related Macular Degeneration (AMD), Retinitis Pigmentosa (RP), Choroidal Neovascularization (CNV), retinitis, and choroiditis, previous eye surgeries within the past year, HbA1C levels exceeding 8, glaucoma, uveitis, dense cataracts, corneal diseases obstructing OCT, and patients with end-stage renal disease (ESRD).

Ophthalmologic Assessments and Outcome Measures

We conducted a pre- and post-intervention analysis. The primary endpoint was the alteration in central subfield thickness (CST), quantified in micrometers (μm) through spectral-domain OCT. The secondary outcome was the change in best-corrected visual acuity (BCVA), evaluated by a Snellen chart and transformed to LogMAR for analytical purposes. At our tertiary center, it is standard protocol to conduct SD-OCT at each scheduled appointment for patients with DME, hence providing the requisite longitudinal data for this study. All data (CST, BCVA, intraocular pressure) were obtained from the clinical records at the two designated timepoints: baseline (before empagliflozin administration) and follow-up (approximately 8 weeks after initiation). CST, characterized as the mean retinal thickness within the central 1-mm diameter of the ETDRS grid, was quantitatively evaluated by applying spectral-domain optical coherence tomography (SD-OCT).

The present study obtained approval from the Ethics Committee of Tabriz University of Medical Sciences (IR.TBZMED.REC.1400.1165). Clinical trial number: not applicable.

Statistical Analysis

Data were analyzed using SPSS version 22 and R (lme4 package). Continuous variables are presented as mean ± standard deviation, and categorical variables as frequencies (%). A linear mixed-effects model with patient as a random intercept was used to compare macular thickness and visual acuity before and after treatment, accounting for the correlation between the two eyes of the same patient. Results are presented as means ± standard deviations. A p-value of less than 0.05 was considered statistically significant.

Results

Table 1 displays the demographic information of the patients. This study evaluated 47 eyes from 30 patients. The study included 30 patients, consisting of 17 men (56.7%) and 13 women (43.3%). The average age of participants was 66.9 ± 8.4 years. In terms of the affected eye, 26 patients (55.3%) had involvement of the right eye, whereas 21 patients (44.7%) had involvement of the left eye. The average HbA1c was 7.29 ± 1.12%, with 26.6% of patients exhibiting values below 6.5% and 73.4% beyond 6.5%. Hypertension was observed in 46.6% of cases, whereas

Table 1. Baseline characteristics of patients with type 2 diabetes treated with empagliflozin

Variable	Frequency (%)
Gender	
Male	17 (56.7)
Female	13 (43.3)
Age (years)	
Mean ± SD	66.9 ± 8.4
<50 years	8 (26.7)
50–60 years	7 (23.3)
60–70 years	6 (20)
70–80 years	7 (23.3)
>80 years	2 (6.7)
Affected eye	
Right	26 (55.3)
Left	21 (44.7)
Hemoglobin A1c (%)	
Mean ± SD	7.29 ± 1.12
<6.5	8 (26.7)
6.5-7.2	12 (40)
7.2-7.7	7 (23.3)
≥7.7	3 (10)
Hypertension	14 (46.7)
Dyslipidemia	6 (20)
Creatinine (mean ± SD)	0.79 ± 0.23
Macular edema pattern	
Diffuse retinal thickening	18 (60)
CME	12 (40)
SRD	0
	Mean ± SD
Baseline central subfield thickness, CST (µm)	400.4 ± 84.8
Baseline visual acuity (logMAR)	0.323 ± 0.209
Baseline intraocular pressure (mmHg)	13.7 ± 2.14

dyslipidemia was noted in 20%. The predominant pattern of macular edema was the diffuse retinal thickening (60%), succeeded by cystoid macular edema (CME) (40%) and serous retinal detachment (SRD) (0%). The mean CST was 400.4 ± 84.8 µm, and the mean intraocular pressure was 13.7 ± 2.4 mmHg.

Figure 1 shows the clinical progression of participants. The mean CST in the study patients before starting empagliflozin was 400.4 ± 8.84 µm. The patients were re-examined at a mean time of 8 weeks after starting the drug, and the mean CST after using empagliflozin was 344.3 ± 7.76 µm. The rate of reduction in CST in the patients at follow-up was significant (rate of reduction = 56.06 µm; *P-value* = 0.003).

The patients' mean initial visual acuity was 0.323 ± 0.209 logMAR (20/41 Snellen), which decreased to 0.266 ± 0.276 logMAR (20/e36 Snellen) after receiving empagliflozin. The mean visual acuity of the patients did not exhibit any significant improvement (*P-value* = 0.61).

Figure 2 illustrates the scatter plot of CST prior to and

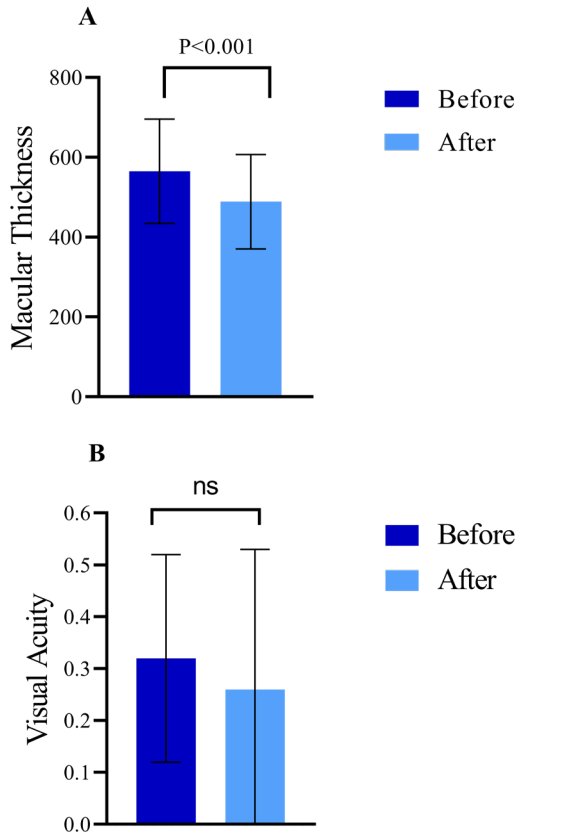

Figure 1. The clinical progression of the study participants. * The clinical progression of the study participants. Paired t-test. *P*-value for differences between central subfield thickness (CST) and visual acuity before and after administration of empagliflozin at an ~8-week follow-up

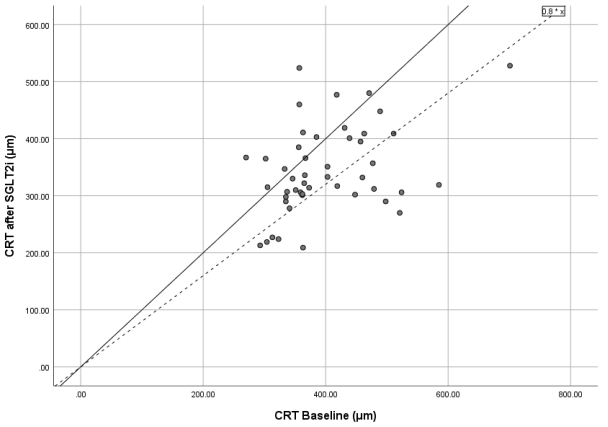

Figure 2. Scatter plot illustrating central subfield thickness (CST) before and after administration of empagliflozin at an ~8-week follow-up

subsequent to empagliflozin administration. As per the results, it was observed that the CST decreased in 16 eyes by over 20%, while it increased in 12 eyes by over 20%.

Discussion

DR is the principal ocular complication of diabetes mellitus, impacting approximately 30 to 40% of individuals. adults with diabetes.[17] This cross-sectional study aimed to compare changes in macular thickness in T2DM patients before and after they received empagliflozin. Based on the findings of the current study, the use of empagliflozin was found to significantly

reduce macular thickness in patients. Additionally, an improvement rate exceeding 20% was noted in 16 eyes, representing 34% of the cases.

The relationship between SGLT2 inhibitors (SGLT2i) and reduced central retinal thickness, along with enhanced diabetic macular edema, has been documented in multiple case reports. Our results align with the results of Mieno et al., which reported a significant enhancement in the median visual acuity (VA) measured in logMAR after initiating SGLT2 inhibitors, as well as a significant decrease in the median central retinal thickness (CRT). They suggest that for chronic DMO in vitrectomy eyes, SGLT2 inhibitors may be structurally effective. [18] Three patients with chronic DME were treated in the Takatsuna et al. trial, and their conditions markedly improved without the incorporation of any supplementary ophthalmological interventions. [19] Su et al. also discovered that SGLT2 inhibitors were associated with to a markedly decreased risk of diabetic macular edema in comparison to GLP-1 receptor agonists.[20] Additionally, a meta-analysis found that while canagliflozin may raise the risk of vitreous illness, empagliflozin and ertugliflozin may lower the risk of retinal disease. [21] Another meta-analysis indicated that SGLT2 inhibitors were linked to a diminished risk of diabetic retinopathy in people with diabetes for less 10 years. [22] Lin et al.'s retrospective cohort study indicated that SGLT2 inhibitors, in comparison to GLP-1 receptor agonists, were associated with a reduced incidence of sight-threatening retinopathy, while they did not affect the onset of diabetic retinopathy. [23] Nonetheless, the post hoc analysis of the EMPA-REG OUTCOME trial revealed that empagliflozin did not demonstrate a significant difference in the risk of vision-threatening retinopathy when compared to placebo.[15] Moreover, SGLT2i treatment was not associated with ocular events in comparison to the absence of SGLT2i intervention, as indicated by three meta-analyses of randomized controlled trials. [21,22,24]

Multiple putative mechanisms exist by which SGLT2 inhibitors are linked to a reduced incidence of sight-threatening retinopathy. SGLT2 inhibitors can decrease blood pressure, blood glucose, uric acid levels, and body weight by enhancing renal excretion of salt, glucose, and uric acid, therefore contributing to a reduced risk of diabetic retinopathy, visceral fat accumulation, intracellular oxidative stress, and proinflammatory cytokines.[25] Furthermore, inflammation contributes to the pathophysiology of diabetic retinopathy. Inflammatory molecules, including interleukin (IL)-1β, nuclear factor kappa-B (NF-κB), tumor necrosis factor α (TNF-α) and cyclooxygenase (COX), have been demonstrated to be involved in pericyte loss, neurodegeneration, capillary degeneration, and alterations in electroretinogram. [26] Multiple pilot investigations indicated that SGLT2 inhibitors resulted in reductions in blood leptin, hypersensitive C-reactive protein (hs-CRP), TNF-α, and IL-6, while elevating adiponectin levels in individuals with type 2 diabetes. [27] SGLT2 inhibitors may also have anti-inflammatory benefits via weight reduction, decreased blood glucose and plasma insulin levels, and an elevation in ketone bodies. [27] Also, SGLT2 inhibitors can diminish pericyte edema, enhance microcirculation regulation, and restore glucose absorption and type IV collagen synthesis in retinal pericytes. Moreover, SGLT2 inhibitors can restore impaired retinal neurovascular coupling (including neuronal retinal dysfunction and dysregulation of retinal blood flow) and suppress retinal glial activation in murine models of type 2 diabetes. Finally, SGLT2 inhibitors may weaken the sympathetic nervous system and provide neuroprotection in the retina. SGLT2 inhibitors are linked to microcirculation of retinal neurovascular coupling and enhanced metabolism, as well as reduced apoptosis of retinal and microvascular endothelial cells, hence diminishing the risk of sight-threatening retinopathy. [25]

This study has several important limitations, most notably its retrospective design and the absence of a control group. Therefore, the observed decrease in CST, although notable, cannot be conclusively ascribed solely to empagliflozin. Confounding factors, such as concurrent enhancements in glycemic management, blood pressure regulation, or the natural history of DME (including regression to the mean), may have influenced the observed change. Notably, we did not systematically obtain paired HbA1c data at the 8-week follow-up to establish a correlation between anatomical changes and short-term glycemic control. Secondly, the sample size was limited. Third, the follow-up period was comparatively brief (around 8 weeks); extended studies are necessary to evaluate the persistence of this effect. Ultimately, the research was carried out at a single tertiary institution, which could restrict the broader applicability of the findings.

Conclusion

This retrospective clinical investigation demonstrated that the initiation of empagliflozin significantly reduced CST in patients with T2DM and DME during an 8-week duration. While uncontrolled for potential confounders, this real-world finding provides preliminary clinical support for the biologically plausible role of SGLT2 inhibitors in DME management. The results underscore the necessity for prospective, randomized controlled trials to assess the potential of SGLT2 inhibitors as a valuable systemic complement to local ocular treatments for DME.

Acknowledgements

The authors would like to thank the Endocrine Research Center and Nutrition Research Center, Tabriz University of Medical Sciences, for supporting this project. We would also like to appreciate the cooperation of the Clinical Research Development Unit of Imam Reza General Hospital, Tabriz University of Medical Sciences, Tabriz, Iran, for their assistance in this research.

Authors' Contribution

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Competing Interests
The authors affirm that they have no conflicting interests.

Ethical Approval
All procedures conducted in studies involving human participants complied with the ethical standards set forth by the institutional and/or national research committee, as well as the 1964 Helsinki Declaration and its subsequent amendments or equivalent ethical standards. This study was approved by the Ethics Committee of the Tabriz University of Medical Sciences. (IR.TBZMED.REC.1400.1165). All participants provided written informed consent before participating in the study.

Funding
This study was financially supported by Tabriz University of Medical Sciences (Grant number: 68227).

Intelligence Use Disclosure
The authors used an AI language model to assist with revising and improving the phrasing of certain sentences in the manuscript. All AI-generated suggestions were critically reviewed, edited, and approved by the authors. The authors take full responsibility for the final content and conclusions presented in this work.

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