Review Article

The Link Between Glycemic Control and Complications after Cataract Surgery in Type 2 Diabetes: A Systematic Review

Saeed Alqahtani[1], Abdulaziz I. Alshehri[2], Mohammed I. Almakrami[3], Ahmed M. Al Rashah[3], Fatimah D. Alqahtani[3], Ali M. Al Murthimah[3], Mohammed H. Al Baalharith[3], Ahmed H. Al Abyah[3], Hussain A. Al Rashah[3], Turki S. Al Sulayyi[4], Ibrahim K. Al Sidran[3], Abdulrahman M. Al Yassain[3]

[1]Associate Professor (Ophthalmology), Najran University College of Medicine: Najran, Najran, KSA.
[2]Resident, Asir cluster, KSA.
[3]Medical intern, College of Medicine, Najran University, Najran, KSA.
[4]General Practitioner, Najran University, Najran City, KSA.

ABSTRACT

Cataract surgery is a common procedure in patients with type 2 diabetes mellitus (T2DM), who face a higher risk of postoperative complications. Glycated hemoglobin (HbA1c) is a standard measure of glycemic control, but its role as a predictor for surgical outcomes in ophthalmology remains unclear, leading to debate over preoperative HbA1c thresholds. This systematic review aimed to synthesize the evidence on the association between preoperative HbA1c levels and the risk of complications following cataract surgery in patients with T2DM. A systematic search was conducted following PRISMA guidelines across PubMed/MEDLINE, Web of Science, SCOPUS, and Embase. Observational studies investigating the link between preoperative HbA1c and postoperative complications in T2DM patients were included. Two independent reviewers performed study selection, data extraction, and risk of bias assessment using the Newcastle-Ottawa Scale. Twelve studies were included. The evidence revealed a complication-specific relationship. Elevated preoperative HbA1c (e.g., >7%) was a significant independent risk factor for cystoid macular edema (relative risk 2.01) and diabetic macular edema, supported by a clear pathophysiological link. Conversely, large-scale studies found no significant association between HbA1c and the risk of acute endophthalmitis. Several studies indicated that overall diabetes severity (e.g., renal function, complication burden) was a more robust predictor of surgical risk than glycemic control. The relationship between preoperative HbA1c and post-cataract surgery complications is not uniform. A comprehensive preoperative assessment that includes retinal status, renal function, and overall diabetes complication severity is recommended for individualized risk stratification and optimized surgical outcomes.

Keyword: Type 2 diabetes, cataract surgery, glycemic control, HbA1c, postoperative complications, macular edema.

Introduction

Cataract, the opacification of the eye's natural lens, remains the leading cause of blindness globally, with its prevalence and incidence dramatically increased in the population with diabetes mellitus [1]. The metabolic derangements characteristic of diabetes, particularly chronic hyperglycemia, accelerate the formation of cataracts through pathways such as the polyol pathway, oxidative stress, and the accumulation of advanced glycation end-products (AGEs) within the lens [2, 3].

Access this article online	
Quick Response Code:	Website: www.smh-j.com
	DOI: 10.54293/smhj.v6i1.182

Address for correspondence: Saeed Alqahtani, Associate Professor (Ophthalmology), Najran University College of Medicine: Najran, Najran, KSA.
E-mail: Alhafezsaeed@gmail.com

Received: 5 Oct 2025 **Accepted:** 6 Nov 2025

Please cite this article as: alqahtani S, Alshehri AI, Almakrami MIA, Al Rashah AMY, Alqahtani FD, Al Murthimah AMA, Al Baalharith MHS, Al Abyah AHA, Al Rashah HAS, Al Sulayyi TSM, Al Sidran IK, Al Yassain AMM. The Link Between Glycemic Control and Complications after Cataract Surgery in Type 2 Diabetes: A Systematic Review: Glycemic Control and Complications after Cataract Surgery. SMHJ [Internet]. 2025;6(1):131-141.

SMHJ
Saudi Medical Horizons Journal

Consequently, individuals with diabetes undergo cataract surgery at a younger age and with a significantly higher frequency than the non-diabetic population, with estimates suggesting that nearly 20% of all cataract procedures are performed on diabetic patients [4]. Cataract surgery, predominantly phacoemulsification with intraocular lens implantation, is one of the most successful and frequently performed surgical procedures worldwide. While outcomes are generally excellent, the presence of diabetes mellitus introduces a layer of complexity and elevates the risk of both intraoperative and postoperative complications [5]. Diabetic eyes are often considered more fragile, with a higher propensity for intraoperative challenges such as poor pupillary dilation, compromised corneal endothelium, and an increased incidence of posterior capsule rupture [6]. Postoperatively, this patient population is at a heightened risk for sight-threatening conditions, including pseudophakic cystoid macular edema, progression of diabetic retinopathy, and the development or worsening of diabetic macular edema (DME) [7]. Furthermore, the systemic nature of diabetes, often accompanied by comorbidities, may also influence the risk of severe inflammatory responses and even infectious complications like endophthalmitis [8]. The cornerstone of long-term diabetes management is glycemic control, universally measured by glycated hemoglobin (HbA1c), which reflects the average blood glucose levels over the preceding two to three months. In the context of general surgery, a wealth of evidence has established that poor preoperative glycemic control, indicated by an elevated HbA1c, is a strong predictor of adverse postoperative outcomes, including surgical site infections, delayed wound healing, and increased morbidity and mortality [9]. This has led to widespread protocols recommending the optimization of HbA1c before elective surgeries. However, the direct translation of this principle to ophthalmologic surgery, particularly cataract surgery, is a subject of ongoing debate and insufficient evidence. The eye, with its unique immune-privileged status and blood-ocular barriers, may not respond to metabolic stress in an identical manner to other organ systems. The question of whether a specific HbA1c threshold should be mandated before clearing a diabetic patient for cataract surgery is therefore clinically paramount. Withholding surgery due to an arbitrary HbA1c value can unnecessarily prolong visual impairment, diminish quality of life, and increase the risk of falls and associated fractures. Conversely, proceeding with surgery in a patient with very poor glycemic control may potentially expose them to an avoidable risk of sight-threatening complications. Current literature on this subject is fragmented, with studies often reporting conflicting results. Some studies suggest a strong correlation between high HbA1c and complications like macular edema [4], while other large-scale analyses find no significant link to other complications such as endophthalmitis [8]. This inconsistency underscores the need for a comprehensive and systematic appraisal of the available evidence to provide clarity and guide clinical decision-making. The objective of this systematic review is to critically evaluate and synthesize the existing evidence on the association between preoperative glycemic control, as measured by HbA1c levels, and the risk of developing complications after cataract surgery in patients with type 2 diabetes mellitus.

Methods

This systematic review was conducted in strict accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines to ensure a transparent and comprehensive reporting of the methodology and findings [10]. A systematic search strategy was designed and executed across multiple electronic bibliographic databases, including PubMed/MEDLINE, Web of Science Core Collection, SCOPUS, and Embase, to identify all relevant published literature. The search utilized a combination of controlled vocabulary terms (e.g., MeSH) and free-text keywords related to the core concepts of "type 2 diabetes mellitus," "glycated hemoglobin A1c," "cataract extraction," and "postoperative complications." No date restrictions were applied to the search to maximize the retrieval of all potentially eligible studies, and the search was limited to articles published in the English language. To mitigate selection bias and enhance the reliability of the study selection process, two independent reviewers performed all stages of the review, including the screening of titles and abstracts, the full-text assessment of potentially eligible articles, data extraction, and the methodological quality assessment of the included studies. Any discrepancies between the reviewers were resolved through discussion or, if necessary, by consultation with a third senior reviewer. Eligibility Criteria: The inclusion criteria for this review were developed using the PICOS framework. Studies were included if they involved adult human participants (≥18 years) with a diagnosis of type 2 diabetes mellitus who underwent cataract surgery. The intervention/exposure of interest was the level of preoperative glycemic control, specifically measured by glycated hemoglobin (HbA1c). The comparator was individuals with different levels of HbA1c (e.g., below and above a specific threshold) or non-diabetic controls. The outcomes of interest

included any postoperative complication, such as cystoid macular edema, endophthalmitis, corneal edema, visual acuity outcomes, or retinal thickness changes. We included observational study designs, including cohort studies (both prospective and retrospective), case-control studies, and cross-sectional analyses, that provided quantitative data on the association between HbA1c and one or more postoperative outcomes. Studies were excluded if they did not specifically report on patients with type 2 diabetes, if the measurement of preoperative HbA1c was not a central component of the analysis, or if they did not report a relevant postoperative complication outcome. Review articles, meta-analyses, editorials, commentaries, case reports, case series with fewer than 10 patients, conference abstracts, and animal studies were also excluded. Furthermore, studies not published in the English language were not considered for inclusion. Data Extraction: The study selection process was managed using the systematic review software Rayyan (QCRI) to allow for blinded screening and to improve efficiency [11]. Following the removal of duplicates, the titles and abstracts of all retrieved records were screened by two independent reviewers against the predefined eligibility criteria. The full text of any article deemed potentially relevant by either reviewer was then obtained and subjected to a detailed eligibility assessment. The same two reviewers independently extracted data from the included studies using a piloted, standardized data extraction form developed in Microsoft Excel. The extracted data included: (1) study characteristics (first author, publication year, country of origin, study design); (2) participant characteristics (sample size, mean age, diabetes duration, baseline HbA1c level, presence of diabetic retinopathy); (3) details of the intervention (cataract surgery technique); (4) outcome data (type of complication, rate of occurrence, measures of association such as odds ratios, risk ratios, or mean differences with corresponding confidence intervals and p-values); and (5) key conclusions of the study. Any disagreements during data extraction were resolved through consensus. Data Synthesis Strategy: Due to the anticipated heterogeneity in the definitions of glycemic control, the specific complications assessed, the measurement of outcomes, and the study designs, a meta-analysis was deemed inappropriate. Therefore, the findings of this systematic review are presented as a narrative synthesis. The results are organized by type of postoperative complication. For each complication category, the evidence from the included studies is summarized descriptively, noting the direction, strength, and consistency of the association with preoperative HbA1c levels. Data are presented in detailed summary tables to provide a clear and concise overview of the study characteristics and findings. Risk of Bias Assessment: The risk of bias in the included observational studies was assessed independently by two reviewers using the Newcastle-Ottawa Scale (NOS) for cohort and case-control studies [12]. The NOS judges studies based on three domains: the selection of the study groups, the comparability of the groups, and the ascertainment of either the exposure or outcome of interest. For any included cross-sectional studies, the risk of bias was assessed using the appropriate Joanna Briggs Institute (JBI) critical appraisal checklist [13]. The results of the quality assessment are presented in a table and were used to inform the interpretation of the review's findings and the strength of the conclusions drawn.

Results

The PRISMA flow diagram in (Figure 1) illustrates the process of study selection for this systematic review. Initially, 713 records were identified through database searches. After removing 440 duplicate records, 273 unique records were screened based on title and abstract, resulting in the exclusion of 135 records. The full texts of the remaining 138 records were sought for retrieval; however, 66 could not be retrieved, leaving 72 records for full-text eligibility assessment. Of these, 60 were excluded due to wrong outcomes (n=49), wrong population (n=2), or being conference abstracts (n=9), culminating in a final inclusion of 12 studies for the systematic review. As detailed in (Table 1), the research spans multiple countries and employs a variety of designs, including large-scale retrospective cohort studies analyzing hundreds of thousands of patients [15, 17, 18] and smaller, focused prospective longitudinal studies tracking specific physiological changes [16, 20, 22]. The patient populations are predominantly comprised of individuals with type 2 diabetes mellitus (T2DM), though several larger datasets also include those with type 1 diabetes [14, 15, 19]. A critical challenge in synthesizing this evidence is the frequent omission of key demographic and clinical parameters, such as mean age and diabetes duration, which are not consistently reported across all studies (denoted as 'NM' - Not Mentioned). This heterogeneity in design, sample size, and reporting standards is a fundamental characteristic of the current literature that must be accounted for in any systematic review. The outcomes and complications assessed, as summarized in (Table 2), are equally varied, ranging from common and sight-threatening issues like cystoid

macular edema (CME) [14] and endophthalmitis [15, 18] to more subtle, device-measured changes in ocular microstructure and vasculature [20, 24]. The role of glycated hemoglobin (HbA1c) as a predictive risk factor yields conflicting results, forming the core of the research question. Several studies provide evidence supporting a significant association. For instance, Rachmilevich et al. [14] identified HbA1c >7% as a strong independent risk factor for CME (RR 2.01), while Ming et al. [25] found higher HbA1c levels to be a significant risk factor for postoperative macular edema. Conversely, other large, robust studies found no statistically significant link between preoperative HbA1c and the risk of acute endophthalmitis [15] or general postoperative ophthalmic complications [19]. Beyond a simple positive or negative association, the data suggest that the relationship is complex and may be complication-specific. Studies like that of Hou et al. [18] indicate that while HbA1c itself may not be the direct predictor, the overall severity of diabetes complications (as measured by the DCSI score) carries a dose-response relationship with endophthalmitis risk. Furthermore, research by Boroojeny et al. [19] proposes that other systemic factors, such as renal function (eGFR), may be more potent predictors of surgical risk than glycemic control alone. Physiological studies add another layer, demonstrating that poor glycemic control correlates with altered ocular fluid dynamics, such as an increased aqueous humor-to-blood glucose ratio [16], which may create a more susceptible environment for complications, even if the direct clinical link is not always established. This body of evidence is further complemented by studies examining other outcome measures. For example, Opala et al. [22] demonstrated that even well-controlled T2DM patients start with a lower corneal endothelial cell density and experience more pronounced damage and slower recovery after phacoemulsification.

Discussion

This systematic review aimed to synthesize the current evidence on the critical question of whether glycemic control, as measured by preoperative HbA1c, is a definitive predictor of complications following cataract surgery in patients with type 2 diabetes mellitus (T2DM). Our analysis of twelve studies reveals a complex and nuanced landscape, indicating that the relationship is not monolithic but is instead highly dependent on the specific complication in question. The findings suggest that while poor glycemic control is a significant contributor to the pathophysiological environment that predisposes patients to complications, it is often not the sole or most direct predictor of surgical outcomes. The most compelling evidence for a direct link between elevated HbA1c and a specific postoperative complication was found for cystoid macular edema (CME) and diabetic macular edema (DME). Our review found that patients with an HbA1c level greater than 7% had a significantly increased risk of developing pseudophakic CME, with a relative risk of 2.01 [14]. This is strongly supported by the work of Ming et al., who identified higher HbA1c as a key risk factor for macular edema post-surgery [25]. This association is biologically plausible. Chronic hyperglycemia induces a state of oxidative stress, inflammation, and vascular endothelial dysfunction, upregulating vascular endothelial growth factor (VEGF) and other inflammatory cytokines [26, 27]. The surgical trauma of cataract surgery further disrupts the blood-aqueous barrier, exacerbating this inflammatory cascade in an eye already primed for vascular leakage. The findings from Gomel et al., which demonstrated a strong correlation ($R=0.62$, $p<0.01$) between HbA1c and the ratio of aqueous humor to blood glucose, provide a direct physiological link, showing that poor glycemic control leads to higher glucose permeability into the eye, thereby fueling this pathological process [16]. In stark contrast to the findings on macular edema, the evidence for a link between HbA1c and postoperative endophthalmitis is weak and largely negative. The large, well-controlled study by Armbrust et al. (2025), involving over 190,000 patients in the VA system, found no significant association between preoperative HbA1c levels and the risk of acute endophthalmitis, a conclusion that held in both univariable and multivariable analyses ($p=0.08$) [15]. This finding is corroborated by the large nationwide cohort study from Taiwan by Hou et al. (2023), which, while establishing diabetes itself as a risk factor (Adj. OR=1.09), pointed to the overall severity of diabetic complications (DCSI score) rather than glycemic control alone as the more influential factor [18]. This suggests that the increased endophthalmitis risk in diabetic patients may be mediated more by systemic immunosuppression, microangiopathy affecting ocular surface healing, and neurotrophic deficits rather than acute perioperative hyperglycemia [28]. This is a crucial distinction for surgeons, as it implies that deferring surgery based solely on a high HbA1c may not mitigate the risk of this devastating infection and that a broader assessment of the patient's diabetic health is more informative. When considering other

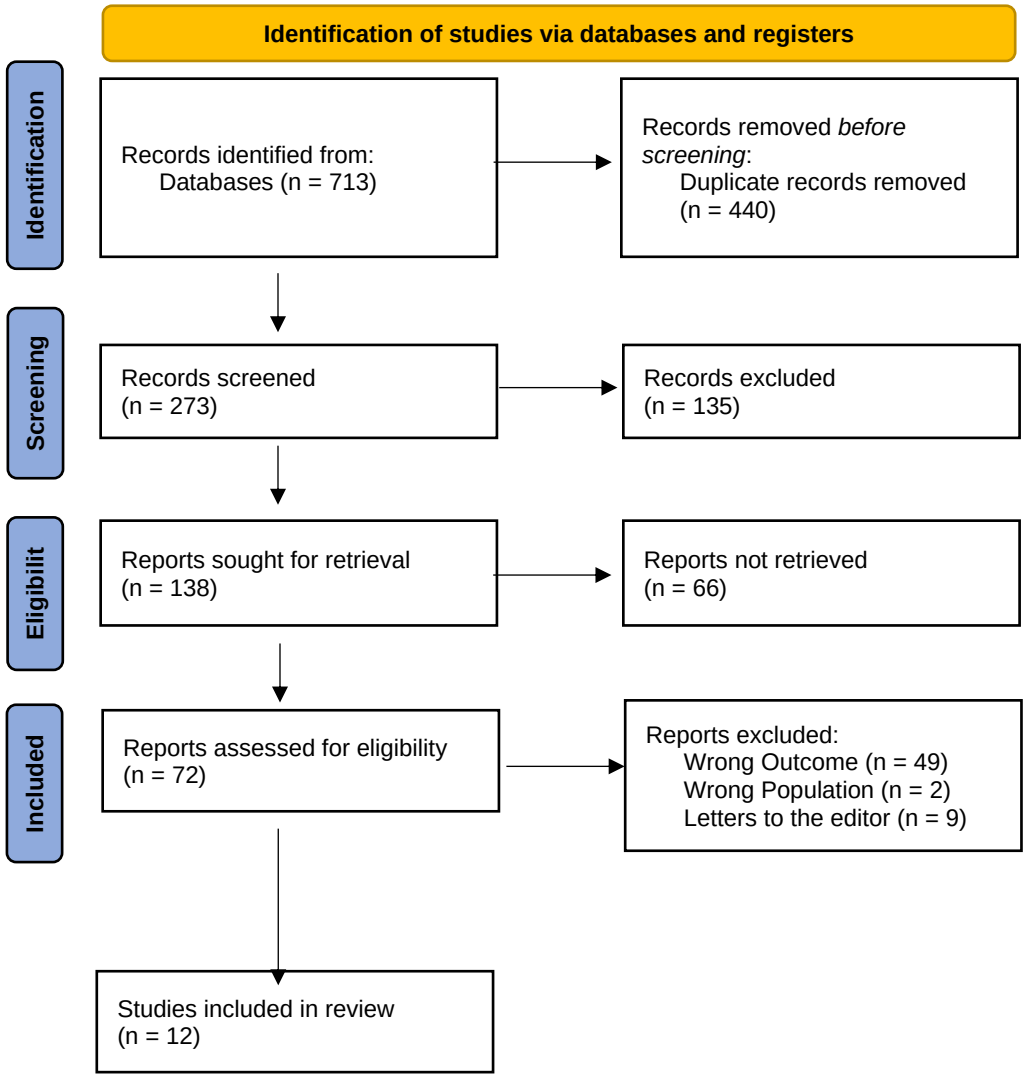

Figure 1: PRISMA 2020 Flow Diagram of the Study Selection Process.

Table 1: Study and Patient Characteristics.

Study (Author, Year) [Ref]	Country	Study Design	Sample Size (Eyes/Patients)	Patient Population	Mean Age (Years)	Diabetes Duration	Pre-op HbA1c (%)	Key Pre-op Ocular Comorbidity
Rachmilevich A, 2023 [14]	Israel	Retrospective Cohort	1285 patients	Type 1 & 2 DM	NM	NM	NM (Threshold: >7%)	Diabetic Retinopathy
Armbrust KR, 2025 [15]	USA	Retrospective Case-Control	190,393 patients	Type 1 & 2 DM	NM	NM	7.3 ± 1.5 (Control)	NM
Gomel N, 2021 [16]	Israel	Prospective	37 patients	Type 2 DM	75.2 ± 11.2	NM	NM	None (Elective surgery)
Chancellor J, 2021 [17]	Multicenter	Retrospective Cohort	179,159 eyes	DM vs Non-DM	NM	NM	NM	Diabetic Retinopathy
Hou CH, 2023 [18]	Taiwan	Retrospective Cohort	1,766,796 eyes	DM vs Non-DM	NM	NM	NM (DCSI score used)	NM
Boroojeny AB, 2022 [19]	UK	Retrospective	1525 patients	Type 1 & 2 DM	NM	NM	50 mmol/mol (~6.7%)	NM
Svjaščenkova L, 2023 [20]	Latvia	Prospective Longitudinal	34 patients	Type 2 DM	NM	NM	NM	DME (29.4% of patients)
C RH, 2025 [21]	India	Prospective	300 patients	Type 2 DM	NM	NM	NM	Diabetic Retinopathy, DME
Opala A, 2025 [22]	Poland	Prospective Longitudinal	80 eyes (DM) 80 eyes (Control)	Type 2 DM vs Non-DM	Matched	NM	NM (Well-controlled)	None (Uneventful surgery)
Koestel E, 2025 [23]	France	Case-Control	44 eyes (DM) 79 eyes (Control)	Type 2 DM vs Non-DM	NM	Subgroup: >10 vs <10 yrs	NM	None (Scheduled for surgery)
Kim J, 2024 [24]	South Korea	Prospective	83 eyes	Type 2 DM	NM	NM	NM	No DR (60 eyes), NPDR (23 eyes)
Ming L, 2025 [25]	China	Case-Control	114 eyes (Edema) 59 eyes (Control)	Type 2 DM	NM	Longer in edema group	Higher in edema group	NM

NM: Not Mentioned; DM: Diabetes Mellitus; DME: Diabetic Macular Edema; DR: Diabetic Retinopathy; NPDR: Non-Proliferative Diabetic Retinopathy; DCSI: Diabetes Complications Severity Index

Table 2: Study Outcomes and Key Findings Related to Glycemic Control**.**

Study (Author, Year) [Ref]	Primary Outcome Measured	Key Complications Assessed	Main Finding Related to HbA1c / Glycemic Control	Statistical Significance (p-value/OR/RR/CI)
Rachmilevich A, 2023 [14]	CME incidence within 1 year	Pseudophakic Cystoid Macular Edema (CME)	HbA1c >7% is an independent risk factor for CME.	RR: 2.01 (95% CI: 1.10-3.67)
Armbrust KR, 2025 [15]	Endophthalmitis within 42 days	Acute Postoperative Endophthalmitis	No significant association between preoperative HbA1c and endophthalmitis risk.	p=0.08 (Multivariable analysis)
Gomel N, 2021 [16]	AH/Blood glucose ratio	N/A (Pathophysiology study)	Strong positive correlation between HbA1c and aqueous humor/blood glucose ratio.	R=0.62, p<0.01
Chancellor J, 2021 [17]	Visual Acuity, Intraop complications	Posterior Capsule Rupture, Dropped Nucleus	Worse postoperative VA correlated with DR severity, but HbA1c itself was not measured/reported.	NM for HbA1c
Hou CH, 2023 [18]	Endophthalmitis within 3 months	Endophthalmitis	DM itself was a risk factor. A dose-response relationship was found with diabetes severity (DCSI score).	Adj. OR=1.09 (95% CI: 1.03-1.16) for DM
Boroojeny AB, 2022 [19]	Post-operative ophthalmological complications	Various (as per nat. guidelines)	HbA1c was not a significant risk factor for complications. eGFR was a better predictor.	OR: 1.00 (95% CI: 0.99-1.05), p=0.85
Svjaščenkova L, 2023 [20]	Microstructural changes on OCT-A	Macular Edema (subclinical)	Better glycemic control (lower HbA1c) reduced probability of FAZ changes post-op.	Effect estimate β = -0.20 to -0.13
C RH, 2025 [21]	Post-op visual acuity ≥6/18	Iritis, Striate Keratopathy	Poor pre-op VA and DR/DME were linked to worse outcomes. Role of HbA1c not explicitly stated.	NM for HbA1c

Opala A, 2025 [22]	Corneal Endothelial Parameters	Corneal Endothelial Damage	Well-controlled DM patients had lower pre-op ECD and were more vulnerable to surgical damage.	p=0.002 (Pre-op ECD)
Koestel E, 2025 [23]	Ocular Surface Parameters	Dry Eye Symptoms, Corneal Thickness	Diabetes duration (>10 yrs) correlated with worse dry eye symptoms post-op. HbA1c not measured.	NM for HbA1c
Kim J, 2024 [24]	OCT-A Parameters	Microvascular Changes (FAZ, VD)	Study assessed DR status, not HbA1c levels. Changes were different in No DR vs. DR groups.	NM for HbA1c
Ming L, 2025 [25]	Post-operative Macular Edema	Macular Edema	Higher HbA1c was a significant risk factor for developing post-op macular edema.	P<0.05

CME: Cystoid Macular Edema; OR: Odds Ratio; RR: Relative Risk; CI: Confidence Interval; AH: Aqueous Humor; VA: Visual Acuity; DR: Diabetic Retinopathy; eGFR: estimated Glomerular Filtration Rate; OCT-A: Optical Coherence Tomography Angiography; FAZ: Foveal Avascular Zone; VD: Vessel Density; ECD: Endothelial Cell Density

Table 3: Summary of Risk of Bias in Included Studies.

Study (Author, Year)	Key Domains with Elevated Bias	Overall Risk
Armbrust KR, 2025 [15]	—	● Low
Opala A, 2025 [22]	—	● Low
Hou CH, 2023 [18]	—	● Low
Rachmilevich A, 2023 [14]	D1 (Moderate)	●● Moderate
Chancellor J, 2021 [17]	D1 (Moderate)	●● Moderate
Boroojeny AB, 2022 [19]	D1 (Moderate), D5 (Moderate)	●● Moderate
Gomel N, 2021 [16]	D1 (High)	●●● High
Svjaščenkova L, 2023 [20]	D1 (High)	●●● High
C RH, 2025 [21]	D1 (High)	●●● High
Koestel E, 2025 [23]	D1 (High)	●●● High
Kim J, 2024 [24]	D1 (High)	●●● High
Ming L, 2025 [25]	D1 (High)	●●● High

Domains: D1 = Bias due to Confounding; D5 = Bias due to Missing Data. All other domains (D2, D3, D4, D6, D7) were judged as low risk for all studies.

complication domains, the picture becomes more mixed. For visual acuity outcomes, the large multicenter study by Chancellor et al. (2021) and others have consistently shown that the presence and severity of diabetic retinopathy are the primary drivers of worse visual results, not HbA1c itself [17, 29]. This underscores the fact that pre-existing microvascular damage is a more powerful determinant of visual potential than a single measure of glycemic control. Similarly, in the context of corneal complications, the study by Opala et al. (2025) revealed that even patients with well-controlled T2DM started with a statistically significant lower preoperative endothelial cell density (2480.76 vs. 2629.64 cells/mm², p=0.002) and showed a trend toward greater endothelial cell loss and slower recovery post-phacoemulsification compared to non-diabetic controls [22]. This indicates that the diabetic state itself, perhaps through advanced glycation end-products altering corneal morphology and regenerative capacity, poses a risk that is not fully captured by the HbA1c value [30]. This is further evidenced by studies showing more pronounced ocular surface changes and dry eye symptoms post-surgery in diabetic patients, particularly those with longer disease duration [23]. The aggregate of this evidence leads to a paradigm shift in preoperative assessment. The study by Boroojeny et al. (2022) directly challenged the focus on HbA1c, finding it to be a non-significant predictor of postoperative complications (OR 1.00, p=0.85), while identifying renal function (eGFR) as a more significant risk factor [19]. This suggests that end-organ damage, reflected by eGFR, may be a superior marker of overall systemic vulnerability to surgical stress than a three-month average of blood glucose. This aligns with the "dose-response" relationship shown by Hou et al. using the DCSI score [18]. Therefore, the most prudent approach is to move beyond a rigid HbA1c threshold. A comprehensive evaluation that includes a detailed retinal exam, assessment of renal function, and consideration of diabetes duration and other comorbidities provides a far more robust and individualized risk stratification model. This holistic view ensures that patients are not denied sight-restoring surgery based on an isolated number while also ensuring that those at highest risk from their overall disease burden are identified for closer monitoring and management. Limitations of the Study: This review has several limitations that must be acknowledged. First, the included studies exhibited significant heterogeneity in their design, patient populations, sample sizes, and, most importantly, the specific complications they investigated. This variability makes it difficult to perform a direct meta-analysis and necessitates a narrative synthesis. Second, many studies failed to report key confounding variables such as mean diabetes duration, the severity of diabetic retinopathy at the time of surgery, and the use of perioperative medications (e.g., NSAIDs), which could significantly influence the reported outcomes. Third, the definition of "good" versus "poor" glycemic control was not uniform across all studies, with some using a threshold of 7% and others using different values or continuous variables. Fourth, the risk of bias assessment revealed that several smaller studies, particularly those exploring physiological mechanisms, had a high risk of bias due to a lack of adjustment for critical confounders. Finally, as the field evolves, newer surgical technologies and techniques (e.g., femtosecond laser-assisted cataract surgery) may alter risk profiles, and the included studies may not fully account for these advancements.

Conclusion

The relationship between preoperative glycemic control and postoperative complications in type 2 diabetics is complex and varies by complication type. Strong evidence indicates that elevated HbA1c is an independent risk factor for macular edema, but studies do not support a significant link between HbA1c and other serious complications like endophthalmitis, suggesting broader diabetic disease factors play a role. Using a universal HbA1c cut-off for surgical eligibility is not evidence-based and should be reconsidered. Future large-scale studies are necessary to refine understanding and develop individualized risk prediction models.

Conflict of Interest

None

Funding

None

References

1. World Health Organization. Report of the 2030 targets on effective coverage of eye care. Available from: http:// World Health Organization.com [Accessed 12th Oct 2022].

2. Kyselova Z. Different experimental approaches in modelling cataractogenesis: an overview of selenite-induced nuclear cataract in rats. Interdisciplinary toxicology. 2010 Mar 29;3(1):3.

3. Pollreisz A, Schmidt-Erfurth U. Diabetic cataract-pathogenesis, epidemiology and treatment. J Ophthalmol. 2010;2010:608751. doi:10.1155/2010/608751
4. Yang J, Cai L, Sun Z, Ye H, Fan Q, Zhang K, et al. Risk factors for and diagnosis of pseudophakic cystoid macular edema after cataract surgery in diabetic patients. J Cataract Refract Surg. 2017;43(2):207-214. doi:10.1016/j.jcrs.2016.11.047
5. Soliman MK, Hardin JS, Jawed F, Uwaydat SH, Faramawi MF, Chu CJ, et al. A Database Study of Visual Outcomes and Intraoperative Complications of Postvitrectomy Cataract Surgery. Ophthalmology. 2018;125(11):1683-1691. doi:10.1016/j.ophtha.2018.05.027
6. Hong T, Mitchell P, de Loryn T, Rochtchina E, Cugati S, Wang JJ. Development and progression of diabetic retinopathy 12 months after phacoemulsification cataract surgery. Ophthalmology. 2009;116(8):1510-1514. doi:10.1016/j.ophtha.2009.03.003
7. Kim SJ, Equi R, Bressler NM. Analysis of macular edema after cataract surgery in patients with diabetes using optical coherence tomography. Ophthalmology. 2007 May 1;114(5):881-9.
8. Gower EW, Lindsley K, Tulenko SE, Nanji AA, Leyngold I, McDonnell PJ. Perioperative antibiotics for prevention of acute endophthalmitis after cataract surgery. Cochrane Database of Systematic Reviews. 2017(2):1-10.
9. Duggan EW, Carlson K, Umpierrez GE. Perioperative Hyperglycemia Management: An Update. Anesthesiology. 2017;126(3):547-560. doi:10.1097/ALN.0000000000001515
10. Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. bmj. 2021 Mar 29;372.
11. Ouzzani M, Hammady H, Fedorowicz Z, Elmagarmid A. Rayyan-a web and mobile app for systematic reviews. Syst Rev. 2016;5(1):210. doi:10.1186/s13643-016-0384-4
12. Wells GA, Shea B, O'Connell D, Peterson J, Welch V, Losos M, et al. The Newcastle-Ottawa Scale (NOS) for assessing the quality of nonrandomised studies in meta-analyses. Our research.2014;12:1-10
13. Moola SZ, Munn Z, Tufanaru C, Aromataris E, Sears K, Sfetcu R, et al. Systematic reviews of etiology and risk. JBI manual for evidence synthesis. 2020 Feb 23;1:217-69.
14. Rachmilevich A, Yanculovich N, Hazan I, Tsumi E, Liberty IF. Glycemic control and macular edema in patients undergoing cataract surgery. Prim Care Diabetes. 2023;17(1):55-59. doi:10.1016/j.pcd.2022.12.004
15. Armbrust KR, Ailani S, Berkenstock MK. Preoperative Glycemic Control and Acute Endophthalmitis after Cataract Surgery in United States Veterans with Diabetes Mellitus. Ophthalmology. 2025;132(8):903-910. doi:10.1016/j.ophtha.2025.03.023
16. Gomel N, Barequet IS, Lipsky L, Bourla N, Einan-Lifshitz A. The effect of the glycemic control on the aqueous humor glucose levels in diabetic patients undergoing elective cataract surgery. Eur J Ophthalmol. 2021;31(2):415-421. doi:10.1177/1120672120910375
17. Chancellor J, Soliman MK, Shoults CC, Faramawi MF, Al-Hindi H, Kirkland K, et al. Intraoperative Complications and Visual Outcomes of Cataract Surgery in Diabetes Mellitus: A Multicenter Database Study. Am J Ophthalmol. 2021;225:47-56. doi:10.1016/j.ajo.2020.12.027
18. Hou CH, Lee JS, Lin KK, Peng WS, Pu C, See LC. Risk and dose-response relationship of diabetes mellitus for endophthalmitis after cataract surgery in a nationwide cohort study in Taiwan. Clin Exp Ophthalmol. 2023;51(7):692-703. doi:10.1111/ceo.14288
19. Boroojeny AB, Nunney I, Dhatariya KK. The Association Between Glycaemic Control, Renal Function and Post-operative Ophthalmic Complications in People With Diabetes Undergoing Cataract Surgery-A Single-Centre Retrospective Analysis. Diabetes Ther. 2022;13(4):723-732. doi:10.1007/s13300-022-01241-z
20. Svjaščenkova L, Laganovska G, Tzivian L. Microstructural Changes in the Macula Following Cataract Surgery in Patients with Type 2 Diabetes Mellitus Detected Using Optical Coherence Tomography Angiography. Diagnostics (Basel). 2023;13(4):605. doi:10.3390/diagnostics13040605
21. Chandran R, Thulaseedharan S, Nair I. Is glycaemic control essential for cataract surgery among patients with diabetes mellitus?. Bioinformation. 2025;21(5):1011-1015. doi:10.6026/973206300211011
22. Opala A, Kołodziejski Ł, Grabska-Liberek I. Impact of Well-Controlled Type 2 Diabetes on Corneal Endothelium Following Cataract Surgery: A Prospective Longitudinal Analysis. J Clin Med. 2025;14(10):3603. doi:10.3390/jcm14103603
23. Koestel E, Dormegny L, Sauer A, Gaucher D, Bourcier T. Ocular surface changes and corneal

epithelial remodeling after cataract surgery in patients with type 2 diabetes: a case-control study. BMC Ophthalmol. 2025;25(1):355. doi:10.1186/s12886-025-04178-6

24. Kim J, Kim S, Borrelli E, Park MS, Cho BJ, Kwon S. Alterations in optical coherence tomography angiography parameters after cataract surgery in patients with diabetes. Sci Rep. 2024;14(1):23814. doi:10.1038/s41598-024-73830-w

25. Ming L, Fang X, Zhijuan X. Analysis of factors influencing macular edema in patients with diabetes cataract after surgery. Int J Ophthalmol. 2025;18(1)

26. Brownlee M. The pathobiology of diabetic complications: a unifying mechanism. Diabetes. 2005;54(6):1615-1625. doi:10.2337/diabetes.54.6.1615

27. Funatsu H, Yamashita H, Shimizu E, Kojima R, Hori S. Relationship between vascular endothelial growth factor and interleukin-6 in diabetic retinopathy. Retina. 2001;21(5):469-477. doi:10.1097/00006982-200110000-00009

28. Pershing S, Lum F, Hsu S, Kelly S, Chiang MF, Rich III WL, et al. Endophthalmitis after cataract surgery in the United States: a report from the Intelligent Research in Sight Registry, 2013–2017. Ophthalmology. 2020 Feb 1;127(2):151-8.

29. Chew EY, Benson WE, Remaley NA, Lindley AA, Burton TC, Csaky K, et al. Results after lens extraction in patients with diabetic retinopathy: early treatment diabetic retinopathy study report number 25. Archives of ophthalmology. 1999 Dec 1;117(12):1600-6.

30. Busted N, Olsen T, Schmitz O. Clinical observations on the corneal thickness and the corneal endothelium in diabetes mellitus. Br J Ophthalmol. 1981;65(10):687-690. doi:10.1136/bjo.65.10.687