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SPECIFIC FEATURES OF CATARACT SURGERY IN DIABETIC PATIENTS AND POSTOPERATIVE COMPLICATIONS

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ABSTRACT Background. Diabetes mellitus (DM) changes the crystalline lens, ocular surface, cornea, iris, retina and wound-healing response. Cataract develops earlier and more often in people with diabetes, and the global number of adults with diabetes reached 589 million in 2024 [1]. Although modern phacoemulsification usually gives meaningful visual benefit, diabetic patients have a distinct perioperative risk profile, particularly when diabetic retinopathy (DR) or diabetic macular edema (DME) is present.

Methods. A structured narrative review of PubMed/MEDLINE and PubMed Central records, publisher pages and professional guidance (ESCRS, ADA, AAO, IDF) available up to 18 October 2026. Priority was given to systematic reviews, meta-analyses, randomized trials, large cohorts and guidelines. Published estimates were reproduced with their original metrics and confidence intervals and presented in tables and native spreadsheet charts; no new pooled estimates were generated.

Results. Diabetes roughly doubles the incidence of cataract (20.4 vs 10.8 per 1,000 person-years in a UK cohort) [2]. Risk of postoperative macular edema is strongly modified by retinal status: relative risk (RR) 1.80 (95% CI 1.36–2.36) in diabetes without DR and 6.23 (5.12–7.58) with DR in an 81,984-eye study [3]. A 2025 meta-analysis (15 studies, 7,287 patients) found increased DR progression after surgery (RR 1.46, 1.28–1.66) and increased progression to sight-threatening DR (RR 1.84, 1.21–2.81), whereas paired-eye analyses were inconclusive [4]. Topical corticosteroid plus NSAID prophylaxis prevented 75.8% of pseudophakic cystoid macular edema events compared with steroid alone [5]. Delayed corneal epithelial healing, poor mydriasis, reduced endothelial reserve, posterior capsule opacification and infection are additional concerns.

Conclusions. Diabetes itself is not a contraindication to cataract surgery. Outcomes depend on preoperative retinal staging (with macular OCT), systemic and ocular-surface optimization, low-trauma surgery with intracameral antibiotic prophylaxis, risk-adapted anti-inflammatory prophylaxis and structured follow-up, especially during the first postoperative year.

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Keywords: diabetes mellitus; diabetic cataract; phacoemulsification; diabetic retinopathy; diabetic macular edema; pseudophakic cystoid macular edema; anti-VEGF; endophthalmitis; postoperative complications.

1. INTRODUCTION

Cataract and diabetes mellitus are two of the most important causes of avoidable visual disability in the world, and they frequently coexist in the same patient. Cataract remains the leading cause of blindness globally: in 2020 an estimated 15.2 million adults aged 50 years and older were blind because of cataract, and a further 78.8 million had moderate or severe visual impairment from it. Diabetic retinopathy accounted for a smaller but rising share, with about 0.86 million blind and 2.9 million with moderate-to-severe impairment [6] (Figure 2). At the same time, the diabetes epidemic continues to expand. The 11th edition of the International Diabetes Federation (IDF) Diabetes Atlas estimates that 589 million adults aged 20–79 years were living with diabetes in 2024, that about 252 million of them were undiagnosed, and that the number will rise to 853 million by 2050 [1] (Figure 1).

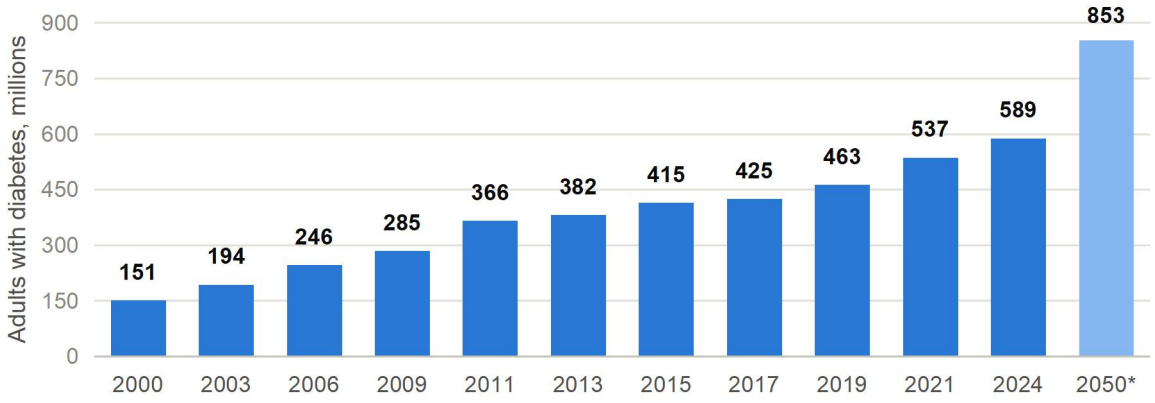


Figure 1. Estimated number of adults (20–79 years) living with diabetes worldwide in successive editions of the IDF Diabetes Atlas, with the projection for 2050 (light bar). Data: IDF Diabetes Atlas, editions 1–11 [1]. Estimates from different editions are not strictly comparable because data sources and methods were updated over time; the trend, not the year-to-year difference, is the key message. The chart is a native Excel chart: right-click → Edit Data to view the underlying worksheet.

Diabetes is associated with an earlier onset and faster progression of lens opacity, as well as a wider spectrum of ocular comorbidity. Hyperglycemia, oxidative stress, polyol-pathway activity and advanced glycation end-products contribute to lens changes; diabetes also affects corneal nerves and epithelium, endothelial function, pupillary responses, the retinal microvasculature and postoperative inflammatory signaling [7–9]. Cataract extraction can therefore restore optical transparency while exposing, unmasking or occasionally aggravating pre-existing retinal disease.

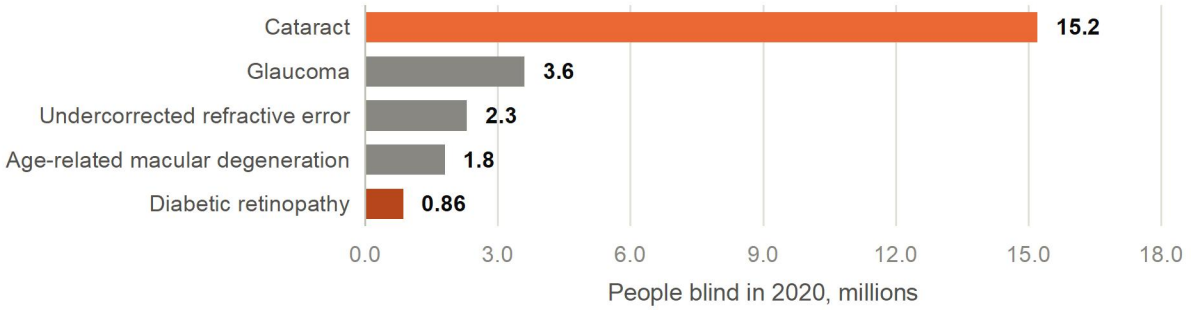


Figure 2. Global causes of blindness among adults aged ≥50 years in 2020 (millions). Cataract (orange) is the leading cause; diabetic retinopathy (dark orange) is highlighted because it frequently coexists with cataract in diabetic patients. Data: GBD 2019 Blindness and Vision Impairment Collaborators / Vision Loss Expert Group [6].

The clinical question is therefore not simply whether phacoemulsification can be performed safely in a diabetic eye. It is how to choose the timing, estimate visual potential, reduce surgical trauma, select prophylaxis and detect postoperative change early enough to preserve vision. Cataract may obscure the fundus and delay necessary retinal treatment; conversely, postoperative inflammation may increase macular edema or coincide with DR progression [9,10]. The balance is individualized rather than governed by a universal rule to delay surgery in all diabetic patients.

Evidence has evolved with small-incision phacoemulsification, optical coherence tomography (OCT), improved biometry and intraocular lens (IOL) formulas, intracameral antibiotics, anti-vascular endothelial growth factor (anti-VEGF) therapy and more targeted retinal care. However, heterogeneity in baseline DR severity, diabetes duration, definitions of postoperative macular edema, follow-up and prophylactic regimens makes direct comparison of studies difficult [3–5,11,12]. The aim of this review is to integrate current indexed literature and guidance into a practical synthesis of the specific features of cataract surgery in adults with diabetes, from epidemiology and pathogenesis to risk stratification, surgical technique, postoperative complications and surveillance, while clearly separating evidence-derived estimates from practice-oriented interpretation.

2. MATERIALS AND METHODS

Study design. This paper is a structured narrative review, not a prospective clinical study, a registered systematic review or a new meta-analysis. No patient records were accessed and no original clinical dataset was created.

Information sources and search. Literature records were searched and cross-checked through PubMed/MEDLINE and PubMed Central, with publisher pages used to verify bibliographic details and DOI links. Recommendations of the European Society of Cataract and Refractive Surgeons (ESCRS), the American Diabetes Association (ADA) and the American Academy of Ophthalmology (AAO), and epidemiological reports of the IDF and the Global Burden of Disease (GBD) Vision Loss Expert Group were also reviewed. The search was current to 18 October 2026. Search concepts included combinations of “diabetes mellitus”, “diabetic cataract”,

"phacoemulsification", "diabetic retinopathy", "diabetic macular edema", "pseudophakic cystoid macular edema", "endophthalmitis", "corneal healing", "posterior capsule opacification", "anti-VEGF" and "postoperative complications".

Eligibility and prioritization. Greater weight was given to systematic reviews and meta-analyses, randomized controlled trials (RCTs), large observational cohorts and evidence-based guidelines concerning adults undergoing cataract extraction. Older landmark studies were retained where they provided essential background. Non-peer-reviewed summaries were not used as the main evidentiary basis when a primary article or guideline was available.

Data presentation and limitations. Findings were organized into epidemiological, pathophysiological, preoperative, intraoperative and postoperative themes. Quantitative estimates were transcribed from the cited sources with their original metrics (relative risk, odds ratio, hazard ratio, incidence rate) and 95% confidence intervals (CI), and were plotted as native Microsoft Excel charts embedded in the manuscript so that the underlying data remain inspectable. Estimates were not re-pooled. Relative measures are not interchangeable and must not be read as an individual patient's absolute probability. Schematic illustrations were drawn specifically for this review and are labelled as schematic. Because this was a narrative synthesis, a PRISMA flow diagram and formal risk-of-bias assessment were not performed, and restriction to accessible English-language records may have missed relevant reports.

3. RESULTS AND DISCUSSION

3.1. Epidemiology of cataract in diabetes

Population-based and database studies consistently show that cataract is more frequent and appears earlier in people with diabetes. In a large UK primary-care cohort (Clinical Practice Research Datalink), the incidence of cataract was 20.4 per 1,000 person-years in patients with diabetes compared with 10.8 per 1,000 person-years in matched individuals without diabetes (Figure 3). The relative excess was greatest in younger patients: at 45–54 years, people with diabetes were roughly 5.8–6.5 times more likely to develop cataract, and a diabetes duration of ≥10 years carried an adjusted odds ratio of 5.14 compared with <2 years [2]. These data illustrate that a diabetic cataract service must be prepared to operate on younger, working-age patients whose retinas often have decades of diabetes ahead of them.

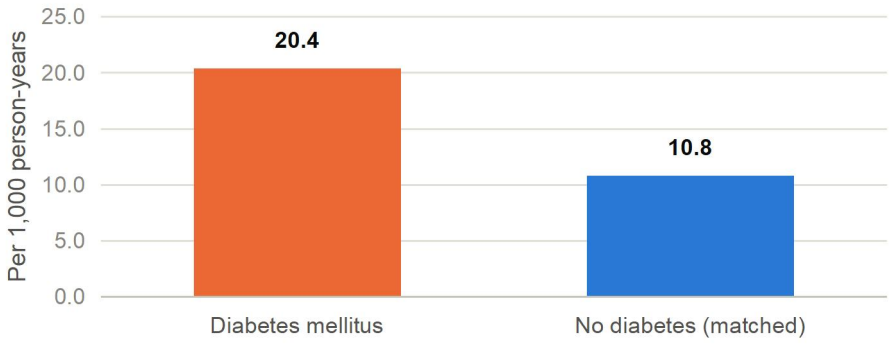


Figure 3. Incidence of cataract in patients with and without diabetes in a UK population cohort (per 1,000 person-years). Data: Becker et al., Eye 2018 [2].

The retina determines much of the outcome, so the epidemiology of DR is equally relevant. A global meta-analysis of population-based studies estimated that 22.27% (95% CI 19.73–25.03) of people with diabetes have DR, 6.17% have vision-threatening DR (VTDR) and 4.07% have clinically significant macular edema (CSME); by 2045 these proportions translate into about 160.5 million people with DR, 44.8 million with VTDR and 28.6 million with CSME [13] (Figure 4). In practice, roughly one in five diabetic patients presenting for cataract surgery can be expected to have some degree of retinopathy, and a smaller but clinically decisive group will have active macular or proliferative disease.

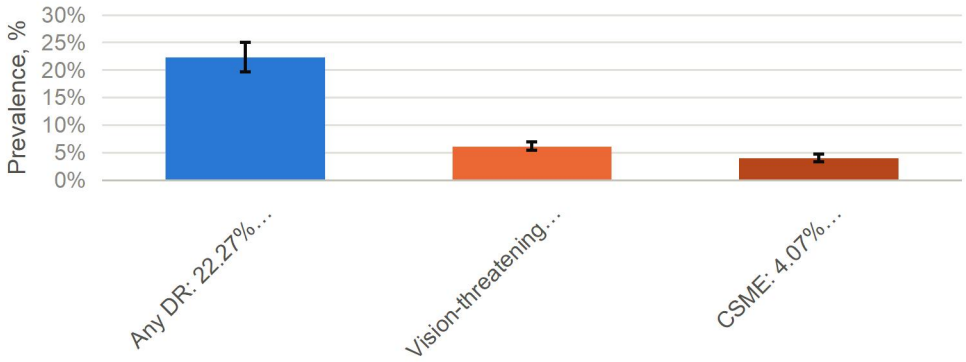


Figure 4. Global pooled prevalence of diabetic retinopathy (DR), vision-threatening DR (VTDR) and clinically significant macular edema (CSME) among people with diabetes. Bars show point estimates; error bars show 95% confidence intervals. Data: Teo et al., Ophthalmology 2021 [13].

3.2. Pathogenesis of diabetic cataract

Several interacting mechanisms link chronic hyperglycemia with lens opacification (Figure 6). In the polyol pathway, aldose reductase reduces excess intracellular glucose to sorbitol, which crosses cell membranes poorly and accumulates within lens fibres. The resulting osmotic gradient draws water into the fibres, causing swelling, liquefaction and the formation of cortical and posterior subcapsular opacities; the effect is most dramatic in the rare acute “snowflake” cataract of young patients with very poor control [7,8]. Non-enzymatic glycation of lens crystallins produces advanced glycation end-products that cross-link proteins, promote aggregation and contribute to yellow-brown pigmentation and nuclear sclerosis. Oxidative stress is increased by both mechanisms: generation of reactive oxygen species rises while glutathione and antioxidant enzyme activity fall, damaging lens epithelial cells and fibre membranes [7,8].

Osmotic changes in the lens also explain the transient refractive shifts experienced by many patients when glycemia changes rapidly, for example after starting intensive insulin therapy. This has direct practical relevance for biometry, discussed below. The same metabolic environment affects other ocular tissues, so the diabetic cataract should be understood as one manifestation of a whole-eye disease (Figure 5).

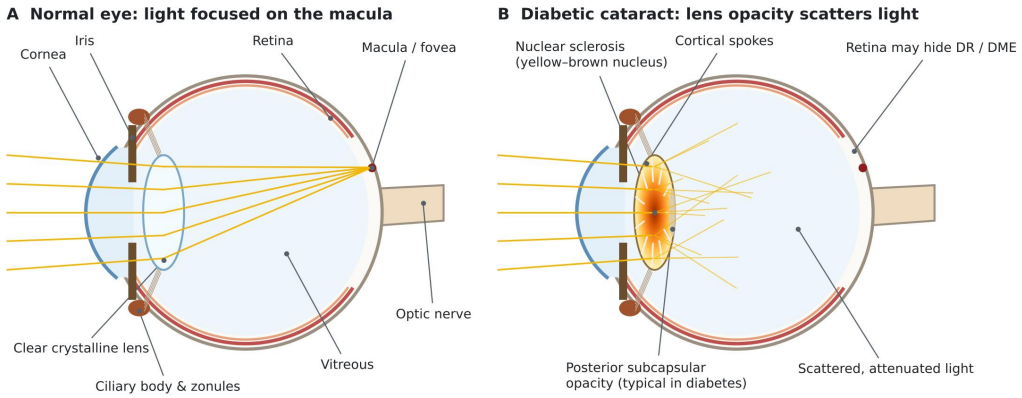


Figure 5. Schematic cross-section of the eye. (A) Normal eye: the clear crystalline lens focuses light on the macula. (B) Diabetic cataract: nuclear sclerosis, cortical spokes and a posterior subcapsular plaque scatter and attenuate light; the opaque lens also hides retinal disease (DR, DME) from examination. Original schematic illustration prepared for this review.

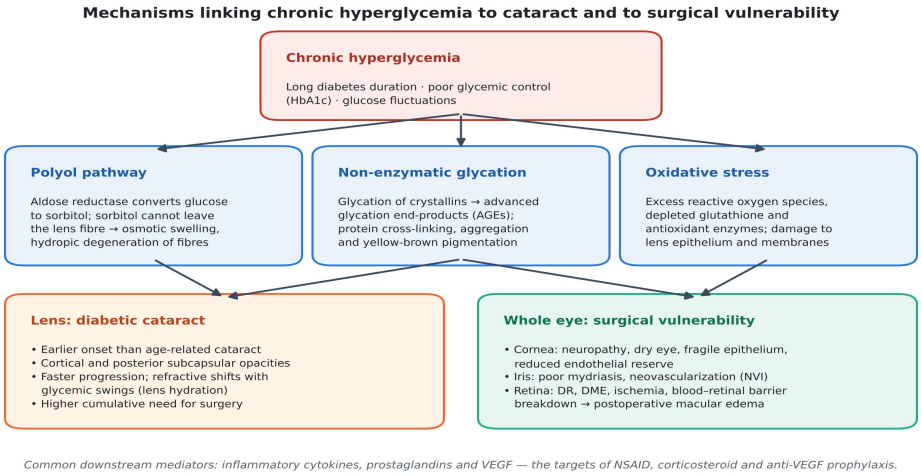


Figure 6. Mechanisms linking chronic hyperglycemia to cataract formation and to the surgical vulnerability of the diabetic eye. Synthesized from Pollreisz and Schmidt-Erfurth, Kiziltoprak et al. and Zarei-Ghanavati et al. [7–9]. Original schematic.

3.3. Why the diabetic eye has a different surgical risk profile

Cornea and ocular surface. Diabetes affects much more than the lens. Ocular-surface disease, meibomian gland dysfunction, tear-film instability, corneal hypoesthesia from small-fibre neuropathy, abnormalities of the epithelial basement membrane and delayed epithelial healing may reduce comfort and open a pathway to persistent epithelial defects or infection. Diabetic corneas may also have lower endothelial cell density, greater polymegathism and pleomorphism, and less reserve to tolerate phacoemulsification-related stress [9,14,15]. These changes justify ocular-surface optimization before biometry and surgery, epithelial protection during the operation and avoidance of unnecessary anterior-chamber manipulation.

A 2026 retrospective OCT study provides a recent quantitative illustration of the wound-healing problem. Among 141 eyes (51 diabetic, 90 non-diabetic), complete epithelial healing of the corneal incision at one week was observed in 80.39% of diabetic eyes versus 92.22% of controls; diabetic eyes also showed more incision-related structural abnormalities and greater corneal edema [16] (Figure 7). The sample is modest and single-centre, so the result should be regarded as supportive rather than definitive, but it agrees with the established need for careful corneal protection.

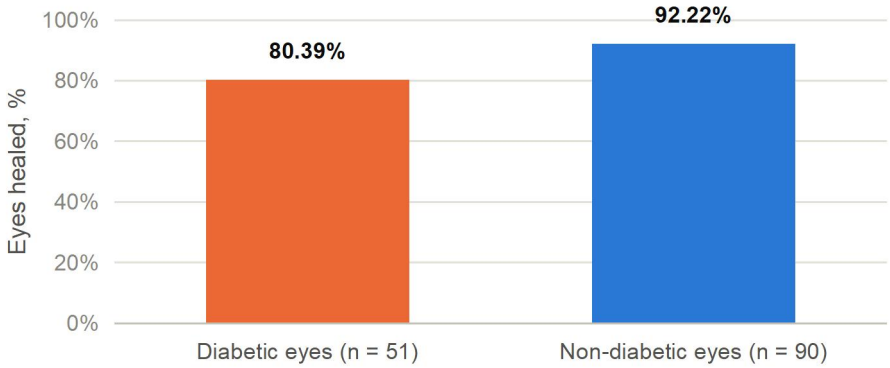


Figure 7. Proportion of eyes with complete epithelial healing of the clear-corneal incision one week after phacoemulsification, assessed by anterior-segment OCT. Data: Chen et al., Cornea 2026 [16] (51 diabetic vs 90 non-diabetic eyes).

Iris and pupil. Poor pharmacological mydriasis is a well-recognized challenge. Autonomic neuropathy, damage to the iris dilator muscle, previous panretinal photocoagulation and posterior synechiae can all limit dilation or cause progressive intraoperative miosis. Eyes with advanced ischemic retinopathy may develop neovascularization of the iris (NVI) or angle, which increases the risk of bleeding and signals sight-threatening retinal ischemia [15]. The surgeon should anticipate pupil-expansion needs rather than react only after visualization becomes inadequate, and mechanical stretching should be avoided when fragile new vessels are present.

Posterior segment. The retina frequently determines the final visual result. A technically perfect cataract operation cannot reverse macular ischemia, established macular scarring, optic neuropathy or the sequelae of advanced DR. Because cataract can prevent reliable fundus examination, preoperative macular OCT and dilated retinal examination are especially valuable whenever media clarity allows. A normal-looking fundus does not exclude structural macular pathology; OCT may reveal edema, an epiretinal membrane or vitreomacular traction that changes counselling, timing and IOL planning [9,10].

3.4. Retinal staging

Retinal disease should be graded with a reproducible scale before surgery. The International Clinical Diabetic Retinopathy Disease Severity Scale [17], used by the AAO Preferred Practice Pattern [18], distinguishes five levels of retinopathy and separately grades macular edema (Table 1). The characteristic lesions are summarized schematically in Figure 8. Grading has direct practical consequences: it determines whether a retinal specialist should be involved, whether retinal

treatment should precede surgery, which prophylactic regimen is appropriate and how intensive postoperative follow-up should be.

Table 1. International clinical diabetic retinopathy severity scale and its perioperative implications (adapted from Wilkinson et al. and the AAO Preferred Practice Pattern [17,18]).

Severity level	**Findings on dilated ophthalmoscopy**	**Perioperative implication**
No apparent DR	No abnormalities	Standard pathway; topical steroid + NSAID; routine diabetic retinal screening
Mild NPDR	Microaneurysms only	Usually good prognosis; baseline OCT recommended; reassess retina after surgery
Moderate NPDR	More than microaneurysms but less than severe NPDR	Baseline OCT; closer review in the first months; consider retinal opinion
Severe NPDR	≥20 intraretinal haemorrhages in each of 4 quadrants, definite venous beading in ≥2 quadrants, or prominent IRMA in ≥1 quadrant, and no PDR (“4-2-1 rule”)	Retinal-specialist involvement; consider treatment before surgery; intensive surveillance
PDR	Neovascularization (disc or elsewhere) and/or vitreous or preretinal haemorrhage	Treat/stabilize (PRP, anti-VEGF) before elective surgery when feasible; consider combined or staged vitrectomy
DME (graded separately)	Retinal thickening or hard exudates; centre-involving vs non-centre-involving on OCT	Treat centre-involving DME first when feasible; distinguish from postoperative PCME using baseline OCT

NPDR, non-proliferative diabetic retinopathy; PDR, proliferative DR; IRMA, intraretinal microvascular abnormalities; PRP, panretinal photocoagulation; DME, diabetic macular edema; PCME, pseudophakic cystoid macular edema.

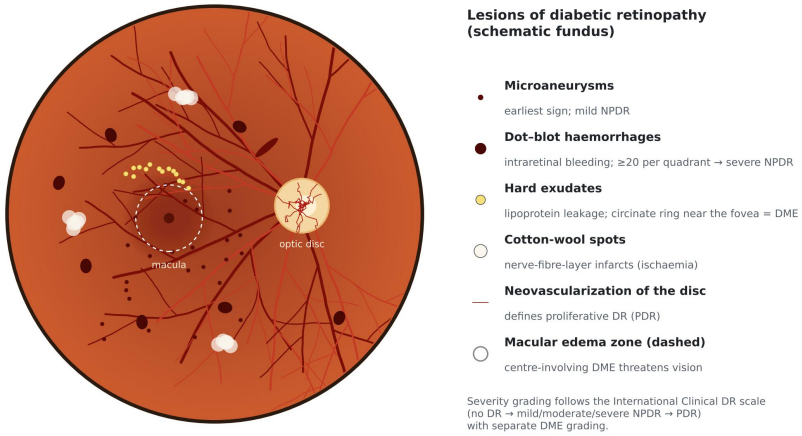


Figure 8. Schematic fundus showing the principal lesions of diabetic retinopathy: microaneurysms, dot-blot and flame haemorrhages, hard exudates in a circinate pattern near the fovea, cotton-wool spots, neovascularization of the optic disc and a zone of macular edema. Original schematic illustration; lesion classification after the International Clinical DR scale [17].

3.5. Preoperative assessment, timing and planning

A complete evaluation should document best-corrected visual acuity (BCVA), pupil responses and dilation, slit-lamp findings including the corneal surface and iris (gonioscopy when NVI is suspected), intraocular pressure (IOP), lens density and retinal status. The history should include diabetes type and duration, current treatment, recent glycemic stability, systemic comorbidity (hypertension, nephropathy, cardiovascular disease), anticoagulant use, previous retinal laser or intravitreal injections, and the patient's functional goals (Table 2). Glycemic and blood-pressure control are important for overall health and retinopathy risk [19], but the literature does not support a single universal HbA1c threshold as an absolute cancellation rule for every cataract procedure; anesthesia and surgical decisions must reflect the patient's overall stability and local perioperative protocols [9].

Timing. Patients with no DR or mild non-proliferative DR (NPDR) usually achieve substantial improvement. Moderate-to-severe NPDR, PDR, centre-involving DME, NVI or suspected traction warrant retinal-specialist involvement. When clinically feasible, active DME and PDR should be treated or stabilized before cataract surgery. If the cataract itself prevents fundus assessment or timely retinal treatment, proceeding may be appropriate, with a coordinated plan for prompt postoperative retinal evaluation and therapy [9,10,18,19]. Expected visual potential, the possibility of postoperative edema and the need for long-term retinal follow-up should be discussed explicitly as part of informed consent.

Imaging. Macular OCT is particularly useful for detecting pre-existing DME, epiretinal membrane or vitreomacular traction. When media opacity prevents OCT, B-scan ultrasonography can exclude gross posterior-segment pathology (retinal detachment, dense vitreous haemorrhage, tractional membranes) but cannot replace OCT for macular edema. Fluorescein angiography may be considered when ischemia or the source of leakage remains unclear and the result would change

management. A baseline OCT is also the only reliable way to distinguish persistent pre-existing DME from new-onset pseudophakic cystoid macular edema (PCME) after surgery [9,10].

Biometry and IOL choice. Glycemic fluctuations can transiently change refraction and corneal measurements; dry eye reduces keratometric repeatability; macular thickening can affect some optical axial-length measurements. Measurements should be repeated when the ocular surface or glycemia is unstable. A hydrophobic acrylic, square-edged, monofocal IOL implanted in the capsular bag is a prudent default: it supports a low rate of posterior capsule opacification and leaves a clear view of the fundus. Silicone IOLs are generally avoided in eyes that may later need vitrectomy with silicone oil. Presbyopia-correcting (multifocal or extended-depth-of-focus) lenses should be considered only after careful review of retinal status, because DR and DME reduce contrast sensitivity [9,15].

Table 2. Practical preoperative checklist for the diabetic cataract patient (synthesized from [9,10,15,19,20]).

Domain	What to check	Why it matters
Systemic	Diabetes type and duration, recent HbA1c and glucose stability, blood pressure, renal function, anticoagulants	Perioperative safety; retinopathy risk; stability of refraction and biometry
Ocular surface / cornea	Tear film, meibomian glands, epithelial staining, corneal sensation, endothelial cell count in dense cataract	Biometry accuracy; epithelial healing; endothelial decompensation risk
Iris / pupil / angle	Dilated pupil size, synechiae, NVI; gonioscopy if suspected	Plan pupil expansion; bleeding and neovascular glaucoma risk
Retina	Dilated fundoscopy, DR grade, macular OCT; B-scan if no view	Visual prognosis, timing, prophylaxis intensity, need for retinal treatment
Biometry & IOL	Repeated keratometry/axial length if unstable; IOL material and design	Avoid refractive surprise; preserve fundus view and future vitreoretinal options
Counselling	Realistic visual expectations; follow-up schedule; warning symptoms	Shared decision-making and early presentation of complications

3.6. Risk stratification: quantitative evidence on macular edema

The largest single source of quantitative data is the UK electronic medical record study by Chu and colleagues, which included 81,984 eyes and assessed macular edema within 90 days of cataract surgery. After excluding eyes that received prophylactic NSAIDs, the baseline incidence of macular edema in eyes without diabetes, operative complications or other recorded risk factors was 1.17%. The relative risk increased to 1.80 in diabetic eyes without DR and to 6.23 in eyes with any DR, and it rose further with increasing DR severity [3] (Figure 9). These estimates support risk-stratified prophylaxis and surveillance, but they do not imply that every diabetic eye with DR has a six-fold absolute probability of edema under contemporary prophylactic regimens.

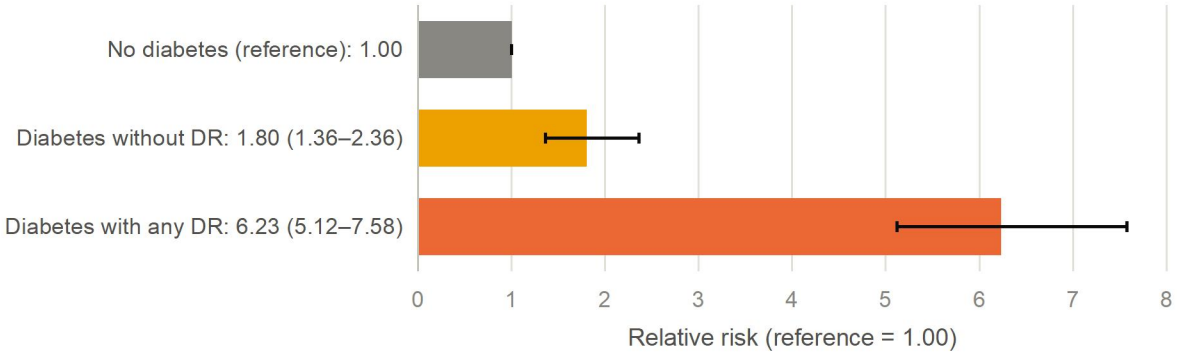


Figure 9. Relative risk of macular edema within 90 days after cataract surgery according to diabetic retinopathy status. Bars show relative risks; error bars show 95% confidence intervals; the reference group has RR = 1.00. Data: Chu et al., Ophthalmology 2016 (81,984 eyes) [3]. Relative, not absolute, estimates.

3.7. Intraoperative considerations and surgical technique

The preferred strategy is careful, low-trauma phacoemulsification with a stable anterior chamber and efficient case flow (Figure 10). Diabetes alone does not require a different operation in every patient, but its anatomical vulnerabilities should shape the technique: protect the corneal epithelium (minimal topical anesthetic toxicity, coating with viscoelastic or balanced salt solution), use a dispersive or soft-shell viscoelastic to protect a vulnerable endothelium, limit ultrasound energy and fluid turbulence, avoid prolonged manipulation and construct a secure, self-sealing wound. The goal is not to minimize surgical steps indiscriminately but to avoid avoidable tissue injury and inflammation, which are the triggers of postoperative macular edema [9,15].

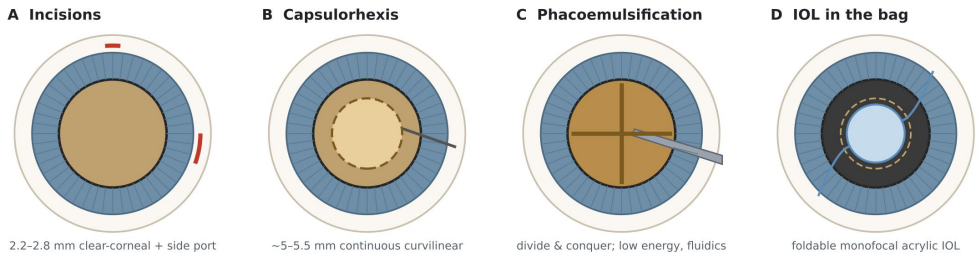


Figure 10. Key steps of small-incision phacoemulsification, as viewed through the operating microscope (schematic): (A) clear-corneal main incision and side port; (B) continuous curvilinear capsulorhexis of adequate size to allow later fundus examination and laser; (C) nucleus emulsification with a low-energy technique; (D) foldable monofocal IOL centred in the capsular bag. Original schematic illustration.

When the pupil is small, options include preoperative topical mydriatics and NSAIDs, intracameral mydriatics, viscomydriasis and mechanical pupil-expansion devices (iris hooks or rings). The choice depends on dilation response, iris anatomy, previous retinal laser and the presence of NVI; fragile vessels increase the risk of hyphema, so aggressive stretching should be

avoided where neovascularization is present [15]. The capsulorhexis should be continuous and of adequate size (about 5–5.5 mm): too small an opening promotes anterior capsule contraction (phimosis), which is more common in diabetes and may hamper later fundus examination and laser, whereas too large an opening increases the risk of posterior capsule opacification and IOL decentration. Dense nuclei increase energy demand and endothelial stress, reinforcing the rationale for preoperative planning. Meticulous cortical clean-up, attention to capsular and zonular integrity and in-the-bag IOL implantation complete the procedure [9,15].

Infection prevention. Endophthalmitis is uncommon but vision-threatening, and diabetes is considered a risk factor for a worse outcome. The randomized ESCRS Endophthalmitis Study (16,603 patients) showed that omission of intracameral cefuroxime at the end of surgery was associated with an approximately five-fold increase in the risk of postoperative endophthalmitis (odds ratio 4.92); clear-corneal incisions, silicone IOL optics and surgical complications were also independent risk factors [21] (Figure 11). Current ESCRS recommendations therefore support intracameral antibiotic prophylaxis together with povidone-iodine antisepsis and sound wound construction [20]. Diabetes is not a reason to substitute topical drops for intracameral prophylaxis; any ocular-surface or adnexal infection should be treated before elective surgery.

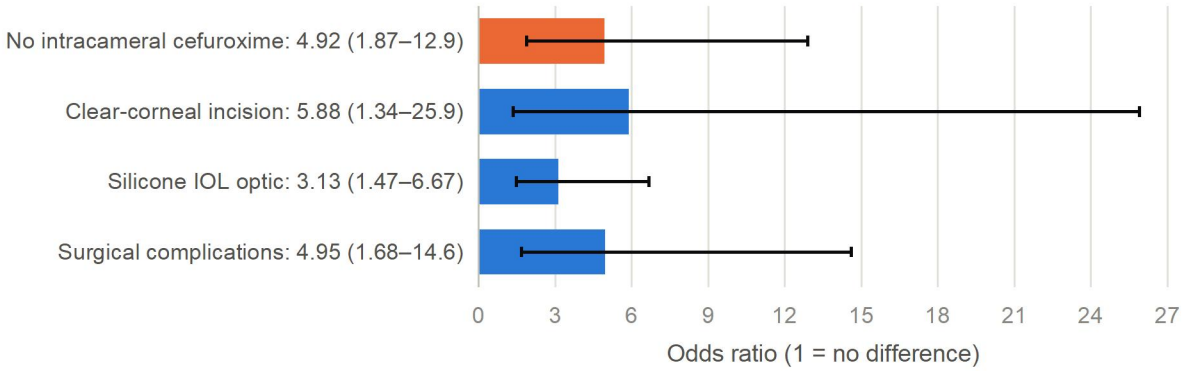


Figure 11. Independent risk factors for postoperative endophthalmitis in the ESCRS multicentre randomized study (16,603 patients). Bars show odds ratios; error bars show 95% confidence intervals (note the wide intervals reflecting the low event rate). Data: Barry et al., J Cataract Refract Surg 2007 [21].

3.8. Special clinical situations

Proliferative DR and vitreous haemorrhage. In eyes with active PDR, vitreous haemorrhage, tractional membranes or inadequate posterior-segment visualization, a combined phacovitrectomy or a staged approach may be needed. Preoperative anti-VEGF injection can reduce intraoperative bleeding, but it must be timed to avoid contraction of fibrovascular membranes and progression of traction. This is not a routine response to diabetes but an individualized decision based on the retinal indication and expected visual benefit [9,15].

Previously vitrectomized and silicone-oil–filled eyes. After vitrectomy, the anterior chamber depth may fluctuate markedly during phacoemulsification (lens–iris diaphragm retropulsion), the posterior capsule may be fragile, and nuclear sclerosis often progresses rapidly. Lower bottle height,

careful fluidics and readiness for capsular complications are required. Silicone IOLs should be avoided because silicone oil adheres to them.

Iris neovascularization and neovascular glaucoma. NVI indicates severe retinal ischemia and should be treated urgently, usually with anti-VEGF therapy and panretinal photocoagulation, before elective cataract surgery whenever possible. Surgery in the presence of active NVI carries a higher risk of hyphema, inflammation, fibrinous reaction and IOP elevation [15,18].

3.9. Postoperative complications: clinical spectrum

Postoperative complications range from transient inflammation and corneal edema to macular edema, DR progression, infection, IOP elevation and posterior capsule opacification. Their probability and consequences depend on pre-existing ocular disease, surgical events, systemic status and the intensity of follow-up. Table 3 groups complications by mechanism and gives risk-adapted responses; it is a synthesis of the cited literature and guidance rather than a validated prediction rule.

Table 3. Postoperative complications in diabetic eyes: mechanisms and risk-adapted responses (synthesized from [9,10,14,15,20,22]).

Complication	Why diabetic eyes may be vulnerable	Clinical implication / response
Pseudophakic CME and/or worsening DME	Breakdown of the blood–retinal barrier plus postoperative inflammatory mediators; pre-existing DME increases concern	Baseline OCT; topical NSAID + corticosteroid prophylaxis; OCT for reduced or delayed visual recovery; retina-directed therapy
Progression of DR, including PDR	Pre-existing retinopathy, chronic hyperglycemia and retinal ischemia; risk differs by baseline severity	Document preoperative grade; treat active PDR when feasible; closer retinal surveillance during the first postoperative year
Corneal epithelial defect, delayed healing, dry eye	Corneal neuropathy, tear-film dysfunction, abnormal basement membrane, reduced sensitivity	Optimize the ocular surface; protect epithelium; investigate persistent pain or staining; use NSAIDs cautiously in compromised corneas
Corneal edema / endothelial injury	Reduced endothelial reserve and susceptibility to surgical stress	Atraumatic technique and viscoelastic protection; monitor edema and recovery
Endophthalmitis or wound infection	Impaired immune defence and healing; consequences can be severe	Antisepsis and intracameral antibiotic; same-day assessment for pain, redness, hypopyon or vision loss
IOP elevation / steroid response	Inflammation, retained viscoelastic, steroid response, neovascular glaucoma	Measure IOP in high-risk eyes; closer monitoring after depot steroids; evaluate NVI and the angle

Posterior capsule opacification, anterior capsule phimosis	Greater lens-epithelial proliferation and inflammation in diabetes	Square-edged acrylic IOL, adequate capsulorhexis; Nd:YAG capsulotomy only after retinal assessment
Refractive surprise	Dry eye and glycemic fluctuation affect keratometry and refraction	Repeat measurements if unstable; avoid definitive refraction during unstable glycemia

3.10. Macular edema: diagnosis, prophylaxis and treatment

Postoperative macular edema in diabetic eyes is heterogeneous: it may represent classic Irvine–Gass PCME driven by prostaglandin-mediated inflammation, worsening of pre-existing DME, or a combination. OCT is the key diagnostic tool; it shows loss of the foveal contour, increased central subfield thickness and intraretinal cystoid spaces (Figure 12). Subclinical increases in macular thickness are common after surgery in diabetic eyes, and Kim and colleagues showed with OCT that eyes with more severe retinopathy and pre-existing edema were more likely to develop clinically relevant thickening [23]. In the prospective DRCR Retina Network study of eyes with DR but without central-involved DME at baseline, about 10–12% of eyes with non-central or possible central-involved DME developed central-involved edema by 16 weeks, and a history of prior DME treatment was associated with higher risk [24].

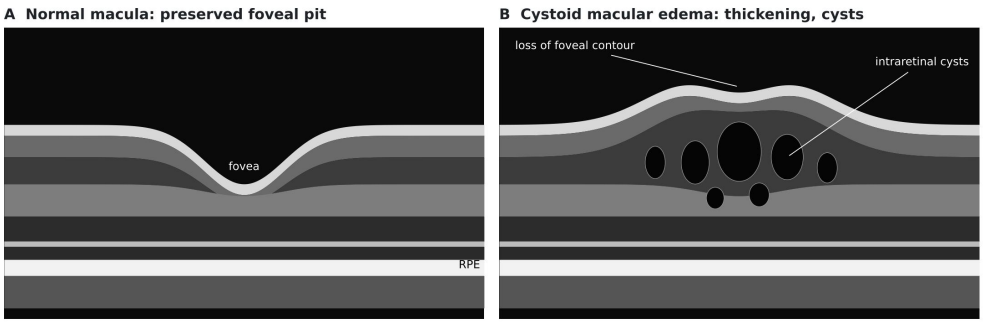


Figure 12. Schematic OCT B-scans of the macula. (A) Normal macula with a preserved foveal depression and regular retinal layers. (B) Cystoid macular edema after cataract surgery: retinal thickening, loss of the foveal contour and hyporeflective intraretinal cysts. Original schematic illustration.

Prophylaxis. The strongest practical distinction is between preventing routine postoperative inflammation and treating established retinal disease. For diabetic patients without DR, ESCRS guidance recommends a combination of topical corticosteroid and NSAID drops; in patients with DR, additional steroid strategies may be considered in selected cases, while depot corticosteroids can raise IOP and require monitoring [20]. Laursen and colleagues' systematic review and meta-analysis reported that topical corticosteroid plus NSAID prevented 75.8% of PCME events compared with corticosteroid alone in diabetic patients without preoperative DME; depot steroid

plus topical therapy was more effective in some analyses but increased the likelihood of raised IOP, and adding anti-VEGF did not reduce PCME prevalence across the diabetic population [5].

In the randomized ESCRS PREMED trial in diabetic patients (213 patients), none of the eyes that received subconjunctival triamcinolone acetonide 40 mg developed CME within 12 weeks, compared with 7.6% of eyes that did not, whereas intravitreal bevacizumab had no additional effect (CME 4.7% vs 4.3%) [25]. A Bayesian network meta-analysis of 17 RCTs (2,566 participants) found a lower risk of macular edema at one and three months with additional topical NSAIDs or intravitreal anti-VEGF compared with placebo or topical steroid alone; anti-VEGF groups also had modestly better BCVA at one and three months, but this advantage was no longer significant at six months [11]. Differences in populations, timing, DR/DME status and outcome definitions caution against treating prophylactic intravitreal injection as mandatory for every diabetic patient. Options are summarized in Table 4.

Table 4. Prophylactic and therapeutic options for macular edema in diabetic cataract surgery.

Strategy	Typical use	Evidence and cautions
Topical corticosteroid + topical NSAID	Default for diabetic eyes without DR or with mild DR and no DME	Prevented 75.8% of PCME events vs steroid alone [5]; ESCRS-recommended [20]; NSAIDs need caution with epithelial disease
Subconjunctival / sub-Tenon triamcinolone	Selected higher-risk eyes (DR without active centre-involving DME)	No CME vs 7.6% without it in PREMED [25]; risk of IOP rise—monitor
Intravitreal anti-VEGF at surgery	Active or recently treated centre-involving DME, PDR	Short-term reduction of edema and BCVA benefit, not sustained at 6 months [11,12]; not universal prophylaxis [5,25]
Intravitreal dexamethasone implant / triamcinolone	Persistent or steroid-responsive DME, pseudophakic eyes	Effective in selected eyes; cataract formation is no longer a concern in pseudophakic eyes, but IOP must be monitored
Panretinal photocoagulation	PDR, severe NPDR with high-risk features	Remains standard for PDR; complete before or soon after surgery [18,19]

3.11. Progression of diabetic retinopathy after surgery

The relation between cataract extraction and DR progression is complex. Removing the lens improves fundus visualization and enables laser or intravitreal treatment, while postoperative inflammatory and vascular changes may coincide with progression that was already likely because of the natural history of diabetes. Observational comparisons are prone to confounding by indication, because patients who undergo surgery may have more advanced cataract, longer diabetes duration or more active eye disease [4,9]. Prospective paired-eye data from Squirrell and colleagues suggested that, in patients with type 2 diabetes, uncomplicated phacoemulsification did not by itself accelerate retinopathy, although maculopathy progressed in some operated eyes [26].

The most comprehensive recent synthesis is the 2025 meta-analysis by Lee and colleagues (15 studies, 7,287 patients). Compared with controls, postoperative incidence of DR was increased (RR 1.38, 95% CI 1.16–1.63) and progression of established DR was also increased (RR 1.46, 1.28–1.66). Progression to sight-threatening DR, including clinically significant macular edema and PDR, was higher (RR 1.84, 1.21–2.81). However, paired-eye analyses of incident DR showed no significant difference (RR 0.85, 0.39–1.83), reflecting imprecision and heterogeneity [4] (Figure 13). A 2025 TriNetX analysis of patients with type 2 diabetes reported an adjusted hazard ratio of 1.52 (95% CI 1.06–2.19) for progression from NPDR to PDR within one year after surgery in a sensitivity cohort with at least five years of diabetes [27]. These data support active monitoring but do not prove that surgery alone causes all observed progression.

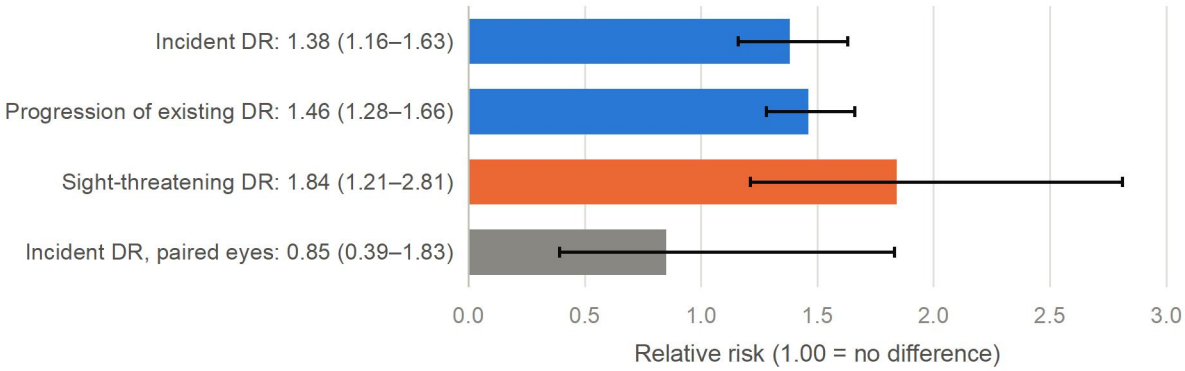


Figure 13. Diabetic retinopathy outcomes after cataract surgery compared with controls: pooled relative risks (bars) with 95% confidence intervals (error bars). Values above 1.00 indicate higher risk after surgery; the paired-eye estimate crosses 1 and is inconclusive. Data: Lee et al., Am J Ophthalmol 2025 (15 studies, 7,287 patients) [4].

3.12. Posterior capsule opacification and late complications

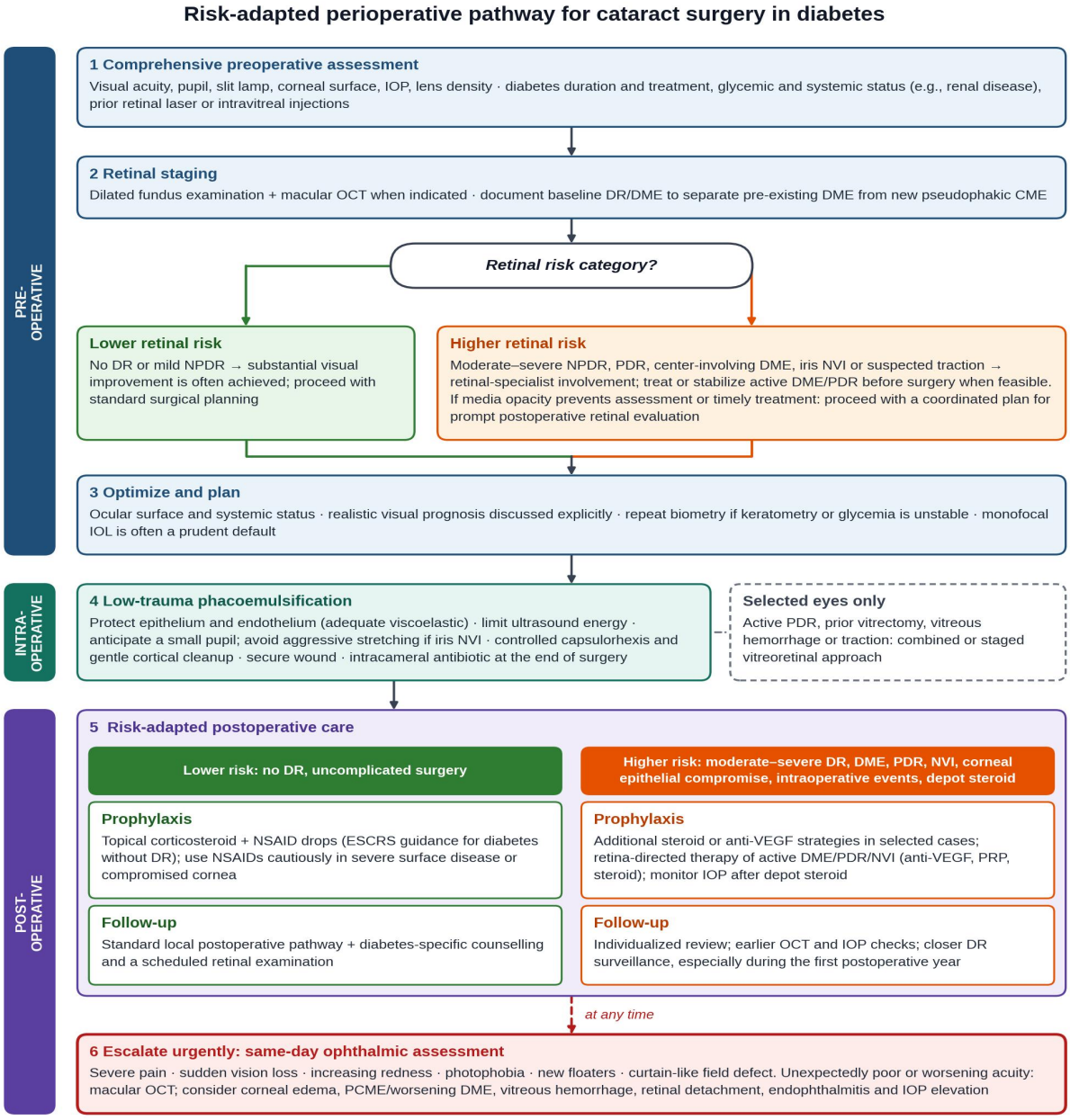
Diabetic eyes show a more pronounced inflammatory response and greater lens-epithelial cell activity after surgery. In a prospective study, Hayashi and colleagues found that the degree of posterior capsule opacification (PCO) was significantly greater in diabetic than in non-diabetic eyes and was related to retinopathy status [22]. PCO causes a late, gradual decline in vision that must be distinguished from macular disease; a macular OCT should precede Nd:YAG capsulotomy when visual loss is out of proportion to capsular haze, and IOP should be checked after laser. Anterior capsule contraction, IOL decentration and, in the long term, late progression of retinopathy also justify continued ophthalmic follow-up beyond the routine postoperative period [9,15].

3.13. Postoperative surveillance and urgent warning signs

Follow-up intensity should be proportional to risk. Eyes with no DR and an uncomplicated operation may follow standard local postoperative pathways, combined with diabetes-specific counselling and a scheduled retinal examination. Eyes with moderate or severe NPDR, current or previous DME, PDR, NVI, corneal epithelial compromise, significant intraoperative events or depot steroid use need individualized review with earlier OCT and IOP checks. A one-size-fits-all schedule can miss clinically important change in higher-risk eyes [4,9,20].

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Unexpectedly poor or worsening acuity should not automatically be attributed to slow recovery. The differential diagnosis includes corneal edema, residual refractive error, PCME, worsening DME, vitreous haemorrhage, retinal detachment, endophthalmitis and raised IOP. Severe pain, sudden loss of vision, increasing redness, photophobia, new floaters or a curtain-like field defect require same-day ophthalmic assessment [15,20]. The risk-adapted pathway is summarized in Figure 14; it is a practical synthesis of the literature, not a separately validated decision instrument.



DR, diabetic retinopathy; NPDR, nonproliferative DR; PDR, proliferative DR; DME, diabetic macular edema; PCME, pseudophakic cystoid macular edema; NVI, iris neovascularization; PRP, panretinal photocoagulation; IOP, intraocular pressure; OCT, optical coherence tomography.

Figure 14. Risk-adapted perioperative pathway for cataract surgery in diabetes, synthesized from current reviews and ESCRS/ADA guidance [9,19,20]. Green and orange boxes denote lower

and higher retinal risk; the red box denotes urgent escalation. Intended for clinical discussion and to be adapted to local protocols.

3.14. Evidence summary

Table 5. Summary of key evidence and its practical implications.

Source	Population / design	Main finding	Interpretive caution
Becker et al., 2018 [2]	UK population cohort	Cataract incidence 20.4 vs 10.8 per 1,000 PY; largest excess at 45–54 y	Observational; coding-based diagnoses
Chu et al., 2016 [3]	81,984 eyes; database cohort	Macular edema RR 1.80 without DR and 6.23 with DR	NSAID recipients excluded; relative, not absolute, risk
Laursen et al., 2019 [5]	Meta-analysis of RCTs	Steroid + NSAID prevented 75.8% of PCME events vs steroid alone	Protocols varied; depot steroid raises IOP
Wielders et al., 2018 [25]	RCT, 213 diabetic patients	Subconjunctival triamcinolone: 0% vs 7.6% CME; bevacizumab no added effect	Short follow-up (12 weeks); IOP monitoring needed
Zhang et al., 2022 [11]	Network meta-analysis; 17 RCTs, 2,566 participants	NSAID or anti-VEGF lowered edema at 1–3 months	No BCVA benefit at 6 months
Lee et al., 2025 [4]	Meta-analysis; 15 studies, 7,287 patients	DR progression RR 1.46; sight-threatening RR 1.84	Heterogeneity; paired-eye results inconclusive
Barry et al., 2007 [21]	RCT, 16,603 patients	No intracameral cefuroxime: OR 4.92 for endophthalmitis	Not diabetes-specific; low event rate
ESCRS recommendations [20]	Evidence-based guideline	Risk assessment; steroid + NSAID in diabetes; intracameral antibiotic	Adapt to local protocols and contraindications

Taken together, the evidence favours planned rather than reflexive care. The most reproducible risk discriminator is the retinal phenotype, particularly existing DR and DME. The most consistently supported process measures are comprehensive preoperative examination, retinal imaging when indicated, atraumatic surgery with intracameral antibiotic prophylaxis, appropriate anti-inflammatory prophylaxis and follow-up that extends beyond the first uncomplicated postoperative visit.

3.15. Limitations of the evidence and future directions

Much of the evidence is observational, heterogeneous in definitions of macular edema and DR progression, and generated before current prophylactic and anti-VEGF practices became widespread. Many trials are small, with short follow-up, and few report outcomes separately for type 1 and type 2 diabetes or for different levels of glycemic control. Future research should prioritize adequately powered randomized trials stratified by DR and DME severity, standardized OCT-based outcome definitions, and patient-centred endpoints such as contrast sensitivity and quality of life. Artificial-intelligence-assisted DR screening, widefield and OCT-angiography imaging, sustained-release intraocular drug delivery and integrated diabetes–ophthalmology care pathways are promising tools for identifying high-risk eyes and delivering timely treatment, particularly in health systems with limited specialist capacity [13,19].

4. CONCLUSIONS

Cataract surgery in diabetic patients is generally effective, but it requires a broader perioperative assessment than surgery in otherwise healthy eyes. Diabetes roughly doubles the incidence of cataract, causes earlier and faster lens opacification through polyol, glycation and oxidative mechanisms, compromises the ocular surface and corneal endothelium, impairs pupillary dilation, destabilizes biometry and coexists with sight-threatening retinal disease. Surgeons should therefore plan around the patient's ocular phenotype rather than diabetes status alone.

Preoperative retinal grading and macular OCT help define visual potential, reveal DME and guide timing. Active DME, PDR or iris neovascularization should be treated or stabilized when feasible, with coordinated cataract–retina decision-making when the cataract impedes diagnosis or treatment. Low-trauma phacoemulsification, corneal and endothelial protection, preparation for a small pupil, an adequate capsulorhexis, an in-the-bag acrylic IOL, a secure wound and intracameral antibiotic prophylaxis are essential elements of care.

Postoperative macular edema is the complication with the strongest evidence of increased risk, especially in eyes with DR. Topical corticosteroid plus NSAID prophylaxis is supported for most diabetic eyes; periocular or intravitreal steroid and anti-VEGF therapy should be reserved for appropriately selected patients and balanced against adverse effects such as IOP elevation. Current meta-analytic evidence also supports enhanced DR surveillance during the first postoperative year. Diabetes is not, by itself, a contraindication to cataract surgery: the best outcomes are achieved when surgery is embedded in an individualized pathway that combines systemic coordination, retinal assessment, tissue-sparing technique, evidence-informed prophylaxis, clear warning-sign counselling and risk-adapted follow-up.

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