Chapter

Cataract Surgery Complications: *Vitreo-Retina Perspectives*

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Abstract

Cataract surgery is one of the most common and successful intraocular surgeries performed worldwide. However, sight-threatening complications that involve the posterior segment can occur. The incidence of such complications is relatively low, but it is important to recognize these complications early and treat them appropriately. In this chapter, we will address some of the important posterior segment complications of cataract surgery such as intraoperative complications (e.g., vitreous loss, retained lens matter, and suprachoroidal hemorrhage) and short- and long-term postoperative complications (e.g., postoperative endophthalmitis, rhegmatogenous retinal detachment, cystoid macular edema and progression of preexisting diabetic retinopathy, and/or diabetic macular edema).

Keywords: cataract surgery, complications, posterior segment, retina, vitreous, diabetic retinopathy, diabetic macular edema

1. Introduction

Cataract surgery is one of the most common and successful intraocular surgeries performed worldwide. With latest innovations and technologies, such a surgery became easy to perform with less operating time and shorter postoperative recovery period. However, sight-threatening complications that involve the posterior segment can occur. The incidence of such complications is relatively low, but it is important to recognize these complications early and treat them appropriately. In this chapter, we will address some of the important posterior segment complications of cataract surgery such as intraoperative complications (e.g., vitreous loss, retained lens matter, and suprachoroidal hemorrhage) and short- and long-term postoperative complications (e.g., postoperative endophthalmitis, rhegmatogenous retinal detachment, cystoid macular edema and progression of preexisting diabetic retinopathy, and/or diabetic macular edema).

2. Vitreous loss and retained lens matter

An intact posterior lens capsule (PC) is an important normal anatomical barrier, which separates the vitreous body from the forces resulting from cataract surgery and intraocular lens (IOL) implantation. The incidence of PC tear and vitreous loss is variable depending on the surgeon's skills, years of experience, surgical volume, and complexity of cataract case mix.

The reported incidence ranges from 0.45–8.22% [1–3].

Vitreous loss can lead to an increased risk of sight-threatening complications, including cystoid macular edema, retinal detachment, and endophthalmitis [4–6]. Significant PC tear might be followed by dislocation of lens fragment(s) into the vitreous cavity [7].

Prevention: Risk stratification is an important measure to reduce such risk by appropriate patient selection, pre-op preparation (for detailed counseling, selecting the appropriate type of anesthesia as well as time of posting the case, and selection of surgeon experienced in dealing with complex cataract surgery).

Intra-operatively, recognizing signs of PC tear and vitreous loss are crucial. The signs to observe are sudden deepening of the anterior chamber (AC), excess sideways shift of the nucleus, sudden appearance of a red reflex, and abnormal movement of the pupillary margin distant from instruments in the AC secondary to traction transmitted through vitreous strands. If any of above signs were observed, viscodispersive substance was to be injected immediately into the AC directly above the site of PC tear while the irrigation is on to minimize vitreous prolapse and to stabilize any remaining lens fragments. At this point, the surgeon should assess the situation and change the approach appropriately for such a challenge, for example, setting up anterior vitrectomy and augmenting the local anesthesia if needed. The ultimate goals of management of PC tear and vitreous loss are removal of the prolapsed vitreous from the AC and surgical wounds, safe removal of the remaining lens fragment(s), and safe IOL implantation. The prolapsed vitreous should be removed with anterior vitrectomy with highest cut rate provided by the machine and maintaining stable AC and IOP. A sutureless vitrectomy technique *via* 23G pars plana anterior vitrectomy whenever appropriately possible might be the most controlled way to carry out safe

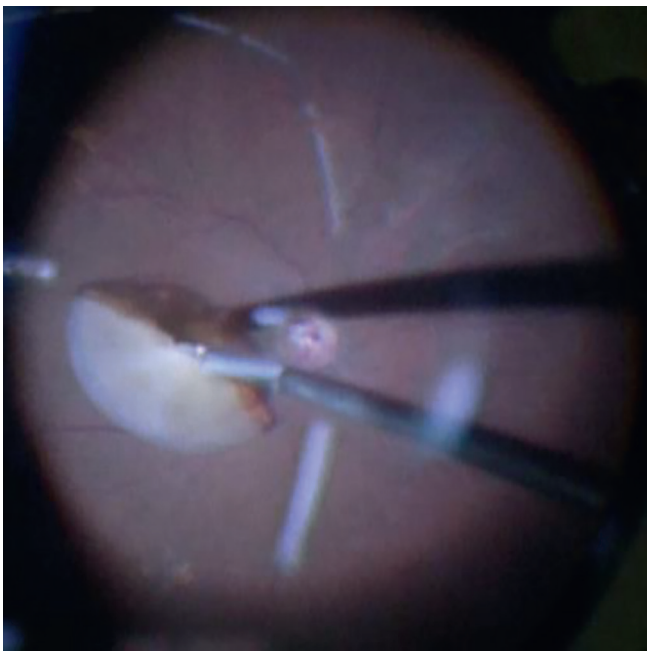

Figure 1.
Intraoperative fundus photo shows retained nuclear fragment being fragmented and removed using 23G vitrectomy probe (courtesy of Dr. M. Al-Abri).

and adequate anterior vitrectomy. This approach can be done with either an anterior chamber or pars plana infusion [8]. Such an approach mitigates AC manipulation, less risk for vitreous incarceration in the surgical wounds, and serves as logical way for adequate and safe removal of vitreous behind the plane of PC, which may preserve the residual PC from further damage. In addition, if the lens fragments threatened to fall posteriorly, pars plana approach may help in pushing the lens fragments into the AC using the vitrector. Residual soft lens matter or cortex can be safely removed using the vitrector itself. However, residual nuclear fragments can be removed either by carefully using the phaco probe, after the AC has been adequately cleaned from vitreous, or can be removed manually through an enlarged main wound. Viscoelastic or a lens glide may help to stabilize nuclear fragments in the AC before removal [9]. If appropriate, insertion of the appropriate IOL is to be done in a suitable position based on the judgment and experience of the primary surgeon. If nuclear fragments are moved posteriorly behind the plane of the PC, aggressive efforts to retrieve these fragments without pars plana vitrectomy can result in giant retinal tears and retinal detachments [10, 11]. In such cases, an urgent referral is to be made to a vitreo-retina surgeon, who can then make the appropriate decisions about the timing of the management meanwhile the patients should be monitored closely for post-op inflammation, corneal edema and IOP, and other potential complications (**Figure 1**).

3. Suprachoroidal hemorrhage

Suprachoroidal hemorrhage (SCH) is one the rare but potentially vision-threatening intraoperative or postoperative complication. It is caused by acute hypotony leading to rupture of long and short ciliary arteries, which bleed into suprachoroidal space. The incidence is extremely low, 0.03–0.81% [12].

Predisposing factors: Advanced age, uncontrolled hypertension, using anticoagulant or antiplatelet, atherosclerosis, diabetes, uncontrolled glaucoma, high axial myopia, aphakia, choroidal hemangioma, retrobulbar anesthesia, Valsalva maneuvers, and prolonged or complicated cataract surgery with vitreous loss and hypotony. Proper preoperative evaluation, identification, and management of avoidable risk factors, for example, control of high BP, high IOP, discontinue anticoagulant or antiplatelet, choose the appropriate method of anesthesia, and address cardiovascular and other systemic issues.

Clinical features: Intraoperative, progressive anterior chamber shallowing, loss of red reflex, increasing IOP with firming of eyeball, iris prolapse, vitreous extrusion, and extrusion of intraocular contents.

Intraoperative management: Terminate the surgery immediately. Gently reposition the expelled contents and quickly close the surgical wounds. Reform anterior chamber either with viscoelastic or air bubble. Immediate drainage and posterior sclerotomy are controversial.

Postoperative management: Intraocular pressure (IOP) and inflammation are controlled with IOP lowering agents (Topical Beta blocker & Carbonic anhydrase inhibitors – CAIs and/or systemic acetazolamide) and topical steroids. Cycloplegics and analgesics relieve the eye pain. NSAIDS and antiplatelets are to be avoided. Serial Ultrasound B – scan to be performed to monitor the progress and liquefaction of suprachoroidal hemorrhage (**Figure 2**).

Drainage is considered if hemorrhage does not resolve spontaneously after 2 weeks. Optimum IOP and sufficient liquefaction of clot are essential for successful

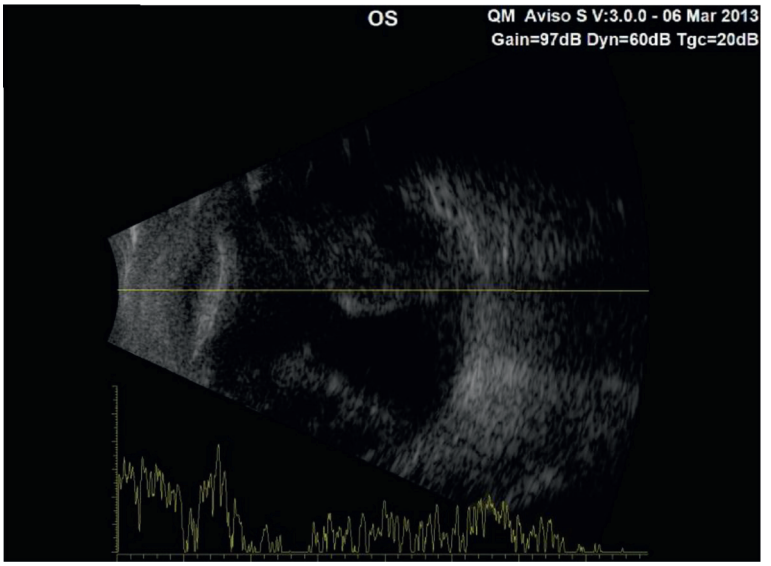

Figure 2.
B-scan ultrasound image of a patient with suprachoroidal hemorrhage post-cataract surgery. Notice the dome-shaped appearance and central apposition of detached choroid. The suprachoroidal space is filled with moderate amplitude spikes suggestive of suprachoroidal hemorrhage (courtesy of Dr. A. Al-Hinai).

procedure. The suprachoroidal lysed hemorrhage is drained either through direct 3-4 mm radial sclerotomies or transconjunctival trocar. Pars plana vitrectomy (PPV) is considered if it is complicated by retinal detachment and/or vitreous hemorrhage. Overall, visual outcome is variable and depends on the extent of SCH, associated retinal detachment as well as other ocular comorbidities. Generally, it carries poor visual prognosis especially if it is associated with retinal detachment, where some reported cases end up with <20/200 – no light perception visual acuity [13, 14].

4. Endophthalmitis post cataract surgery

Endophthalmitis is defined as intraocular inflammation involving the anterior and posterior segments of eye that could be acute, chronic or delayed onset, infective or noninfective, and exogenous or endogenous.

Acute postoperative endophthalmitis: Acute infectious endophthalmitis is a highly catastrophic and sight-threatening rare complication of cataract surgery. Presented within 6 weeks, mostly in 3–5 days with fulminant or acute course. Classic presentation is eyelid swelling, ocular pain, ciliary congestion, and sudden drop of vision, which may be associated with corneal edema, hypopyon, loss of red pupillary reflex, and purulent vitritis. (**Figure 3**).

*Incidence***:** The incidence of acute infectious post-cataract endophthalmitis is inconsistent and varies, 0.04–0.092% [15, 16]. According to scientific literature, the incidence is globally declined with off-label (No FDA approval) use of intracameral antibiotic injections [17, 18].

Source of infection and risk factors: The flora from eyelids, conjunctiva, nasolacrimal, and infected anophthalmic socket are major sources and perioperative surgical wound contamination through a contaminated instrument or theater personnel.

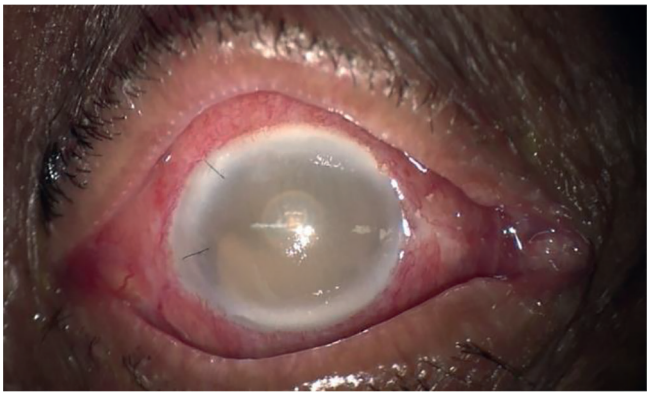

Figure 3.
Anterior segment photo of the right eye with acute endophthalmitis post-cataract surgery. Notice the diffuse conjunctival congestion, marked corneal edema, organized hypopyon, and loss of red pupillary reflex (courtesy of Dr. M. Al-Abri).

While elderly patients and immune-compromised due to diabetes mellitus, systemic malignancy, and HIV are risk factors for endogenous endophthalmitis. Other complicated surgery associated with posterior capsule rupture, vitreous loss, anterior chamber IOL, prolonged surgery, and wound leak.

Pathogens and pathogenesis: Postoperative endophthalmitis is predominantly caused by bacteria. Gram-positive isolates in almost 85% of cases. Most commonly coagulase-negative - the normal bacterial flora from eyelids. Mostly *Staphylococcus epidermidis* in 30.3%, and others are Streptococcus viridans and *Staphylococcus aureus*. The Gram-negative accounts for 10.3% (*Pseudomonas aeruginosa*, Klebsiella pneumonia) and fungi for 4.6%, including *Candida albicans* and *Aspergillus* species [19].

Once microbes get access into the eye, initiate to release intravitreal inflammatory cytokines, like tumor necrosis factor-alpha, interleukin-1 beta, and interferon-gamma that result in neutrophil migration and aggregation leading to moderate-severe suppurative inflammation and retinal tissue necrosis.

Prevention and prophylaxis: Strict aseptic measures throughout surgical process are key to infection prevention. Meticulous cleaning of periocular skin, and eyelids with 10% Povidone-Iodine solution and instillation of few drops of 5% povidone-iodine antiseptic into conjunctival cul de sac and left for 3–5 minutes followed by irrigation with sterile normal saline significantly reduce the risk of infection from adnexal flora. Proper sterile draping of surgical site with complete covering of eyelid margins and eyelashes is considered standard care. Currently, preoperative use of topical antibiotics and intracameral cefuroxime (1 mg/0.1 ml) injection at the end of surgery is common in practice [20]. Immediate closure of postoperative surgical wound leak, if noticed, is recommended.

Differential diagnosis: Post-cataract endophthalmitis must be differentiated from toxic anterior segment syndrome [TASS], keratitis, postoperative uveitis (e.g., related to retained lens material), and vitreous hemorrhage.

Management: When any suspicious postoperative endophthalmitis makes a prompt arrangement to collect specimen from aqueous with 30-G tuberculin syringe (0.1–0.2 ml) and vitreous with 23–25-G syringe (0.2–0.4 ml) to detect the causative organisms. Prepare smear for staining of Gram, Giemsa, potassium hydroxide [KOH], and culture – blood and chocolate agar, Sabouraud. Modern technology of

DNA sequencing and polymerase chain reaction (PCR) testing of vitreous biopsy is rapid and highly specific and sensitive in microbe detection but still uncommon in practice [21, 22]. Ultrasound B-scan is routinely performed to assess the severity of vitreous activity and to exclude retinal detachment.

Treatment: The intravitreal antibiotics are mainstay of treatment if visual acuity is better than light perception. Intravitreal administration of antibiotics maintains the adequate minimum inhibitory concentration (MIC) for sufficient time. The initial empirical and standard therapy is intravitreal vancomycin (1 mg/0.1 ml) for Gram-positive and ceftazidime (2.25 mg/0.1 ml) for Gram-negative bacterial coverage [23]. Ceftazidime has much better safety profile than amikacin (0.4 mg/0.1 ml), which can be used if patient is allergic to cephalosporin.

Dexamethasone (0.4 mg/0.1 ml) to halt the inflammation is an option (provided no fungal infection), however, its use is controversial because of no impact on final visual outcome. Adjuvant-fortified topical vancomycin and ceftazidime are used to address contaminated surgical wound and anterior segment involvement. Atropine 1% eye drops as cycloplegic agent is used to relieve ciliary spasm and eye pain. Systemic antibiotics are often administered.

If fungal infection is suspected or detected, intravitreal amphotericin B (5 mcg/0.1 ml) or voriconazole (100 mcg/0.1 ml) are considered. Practicing ophthalmologists are still following Endophthalmitis Vitrectomy Study (EVS) guidelines which state that pars plana vitrectomy (PPV) with intravitreal vancomycin and ceftazidime is recommended if visual acuity is only light perception [24]. However, the current practice has shifted from the EVS recommendations and recommends early PPV in hand motion or better [25, 26]. Moreover, PPV is preferred if the condition clinically worsens within 48 hours of intravitreal antibiotics rather repeat IVT injection and rarely for globe salvage in no light perception [27].

Outcomes: Overall, outcome of endophthalmitis is poor. However, outcome depends on onset, microbial virulence, and duration of the infection. Poor vision at presentation, fulminant onset, hypopyon >1.5 mm, invisible fundus and retinal detachment often have poor prognosis.

EVS describes if visual acuity is light perception at presentation (33% achieve 20/40, 56% gain 20/100 or better, and 5/200 or worse in 20% of patients [24]. Gram-negative organism-*P. aeruginosa,* Klebsiella, and infection with Bacillus species (e.g., cereus) have aggressive course and end up with severe visual loss [28].

Delayed postoperative endophthalmitis: Delayed postoperative endophthalmitis develops after 6 weeks to years of cataract surgery, and often masquerades as autoimmune uveitis. Delayed endophthalmitis has indolent course, caused by low-virulent pathogen—*Propionibacterium acnes* or *S. epidermidis*, sometimes *Aspergillus* or Candida fungi [29].

Clinical features: Painless progressive decreased visual acuity, low-grade chronic granulomatous uveitis associated with large keratic precipitates, +/− hypopyon, plaque-like material on the posterior capsule, and vitritis.

Treatment: Aqueous and vitreous specimen for staining and culture/sensitivity of microbes includes anaerobes.

Injecting antibiotics into the capsular bag or vitreous cavity usually does not eliminate the infection. Pars plana vitrectomy with complete or partial removal of capsular beg and exchange of the intraocular lens followed by intravitreal vancomycin plus ceftazidime. It is advisable to send the explanted lens and capsular beg for cultures.

Toxic anterior segment syndrome (TASS): TASS is an acute postoperative anterior chamber sterile inflammatory reaction. Noninfectious material, such as contaminants

from surgical equipment, viscoelastic, intraocular lens implant, or solution toxins, enters the anterior segment and develops toxic reaction to the intraocular structure within 12–24 hours after uneventful cataract surgery.

Clinical features: Decreased visual acuity, mild or no eye pain, limbus to limbus corneal edema, moderate-severe anterior chamber reaction with cells, flares, sterile hypopyon and fibrin, and pupil may be dilated and sluggish to nonreactive and +/− high intraocular pressure.

Treatment*:* It responds very well to intensive topical steroid q1 hourly (e.g., prednisolone 1%), and cycloplegic (e.g., cyclopentolate 1%) with IOP monitoring.

5. Pseudophakic rhegmatogenous retinal detachment

Cataract surgery is one of the most commonly performed intraocular surgery with this there is increased interest in the potential postoperative pseudophakic rhegmatogenous retinal detachment (PRD) (**Figure 4**).

PRD is an uncommon complication after cataract surgery; the reported incidence varies widely within a range of 0.16 to 3.55% and an average of 0.7% [30–32], which is higher than in the general population (incidence of 0.0063–0.0179%) [33, 34]. Most of the reported PRD occurs within 1–2 years of the cataract removal. In a large cohort Korean study, it was found that 80% of PRD occurred within 1 year after cataract surgery [35] and another study from Moorfield's Eye Hospital found that 75% of PRD occurred within the first 2 postoperative years [36]. However, others found that the mean time between cataract surgery to PRD diagnosis was 40 months and even after 4 years [37].

Risk factors: Myopia is a well-known risk factor for PRD with 4.2- to 6.1-fold increased risk of PRD in high myopia and 1.6 to 3.2 in moderate myopia [31, 38–40]. Therefore, peripheral retinal degeneration changes predisposing to retinal detachment are commonly found in myopic eyes to be thoroughly evaluated and treated

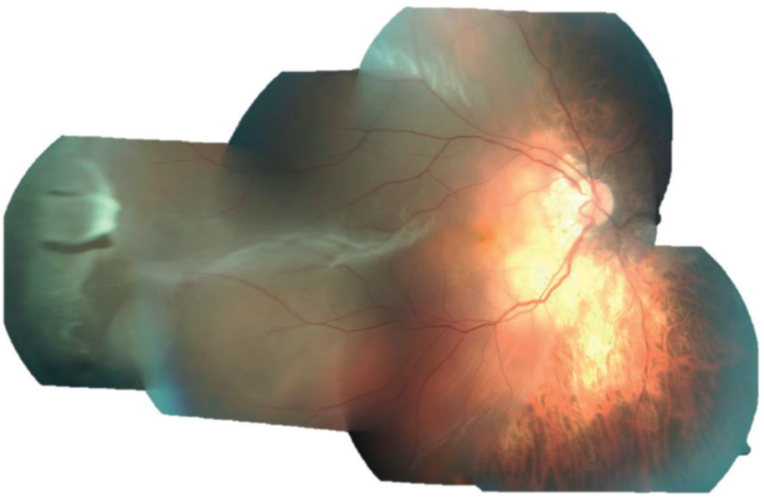

Figure 4.
Right eye fundus photo of a 27-year-old male status post ICL implantation for high myopia (AL 31.06) showing myopic fundus, macula-off retinal detachment with HST at 9 o'clock position (courtesy of Dr. M. Al-Abri).

prophylactically at least 2 weeks prior to performing cataract surgery. It has been suggested that younger age had a linear relationship with risk of PRD [40]. In a Korean population-based study, the estimated 5-year risk of RD was inversely correlated with the mean age of the patients (4.26, 2.38, 0.94, and 0.43% for patients of age groups 40–54, 55–64, 65–74, and ≥ 75, respectively; $p < 0.0001$) [35].

The increased risk of PRD in young population could be attributed to various etiologies; cataract surgery itself may induce posterior vitreous detachment (PVD), which may lead to retinal breaks, leading to RD [41, 42]. Another explanation is that the underlying causes for cataract formation in younger age group might act as predisposing factors for PRD. For example, the effect of unreported and underdetected eye trauma or myopia may be misinterpreted as the effect of younger age. In recent years, relatively early cataract surgery with multifocal IOL implantation is being performed as a treatment for presbyopia or clear lens exchange in even younger age groups for refractive errors. Considering the higher incidence of RD after lens extraction surgery in younger population, it is important to determine if cataract surgery is medically indicated and to have thorough discussion with patient about the risk of retinal detachment. Previous studies have reported male gender is a risk factor for the development of PRD. The cumulative 6-year RD rates were 1.90% in the male and 0.56% in the female subgroups of the prospective cohort study done in Taiwan [43]. This could be due to the possibility that men may have more history of under-reported trauma. The integrity of the posterior capsule is an important determinant of the onset of PVD and, hence, the risk of RD. The anterior movement of the vitreous, persistent anterior traction on the vitreous base due to vitreous adherence to the wound, IOL, and anterior segment structures may result in an increased risk of traction-induced peripheral retinal tears [35]. According to many studies, posterior capsular rupture (PCR) is a major risk factor for PRD [31, 38, 44]. It increases the risk of RD by 10-fold in one study [45] and in another report, an association was confirmed between PCR and RD occurrence with an odds ratio of 19.9 (95% CI 10.8–36.7) [40].

6. Cystoid macular edema

Cystoid macular edema (CME) following cataract surgery, also known as Irvine-Gass syndrome, is a common cause of visual impairment following cataract surgery, with or without intraocular lens implant. It was first reported in 1953, by A. Ray Irvine J, and then enlightened with use of fluorescence angiography by J Donald M. Gass, in 1969 [46].

Pathogenesis: There are numerous factors involved in the pathophysiology of CME. These factors include inflammation, vitreous traction, vascular instability, and light toxicity. Of all these factors, inflammation appears to be the core. Surgical manipulation triggers intracellular inflammation and releases inflammatory mediators such as prostaglandins; cytokines diffuse posteriorly into the vitreous causes breaking down the blood-aqueous and blood-retinal barriers, which leads to increased vascular permeability, accumulation of transudates in outer plexiform layer, inner nuclear layer of the retina, and microcysts coalesce to form a larger intraretinal cyst [47].

Incidence: Although CME following cataract surgery is documented to be the cause of decreased vision following surgery, the incidence of CME remains variable [48]. It is highly dependent on diagnostic criteria; earlier studies are either based on clinical findings with visual impairment or based on fluorescein angiography. However,

recent research is more dependent on optical coherence tomography (OCT). Based on OCT findings, CME occurs in up to 50% of patients at 4–8 weeks postoperatively and is found in less than 3% of patients based on clinical findings along with visual impairment [48, 49].

Diagnosis*:* OCT is superior in looking at the retinal morphology and macular thickness. The test is noninvasive and can be repeated at any time, which is helpful in monitoring patients' response to treatment [50–52]. Fluorescein angiography (FA) is semi-invasive procedure, helpful to roll out other associated causes of CME for example diabetic retinopathy (DR) and retinal vein occlusion. Findings of FA include perifoveal capillary leakage giving the classic "petaloid" appearance and/or capillary dropout. Optical coherence tomography angiography (OCTA) is a noninvasive imaging modality that helps to visualize retinal vasculature and evaluation in retina perfusion [53] (**Figure 5**).

Risk factors: The predisposing risk factors include intraoperative complications (e.g., posterior capsule rupture, vitreous loss, iris trauma, and vitreous traction at the wound), previous surgical procedures (e.g., PPV and penetrating keratoplasty), preexisting ocular conditions (e.g., epiretinal membrane, retinal vein occlusion,

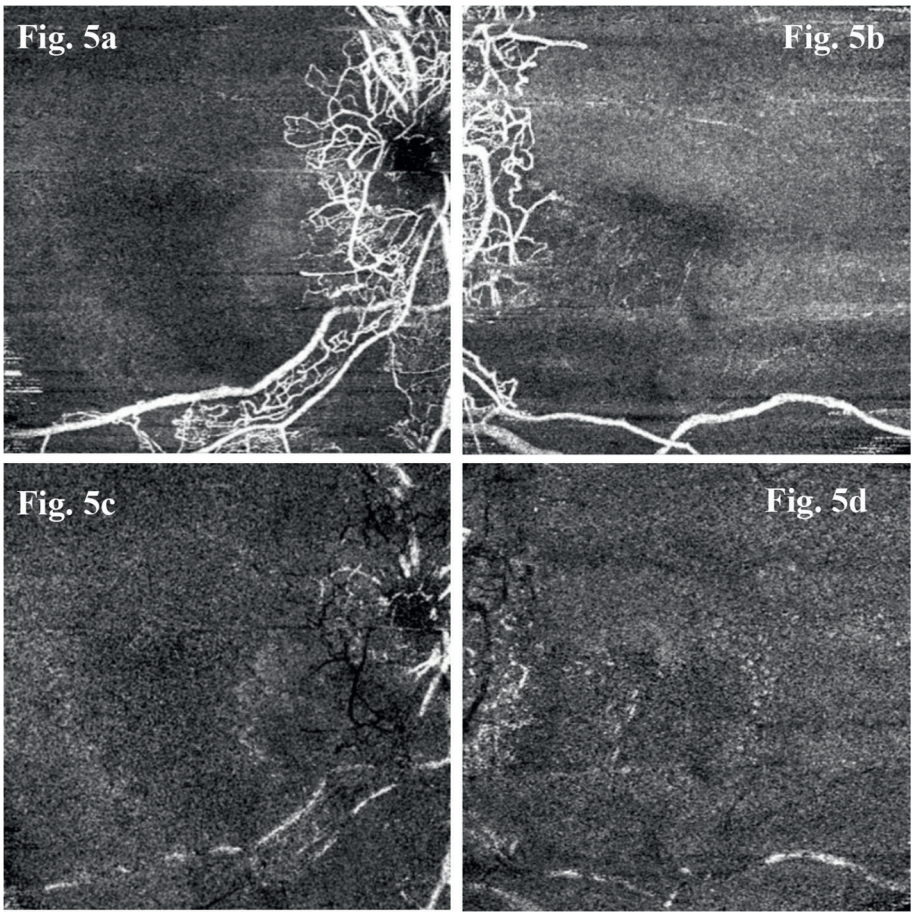

Figure 5.
Macula OCTA showing profound macular capillary dropout in both the superficial (a,b) and deep (c,d) retinal capillary plexus in both eyes (courtesy of Dr. M. Al-Abri).

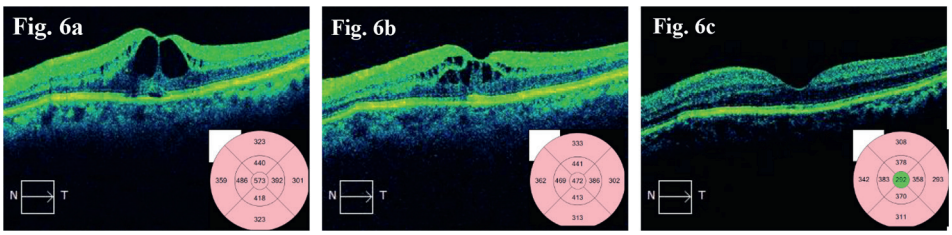

Figure 6.
OCT of the left eye with CME 4-week post-cataract surgery [BCVA 0.3] (a) and follow-up OCTs showing improvement of CME after treatment with topical nepafenac 0.1% TID for 3 and 6 months [BCVA 0.5] (c,d) (courtesy of Dr. M. Al-Abri).

uveitis, ocular hypertension and use of prostaglandin, and dry ARM), and systemic comorbidities (e.g., diabetic mellitus, hypertension, and hyperlipidemia) [54–57].

Management: CME following cataract surgery is self-limiting, where spontaneous resolution occurs within 3–6 months. Therefore, three-month observation period is recommended [56].

Although there is no high level of evidence-based guidelines for the treatment of CME following cataract surgery, most surgeons prefer to start with prophylactic topical non-steroidal antiinflammatory drugs (NSAIDs) combined with corticosteroid drops for high-risk patients [57].

NSAIDs are usually administered topically for 3–4 months and on a needed basis [58]. Previous studies concluded that a combination therapy of topical NSAIDs with corticosteroid drops is more effective than monotherapy post-operative period [59] (**Figure 6**).

Unlike NSAIDs, corticosteroids act on both cyclooxygenase and lipooxygenase pathways; hence, it is presumed to be superior to NSAIDs in controlling postoperative inflammation. Corticosteroids can be used in different ways: topical, periocular, or intravitreal. Intravitreal triamcinolone and dexamethasone implant (OZURDEX®; Abbvie) have been reported to be effective in the treatment of CME [60].

Antivascular endothelial growth factor (Anti-VEGF) plays a major role in inhibition of angiogenesis, inflammation, and capillary permeability. It is used as the first line in treating CME secondary to various vascular pathologies (e.g., diabetic macular edema, retinal vein occlusion, and choroidal neovascular membranes) that are well-established. However, its role in the treatment of CME following cataract surgery remains unclear [56].

7. Progression of diabetic retinopathy after cataract surgery

Diabetes mellitus (DM) is a known risk factor for developing early cataract, including younger age groups. Factors that may influence the progression of diabetic retinopathy (DR) and development of diabetic macular edema (DME) after cataract surgery include the following: the type and duration of diabetes, high level of glycated hemoglobin (HbA1c), the stage of retinopathy, pre-existing DME, and associated systemic diseases (e.g., hypertension and hyperlipidemia) [61].

The association between cataract surgery and progression of diabetic retinopathy has been highlighted by many authors [57, 62]. Some authors have reported an increase in the risk of progression after cataract surgery, whereas others concluded no

significant difference and the progression of diabetic retinopathy (DR) is part of the natural course of the disease [63–65].

Evaluation and management: Surgery is the ultimate treatment for cataract. Planning early cataract surgery before progression of cataract further may limit retina visualization and optimization of the DR and/or DME treatment is important for continuity of care and better postoperative results. The advances in surgical techniques and pre- and postoperative diagnostic and pharmacological management of DR and/or DME have made cataract surgery safer and resulted in better outcomes, particularly for people with diabetes [66].

Prior to cataract surgery, patients with or without DR must undergo thorough eye evaluation, including dilated fundus examination to identify the level of DR and roll-out presence of DME.

Thereafter to address the need for any treatment of DR and/or DME prior to cataract surgery. Optimal management of DR and/or DME prior to cataract surgery contributes to better visual outcome and may reduce the progression of DR and/or DME after cataract surgery [65] (**Figure 7**).

It has been agreed upon that pre-excising DME should be optimally treated prior cataract surgery and careful monitoring postoperatively is recommended. The treatment options for pre-existing DME include the following: intravitreal anti-VEGF, intravitreal steroids injection or dexamethasone implants, and macular laser in particular for non-centrally involved DME. Each of those options have variable benefits and potential risks, and, therefore, the optimal choice for such treatment options is to be individualized and tailored based on the characteristics of the condition [67].

Patients with pre-existing active proliferative diabetic retinopathy (PDR) are more likely to progress further and might be complicated by vitreous hemorrhage and/or diabetic tractional retinal detachment. Therefore, panretinal photocoagulation (PRP) should be considered preoperatively [68]. Moreover, the use of anti-VEGF for PDR is emerging. In a recent systemic review with network meta-analysis (MMA) of randomized clinical trials (RCTs) comparing PRP versus anti-VEGF treatment alone or in

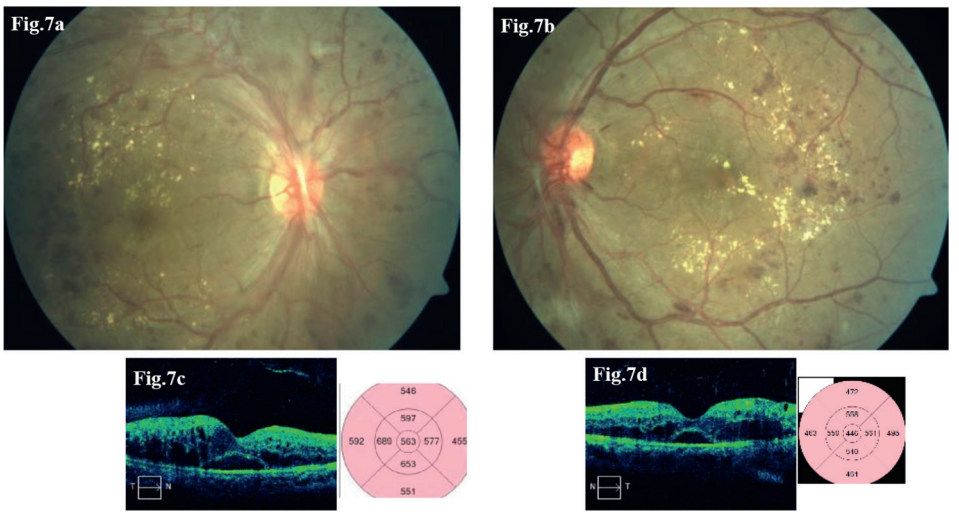

Figure 7.
Fundus photos and macula OCT showing PDR (a, b) and center-involved diabetic macular edema (c,d) in both eyes (courtesy of Dr. M. Al-Abri).

combination with PRP for PDR, it has been concluded that there is limited evidence of comparable efficacy in terms of neovascularization regression between PRP for PDR and anti-VEGF therapy alone or in combination with PRP for PDR. However, better visual outcomes were associated with anti-VEGF use [69].

8. Conclusion

In this chapter, we have discussed the most important aspects of posterior segment complications and challenges that might be encountered in the setting of cataract surgery. Though modern cataract surgery has become reasonably safe and successful as a result of the advancement in ophthalmic technology, surgical skills, and training, yet such complications might occur and, therefore, the primary surgeons and patients should be fully aware of such potential challenges and to adopt safe and evidence-based approach to overcome such challenges, which ultimately will improve the outcomes.

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