

Posterior segment complications following glaucoma surgeries

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The present review describes the posterior segment complications following surgical management of glaucoma. Although the majority of glaucoma cases are managed medically, still a large number of patients may require surgery. Moreover, with the advent of newer surgical techniques and adjuncts, encountering retinal complications post-surgery is not uncommon. The incidence, signs, management, and prognosis of common complications such as anesthesia-related retinal toxicity, vitreous loss, suprachoroidal hemorrhage, serous choroidal detachment, hypotonic maculopathy, vitreous hemorrhage, retinal detachment and endophthalmitis will be discussed in detail. Awareness of these complications is necessary as their proper and timely management can save vision in an already compromised eye.

Key words: Complications, glaucoma, posterior, segment, surgeries

Glaucoma is one of the leading causes of nonreversible blindness in the world.^[1] Though with the advent of new anti-glaucoma medications and laser-based therapies, a majority of cases may be controlled medically, still a large number of patients require filtration surgery to meet target intraocular pressure (IOP). This is especially true for secondary glaucoma, which is often uncontrolled despite maximally tolerable medication.^[1]

With newer surgeries and wide usage of surgical adjuncts, a variety of conjunctival, corneal, lenticular, and retinal complications have come up, which are often visually debilitating to an already compromised visual system. This is specifically true for retinal complications, whose management requires a procedure that can lead to failure of the filtration surgery itself, thus severely affecting visual outcomes. In this article, we review the posterior segment complications, their management, and prognosis.

Anesthesia

Glaucoma surgeries can be done under topical anesthesia with or without intracameral anesthesia, regional anesthesia and general anesthesia. There is no documented case of retinal complication of topical anesthesia but intracameral anesthesia is known to have retinal toxicity. Both lignocaine and bupivacaine have proven retinal toxicity. Hoffman *et al.* and Chia *et al.* have reported cases of transient loss of vision after intracameral lignocaine.^[2,3] Retrobulbar and peribulbar injections can have procedure-related complications such as globe perforation and subretinal bleed. Inadvertent injection of anesthetic agents in vitreous can cause retinal toxicity but

due to the accidental nature of such occurrences, determining exact volume of drug injected and vitreous concentration is difficult and not available in literature. However, in animal studies using rabbit eyes 0.2 mL of lignocaine (2%) and bupivacaine (0.75%) have been found to induce reversible ERG changes.^[4]

Vitreous loss

It is a rare complication of trabeculectomy especially in phakic patients with intact zonules. Possible risk factors include increased venous pressure due to arteriovenous fistulas, Valsalva, exophthalmos, high myopia, aphakia, subluxated lens, excessive peribulbar or retrobulbar block or hematoma, tight eyelids, and so on. Direct trauma to zonules during iridectomy can lead to disruption of the anterior hyaloid phase. Vitreous loss/prolapse may lead to corneal edema, uveitis, retinal detachment, cystoid macular edema, vitreous fibrosis, and so on.^[5] Whenever there is vitreous loss, effort should be made to identify the cause and intraoperative suprachoroidal hemorrhage should be ruled out.

To prevent vitreous prolapse, preoperative IOP should be lowered by using oral acetazolamide or intravenous mannitol, and undue pressure from speculum or excessive block should be avoided. The patient should be made to lie comfortably and any history of chronic obstructive pulmonary disease (COPD)/benign prostatic hyperplasia (BPH) should be ruled out. One should avoid using the vitreous cutter in

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phakic patients as it can cause damage to lens. Instead a cotton wick may be applied to gently prolapse the vitreous then cut it. Vitreous tends to fall back and can further be pushed back with a cohesive viscoelastic.^[6] It is better to keep scleral flap during trabeculectomy sutured to avoid any leak and instruct patient not to rub his/her eyes.

Suprachoroidal hemorrhage

Filtration surgeries are one of the most common ocular surgery to be associated with this complication. It occurs due to high preoperative IOP which drops suddenly. This is a potentially vision-threatening complication. It is a collection of blood in suprachoroidal space due to rupture of long or short posterior ciliary artery. It can occur either intraoperatively, known as expulsive choroidal hemorrhage or in the postoperative period, known as delayed or postoperative suprachoroidal hemorrhage (SCH). Appositional SCH also is known as kissing choroidal can occur after glaucoma surgery. The patient presents with intense pain and decreased vision. The incidence of SCH is very low, varying from 0.7 to 2% in different reports.^[7-9] Incidence is however higher among aphakes, pseudophakes, and high myopes. Vaziri *et al.* reported that it is almost twice more likely to occur in patients with tube shunt surgeries than with trabeculectomy.^[9] Other risk factors include advanced age, diabetes mellitus, high preoperative IOP, low postoperative IOP, use of systemic anticoagulants, hypertension, prior intraocular surgery, ischemic heart disease, and pulmonary disease.^[10,11]

Signs of intraoperative SCH include increased IOP, change in red reflex, and vitreous prolapse. If an intraoperative SCH is suspected, prompt closure of the surgical site is warranted. Increased IOP is managed with topical and oral IOP lowering medications. Small bleeds can be observed and good recovery is expected. Ultrasonography (USG) is helpful in localization and marking the extent of expulsive hemorrhage. It can also help in assessing for clot lysis during follow-up, evident by a decrease in internal reflectivity of choroidal detachment (CD), and mobility of these opacities during dynamic examination. This is helpful in deciding the time for surgical intervention. Drainage may be required in cases with high IOP, intolerable pain, flat anterior chamber, retinal detachment, deteriorating vision, and central retinal apposition, and so on. Clot liquefaction on USG is noted to be around 6 to 25 days (mean-14 days). Intervention is recommended when liquefaction is nearing completion. Hence, these surgeries are usually performed after 10 to 14 days so that the blood in suprachoroidal space is easily drained through sclerotomies and inadvertent probing of suprachoroidal space for residual clots is avoided [Fig. 1]. The surgical approach may be either via external drainage alone through sclerotomy or vitreoretinal surgery along with external drainage. In the former, drainage sclerotomies are created in the quadrant(s) of the involved SCH [Fig. 2]. The IOP is then maintained by continuously injecting a vitreous substitute into the globe. These methods are ideally suited for management of SCH in absence of vitreoretinal traction and when no retinal detachment exists. In presence of retinal detachment, vitreoretinal traction, vitreous hemorrhage, and/or dislocated lens fragments, vitreoretinal surgery at the time of the SCH drainage procedure is usually advisable. Surgical intervention with trans-scleral drainage of hemorrhage along with vitrectomy and silicone oil tamponade can be used to manage patients with massive suprachoroidal hemorrhage. Despite

timely management, outcome in massive suprachoroidal hemorrhage is guarded.^[12]

Serous choroidal effusion

Serous choroidal effusion is composed of transudate fluid that accumulates in the suprachoroidal space due to decreased intramural pressure across capillaries. This may occur both during and after the surgery. They are usually painless and may progress over days which differentiates them from SCH. It can be asymptomatic or can cause significant visual loss. On fundus examination, they can be seen as smooth lobes of elevated retina and choroid whose extent is limited by vortex veins. Incidence after trabeculectomy varies from 7.9 to 18% according to various reports.^[13,14] Incidence with glaucoma valves varies between 11.7 to 15%.^[15,16] Risk factors include nanophthalmos, arteriovenous shunts, Sturge-Weber syndrome, idiopathic high episcleral venous pressure, choroidal hemangiomas, and prolonged surgical time.^[17] In most cases, they can be simply observed with the administration of topical steroids and cycloplegics. In cases with massive effusion surgical drainage may be needed. The visual outcome is usually good.

Hypotonic maculopathy

Ocular hypotony is low IOP (<6 mmHg) leading to functional and structural changes in the eye. Its occurrence has increased with the increased use of antimetabolites in glaucoma surgeries with incidence being as high as 20%.^[18] Risk factors include high myopia, male gender, young age and primary filtering surgery with antimetabolite adjuncts. The proposed mechanism for the above risk factors is reduced scleral rigidity which results in collapse of scleral wall during hypotony.^[19] Chronic hypotony is defined as IOP less than 6 mmHg for 3 months or more and may lead to permanent decrease in vision.^[20] It can occur due to excessive filtration of aqueous from an external fistula or due to an internal fistula connecting anterior to suprachoroidal space. Ciliary body insufficiency after cyclophotocoagulation and inflammation due to surgery can also be the causes.^[21] Patients with hypotonic maculopathy present with reduced vision, distorted images, and relative hyperopia due to anterior shifting of retina. On examination one can see retinochoroidal folds, tortuous vessels, and optic disc swelling [Fig. 3]. Retinochoroidal folds often radiate outwards from the macula. Increased vascular permeability results in cystoid macular edema and serous retinal detachments.^[22] Optical coherence tomography (OCT) is a valuable tool for detecting subclinical serous detachment and edema.

All patients should undergo preoperative risk counseling. Patients at risk require careful operative and postoperative management. Removable/adjustable sutures should be used during trabeculectomy and they are to be removed gradually to avoid sudden hypotony. Valved glaucoma drainage devices help in avoiding sudden hypotony. A two-stage drainage device implantation can be done in case of non-valved devices. Internal tube ligation bypassing 4-0 or 5-0 nylon prolene suture through lumen of tube and external tube ligation with a releasable knot are other techniques for reducing early post-op hypotony after non-valved devices. The overuse of antimetabolites is to be avoided. It is imperative to rule out any conjunctival buttonholing, tear in scleral flap, frank leak and displacement of tube in case of glaucoma drainage device, at the end of surgeries. In the early post-op period, non-contact IOP measuring devices can be used to monitor hypotony. Treatment of hypotonic

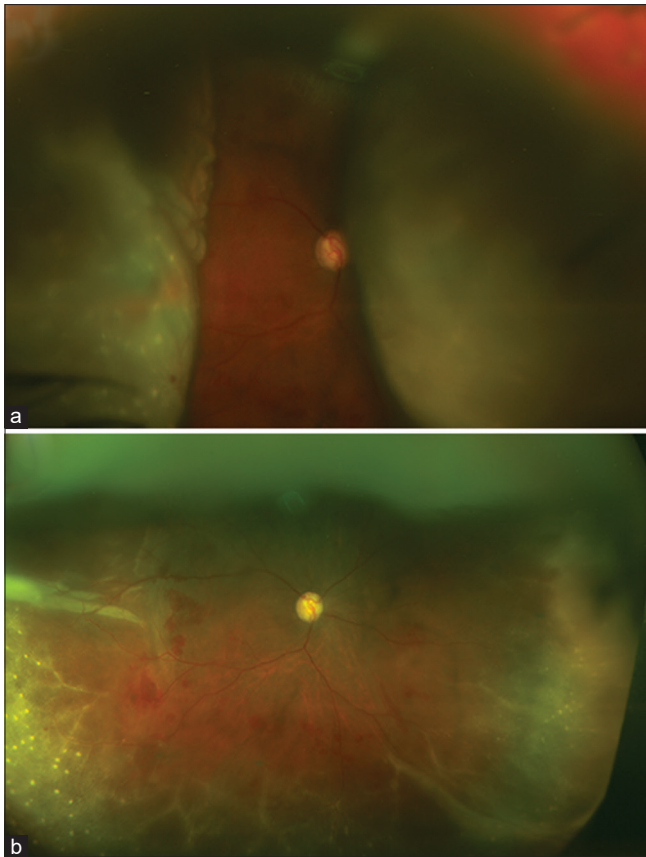


Figure 1: (a) Post-trabeculectomy wide-field fundus photograph showing choroidal mounds suggestive of supra choroidal hemorrhage. (b) Post drainage



Figure 3: Raised disc margins with tortuosity of blood vessels suggesting hypotonic maculopathy

maculopathy requires early identification of the causative factor and its control. In case of a leaking bleb conservative management includes pressure patching, placement of bandage contact lenses, and collagen shields.^[23] Other methods include resuturing of the scleral flap, placement of full-thickness scleral graft, injection of autologous blood in bleb,^[20] application of fibrin glue,^[24] low power argon laser^[25] or cryotherapy^[26] to the leaking bleb, and compression sutures in case of a large leaking

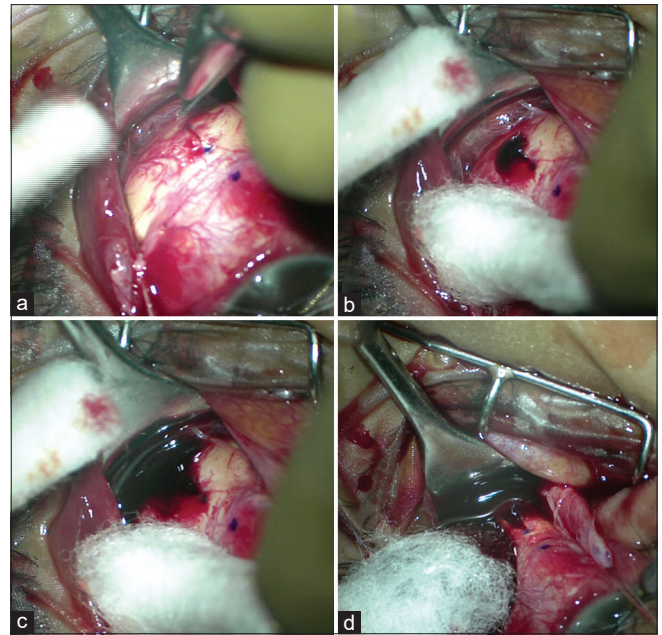


Figure 2: (a) Sclerotomy incision being made. (b) Slow egress of bleed noted. (c) Counter pressure given to enhance drainage. (d) Completion of drainage

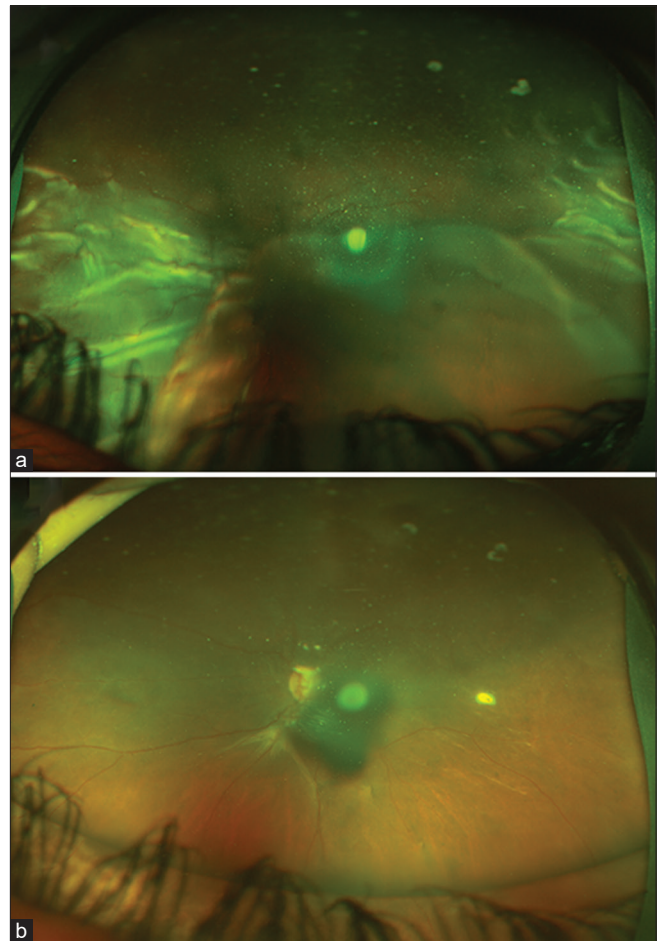


Figure 4: (a) Retinal detachment post-trabeculectomy. (b) Attached retina post-vitreoretinal surgery with silicone oil injection

bleb.^[27] Patients who present with shallow anterior chamber with low IOP in early postoperative period can be managed with injection of air or high viscoelastic agents in anterior chamber. Chambers *et al.* treated 13 eyes with hypotony after glaucoma with injection of perfluoropropane gas and viscoelastic and reported successful outcomes in 12 of them. Friedman *et al.* used intravitreal gas for treatment of hypotony and reported favorable outcomes.^[28] Duker and Schuman showed that long-standing cases and those not responding to above management strategies can undergo vitrectomy with use of perfluorocarbon for few minutes to flatten chorioretinal folds.^[29]

Vitreous hemorrhage

Law *et al.* reported that the incidence of vitreous hemorrhage after aqueous shunt surgical procedure is about 5%.^[7] Source of this bleed can be from a deep scleral suture, a retinal break, breakthrough bleed from a suprachoroidal hemorrhage, or a wound during tube insertion. Blood can also migrate from the anterior chamber especially in the case of aphakia. Systemic risk factors include sickle cell anemia, use of anticoagulant medication, homocystinuria, and so on. Flynn *et al.* reported a case that developed hyphema and intravitreal blood after intrable injection of autologous blood after trabeculectomy.^[30] It may also occur from a coexisting pathology such as diabetic retinopathy and neovascular ARMD.

Management includes prompt identification of cause and appropriate treatment. Rule out any systemic risk factor. Most hemorrhages tend to resolve spontaneously and can simply be observed. In non-resolving cases, coexisting rhegmatogenous retinal detachment, suprachoroidal hemorrhage, and where the bleed is massive enough to itself raise IOP a prompt pars plana vitrectomy with gas tamponade might be required.

Retinal break and detachment

Rhegmatogenous and serous retinal detachments have been reported after glaucoma surgeries. Excessive manipulation, a sudden change in IOP and vitreous loss can result in tears leading to rhegmatogenous retinal detachment after glaucoma surgeries [Fig. 4]. The incidence of rhegmatogenous retinal detachment after molteno implants has been reported to be about 5%. In about 70% of cases patients were presented within 4 months of surgery with retinal tear and dialysis being the most common cause. Around 41% patients developed proliferative vitreoretinopathy. Presence of predisposing lesions such as lattice degeneration, previous history of detachment in same or fellow eye, trauma and uveitis lead to an increased risk of detachment.

Retinal detachment in such patients presents with a unique set of problems. Tamponade with silicone oil can lead to worsening of IOP due to the failure of glaucoma surgery. Oil can come in anterior chamber further compromising trabecular meshwork in long-standing cases. Subconjunctival migration of oil can also be seen. The use of expansile gases can make management of IOP very difficult in immediate post-op period. PPV with fluid gas exchange is more successful in achieving anatomical success than scleral buckling and pneumatic retinopexy in such patients, however, the functional outcome remains poor due to pre-existing glaucoma and difficulty in managing IOP in postoperative period.^[31] All efforts should be made not to damage the bleb and compromise as less conjunctiva as possible. Displacement or extrusion of tube or plate should be looked for in case of an existing Glaucoma

drainage device (GDD). Dellacroce *et al.* reported three cases with serous retinal detachment following glaucoma surgery which subsided spontaneously with oral and topical steroids.^[32] Lavin *et al.* reported six patients who developed serous retinal detachment with choroidal effusion in postoperative period out of which 6 resolved with conservative treatment and one underwent vitrectomy.

Endophthalmitis

Bleb related infections range from an isolated infection of bleb known as blebitis, to a potentially blinding bleb associated endophthalmitis (BAE). The presence of inflammatory cells in vitreous helps in clinching the diagnosis of BAE in a patient with bleb infection. The incidence of endophthalmitis after trabeculectomy is around 1 to 2% which is higher than other causes of exogenous endophthalmitis. As with any intraocular surgery, the infection can occur in early postoperative period but most of the cases present months to years after surgery with a higher rate in children mostly due to a presence of long-standing thin filtering bleb. Cases presenting within a month of surgery are classified as early-onset and those presenting after a month are classified as late-onset BAE.^[33] *Staphylococcus epidermidis* is the most common organism responsible in blebitis and is thought to be secondary to intraoperative contamination whereas *Streptococcus* and gram-negative bacteria's such as *H. influenzae* are more common in cases of BAE with infection thought to arise from transconjunctival spread of bacteria through a bleb leak or exposed tube.^[34] Latter organisms are more virulent and are associated with poorer prognosis.^[35,36] The presence of thin cystic bleb and use of antimetabolites intraoperatively are major risk factors for blebitis and endophthalmitis. Wallin *et al.* reported incidence with usage of MMC to be 2.6%.^[37] Other risk factors include inferiorly or nasally located bleb, myopia, bleb with leaks, additional surgeries, diabetes mellitus and presence of concurrent upper respiratory tract infections and so on.^[38] Common symptoms and signs include pain, reduced vision, floaters, foreign body sensation, tearing, white milky looking bleb, purulent discharge, etc. The appearance of bleb is typically called "white on red", with white opaque bleb surrounded by red hyperemic conjunctiva. All the patients should be made aware of these and instructed to consult an ophthalmologist as early as possible. Morbidity of these infections is very high and almost 1/3 of the patients treated on intensive medical therapy end up with PL (perception of light) negative status.^[39]

To prevent it, any leaking bleb should be repaired. Any patient with conjunctival and scleral erosion with a tube of a drainage device should undergo patch graft with sclera, conjunctiva or other appropriate material. While EVS study has given us a guideline for post-cataract surgery endophthalmitis, no such guidelines exist for bleb associated endophthalmitis.^[40] While most evidence suggests that blebitis can be managed successfully on an out-patient basis, endophthalmitis usually requires much more intensive management.^[41] In suspected cases, smears and cultures should be obtained from lid conjunctiva and filtering bleb and antibiotic sensitivity should be performed. However, their role remains uncertain because organisms cultured may be different from those in vitreous culture.^[42] Vitreous tap using a 20 gauge cutter and intravitreal injection of ceftazidime (2.25 mg/0.1 mL) and vancomycin (1 mg/0.1 mL) may be considered. However, it has been suggested that prompt vitrectomy and simultaneous

injection of 1/10 dose of intravitreal antibiotics is more appropriate because of high virulence of involved organisms and leads to better visual outcomes.^[43] With recent advancements in instrumentation, it is better to use smaller gauge cutter for vitreous biopsy or vitrectomy. Though EVS study showed no added advantage of systemic antibiotics, these may be added in treatment because of different nature of organisms here. Third generation fluoroquinolones such as ofloxacin and ciprofloxacin can be used owing to their high penetrance in vitreous. Fourth-generation fluoroquinolones such as gatifloxacin have more effect on gram-positive organisms than third generation and equal effect on gram-negative organisms, hence they can be a better option.^[39] Another consideration is the use of intravitreal dexamethasone which has shown to reduce intraocular inflammation and improve short-term visual outcomes.^[44,45] Fortified topical antibiotics should also be run hourly. Rarely, patients who have undergone diode laser cytophotocoagulation for refractory glaucoma may present with panophthalmitis.^[46]

Sympathetic ophthalmia

Sympathetic ophthalmia is a rare but devastating complication of any intraocular surgery. The exact nature of etiology is still unknown but it is thought to be autoimmune in nature with CD-4 cells playing a key role.^[47] We propose a possible association between ciliary body stem/progenitor cells and sympathetic ophthalmia as its incidence matches the number of ciliary body epithelial cells with progenitor properties (0.2% to 2%).^[48] Though it may occur after any intraocular surgery, it is more common in surgeries in which there is intentional or accidental incarceration of uveal tissue.^[49] An incidence of approximately 0.08% has been estimated after glaucoma surgery. It can present anytime between 2 weeks to years but around 80% of cases are seen within 3 months of causative trauma.^[50] Symptoms include blurring of vision, photophobia, and redness first in the exciting eye and then in the sympathizing eye.^[42] It is a type of granulomatous uveitis and signs include keratic precipitates, iris nodules, and cells and flare in anterior chamber.^[51] Dalen-Fuchs nodules can be seen as yellowish-white spots in peripheral retina. Sympathetic ophthalmia is known to occur even after ciliary body destruction with trans scleral laser and cryotherapy.

Systemic immunosuppression is the mainstay of treatment with corticosteroids being the first line of therapy which may be administered topically, by sub-Tenon or systemic routes.^[51] Treatment is started with high dose oral methylprednisolone at about 1–2 mg/kg per day and then slowly tapered with close monitoring. The dose is to be brought at a starting level whenever there is worsening of eye. Topical corticosteroids and cycloplegics are added to prevent synechiae formation. Patients who are steroid-resistant or have intolerable side effects it can be maintained on immunosuppressives.^[51]

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Conflicts of interest

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