Glaucoma and elevated IOP can be challenging to manage, particularly when these conditions occur secondary to vitreoretinal procedures. Key considerations for successfully managing glaucoma or high IOP in this setting include the preexistence of either condition, the type of vitreoretinal procedure, the timing of IOP elevation, and close collaboration between the vitreoretinal surgeon and other care providers. The choice of treatment should be based on awareness of the pathophysiology, the degree of IOP elevation, and the risk or severity of glaucomatous damage.

**Scleral Buckle**

The incidence of angle-closure glaucoma (ACG) following scleral buckle surgery ranges from 1.4% to 4.4%, and IOP elevation has occurred in up to 28.8% of cases. The pathophysiology of ACG in these cases involves compression of the vortex veins by the scleral buckle. This compression impedes venous drainage and results in congestion and anterior rotation of the ciliary body, with subsequent anterior shift of the lens-iris diaphragm.

Risk factors for ACG after scleral buckling include preexisting narrow angles, high myopia, and placement of the encircling band anterior to the eye's equator.

**Management.** Most cases of angle closure after scleral buckle resolve without intervention, as congestion of the ciliary body decreases. Cycloplegia can be helpful to open the angle because it relaxes the ciliary muscle and promotes posterior movement of the lens-iris diaphragm. Note that miotics can worsen angle closure.

Aqueous suppressants are effective for lowering IOP.

Although laser iridotomy is not indicated in these cases because the angle closure is not caused by pupillary block, laser iridoplasty might help to open the angle. In refractory cases, trabeculectomy or tube shunt surgery may be required; however, the conjunctival recession and scarring that typically accompany scleral buckling may preclude trabeculectomy. Tube shunts have high rates of success, and their endplates can be sutured directly to an existing scleral buckle.

Another approach to consider is the modified Schocket procedure, in which silicone tubing is used to shunt aqueous from the anterior chamber to the fibrous capsule of an existing scleral buckle.

**Pre-op advice.** For eyes at high risk of IOP elevation after scleral buckle, such as those with advanced glaucomatous damage and/or synechial angle closure, it may be prudent to consider placing a tube shunt during the primary buckle surgery.

**Silicone Oil**

Silicone oil is often used when repairing complex retinal detachments. The incidence of elevated IOP after injection of silicone oil is approximately 2% at six months and 56% at eight months.

There are various mechanisms by which silicone oil can elevate IOP.

Therefore, gonioscopy is required to be able to distinguish between a closed angle (in which the glaucoma or elevated IOP results from pupillary block, synechial angle closure, intraoperative overfill of silicone oil, or anterior segment neovascularization) and an open angle (in which glaucoma or elevated IOP is due to migrated emulsified oil or an idiopathic cause). The management approach depends on the mechanism.

**Management.** IOP elevation on
post-op day 1 may signify overfill of silicone oil, necessitating partial aspiration of oil by the vitreoretinal surgeon, via the pars plana. Most cases of high IOP related to silicone oil can be managed with aqueous suppressants.

For cases that do not respond to aqueous suppressants, oil removal can be considered if the risk of retinal re-detachment is low. If re-detachment is a concern, placement of a tube shunt can be considered. Given that oil is buoyant in aqueous, the tube should be positioned inferiorly within the anterior chamber (especially in aphakic eyes) to avoid escape of oil through the tube, which could lead to tube obstruction and/or subconjunctival inflammation. Another surgical option is cyclophotocoagulation, but repeated treatments may be needed in an oil-filled eye.

Pupillary block caused by silicone oil requires inferior laser iridotomy or surgical iridectomy. An important and easy-to-miss clinical phenomenon is the aphakic eye without a patent iridotomy/iridectomy, in which silicone oil prolapses forward and fills the anterior chamber (Fig. 1). High IOP is a result of pupillary block and direct obstruction of the trabecular meshwork. In such cases, the anterior chamber is deep, and the angle appears open on gonioscopy, with angle structures being visible. High-level vigilance is needed to recognize the often subtle sheen of silicone oil on the iris surface of these eyes.

Prophylactic inferior surgical iridotomies are often performed at the time of oil injection to prevent pupillary block in aphakic eyes; however, iridotomies are reported to close in 11% to 32% of eyes due to scarring and/or obstruction by capsular remnants. An in-office laser iridotomy or a surgical iridectomy allows aqueous to regain access to the anterior chamber and enables return of the oil to the posterior segment.

A note of caution. Silicone oil is inflammatory; hence, long-term treatment with low-dose steroids is crucial to prevent scarring of an iridotomy or iridectomy.

Intravitreal Injections

The incidence of elevated IOP after intravitreal steroid injections ranges from 29% to 40%. These injections increase resistance to aqueous outflow by altering the anatomic structure of the trabecular meshwork through the extracellular matrix; in turn, phagocytosis is inhibited in the meshwork, causing increased debris buildup and physical obstruction of the outflow system. Although some degree of acute IOP elevation is expected because the injection adds volume to the eye, the incidence of sustained IOP elevation after injection of anti-VEGF agents can be as high as 15%.

The proposed mechanisms for sustained IOP elevation include chronic inflammation, trabecular meshwork obstruction, and damage to the trabecular meshwork by the anti-VEGF agent or by particles from the compounding or packaging process. The risk of chronic or long-term IOP elevation appears to be higher for patients with preexisting glaucoma, those with a history of steroid-induced IOP elevation, and those who receive frequent injections.

Management. Topical aqueous suppressants are the first-line treatment and will adequately control IOP in most cases. If the response to aqueous suppressants is inadequate, incisional glaucoma surgery may be required.

Panretinal Photocoagulation

The reported rates of transient IOP elevation after panretinal photocoagulation vary widely, from 34% to 97% of cases, and the pressure usually rises immediately after the procedure. A high amount of laser energy may cause elevated IOP and ciliary body congestion, raising the risk of angle closure. When the angle is open, there can be various reasons for IOP elevation, including blood-retinal barrier breakdown causing fluid movement from the choroid into the vitreous; ciliary body congestion causing decreased uveoscleral outflow; and episcleral venous compression by a contact lens, leading to blockage of aqueous outflow.
Management. IOP elevation after panretinal photocoagulation usually is self-limited and resolves spontaneously. However, cycloplegia and aqueous suppression may be needed in refractory cases.

Takeaway Messages
After any vitreoretinal procedure, early recognition and prompt treatment of IOP elevation, with or without glaucoma, are essential to reduce the risk of glaucomatous damage and visual decline. Gonioscopy is a crucial tool to identify the underlying mechanism responsible for the IOP rise. The choice of treatment depends on the pathophysiology, degree of IOP elevation, and risk or severity of glaucomatous damage. Management options include medical therapy, laser treatment, and surgical intervention. A multidisciplinary approach that involves vitreoretinal specialists is vital to optimize outcomes for patients.

1 Kawana K et al. Ophthalmology. 2006;113(1):36-41.

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