

Diagnosis and Management of Choroidal Effusions

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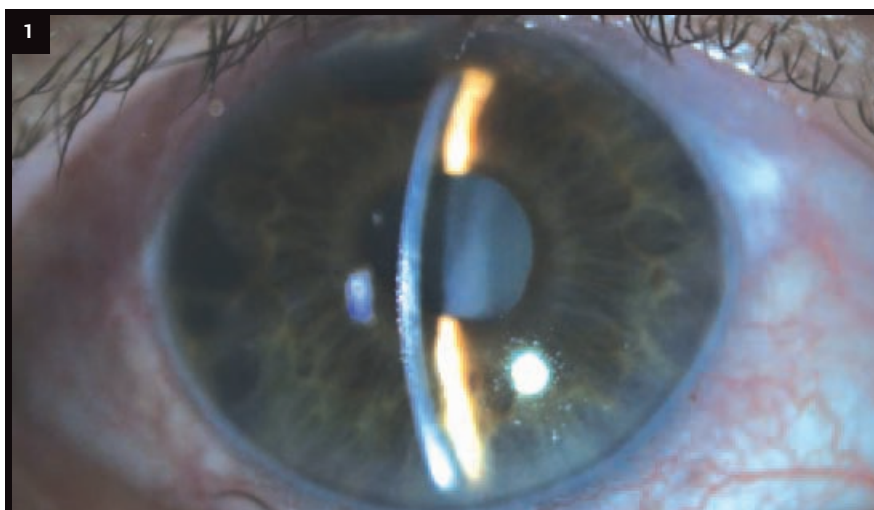
Choroidal effusion—an abnormal accumulation of fluid in the suprachoroidal space—is a common complication of glaucoma surgery. However, this may arise from other intraocular surgeries and a number of conditions, including inflammatory and infectious diseases, trauma, neoplasms, drug reactions, and venous congestion. Idiopathic causes fall under the umbrella of uveal effusion syndrome, a rare condition usually considered a diagnosis of exclusion.

A note on nomenclature: Various terms are used interchangeably in the literature to describe an abnormal collection of fluid in the suprachoroidal space, including ciliochoroidal effusion, ciliochoroidal detachment, and choroidal detachment. For purposes of clarity, this review uses the term choroidal effusion(s).

Hypotony is the main cause of fluid accumulation in the suprachoroidal space after glaucoma surgery, although inflammation and venous congestion may also be contributing factors. Choroidal effusion further exacerbates hypotony by reducing aqueous humor production and, possibly, by increasing uveoscleral outflow. This review will discuss various aspects of choroidal effusions, with an emphasis on clinical presentation, management, and prevention.

Pathophysiology

In a normal eye, the suprachoroidal space is essentially nonexistent because



1 **CLINICAL PRESENTATION.** Shallow anterior chamber in the presence of an overfiltering bleb and choroidal effusions after glaucoma filtration surgery.

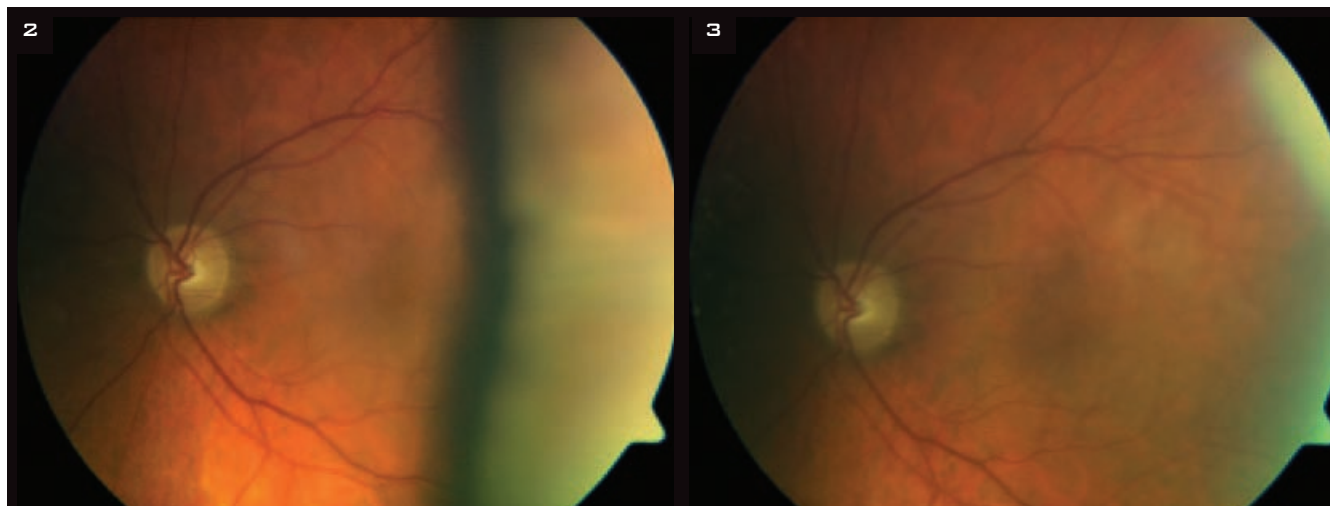
of close apposition of the choroid to the sclera. In pathologic conditions that disrupt the normal ocular fluid dynamics and hydrostatic and oncotic pressure gradients, fluid accumulates in this potential space.

Serous choroidal effusions involve transudation of serum into the suprachoroidal space, whereas hemorrhagic choroidal effusions involve blood accumulation from rupture of choroidal vessels. Choroidal effusions represent tissue edema and are best understood through Starling's law, which elucidates the balance of hydrostatic and osmotic gradients between the choroidal capillaries and interstitial space of the eye. With this approach, the intraocular pressure (IOP) can be thought of as the hydrostatic pressure in the interstitial space. Thus, any

process that shifts flow from the choroidal capillaries into the interstitium may lead to an effusion. A decrease in IOP allows fluid to accumulate in the interstitial spaces, while inflammation increases the permeability of the choroidal capillaries. These mechanisms suggest that choroidal effusions form either as a result of increased transudation through the choroidal capillary walls or from a drop in IOP caused by an increase in uveoscleral outflow of aqueous humor.¹ Choroidal effusions may also be a precursor of suprachoroidal hemorrhage.

Risk Factors

In general, any condition that results in a low IOP can be considered a risk for choroidal effusion development. The most common risk factor for cho-



RESPONSE TO DRUGS. (2) Presence of choroidal effusion on dilated fundus examination and (3) subsequent resolution with medical therapy, using topical cycloplegia and steroids.

roidal effusions is glaucoma surgery in the setting of overfiltration or a bleb leak (Fig. 1). Other risk factors are as follows:

Medications. Both perioperative antimetabolites and aqueous suppressants have been identified as potential risk factors for choroidal effusions. The use of antimetabolites—particularly mitomycin C (MMC)—during trabeculectomy is a significant risk factor that may lead to prolonged hypotony and, thus, to persistent choroidal effusion.

Patients treated with aqueous suppressants, including timolol or dorzolamide, after trabeculectomy or glaucoma drainage device implantation may be at increased risk for developing hypotony and choroidal effusions postoperatively.² Late choroidal effusions have also been reported with latanoprost use in eyes with prior cataract extraction.

Various other systemic medications, including sulfonamides, tetracycline, diuretics, and selective serotonin reuptake inhibitors, have also been reported to be associated with choroidal effusions and secondary angle closure. In these cases, discontinuing the medication typically leads to resolution of the effusion.

Iridotomy. Sakai and colleagues³ reported an increased risk of subclinical choroidal effusions after laser iridotomy. In their study of 38 eyes,

they reported a 52 percent rate of choroidal effusion in eyes receiving treatment with the argon laser, versus a 12 percent rate in eyes receiving treatment with a combination of argon and Nd:YAG lasers. This difference was attributed to the higher total energy delivery in the argon laser group than in the combined laser group.

Preexisting conditions. There is evidence for an increased association of choroidal effusion with nanophthalmos and with Sturge-Weber syndrome. In patients with Sturge-Weber syndrome, the risk is even higher in the presence of choroidal hemangiomas. Patients with nanophthalmos or Sturge-Weber syndrome should be informed about their risk of choroidal effusions, and preventive measures should be taken during any intraocular surgery to minimize potential complications.

Clinical Features

Symptoms of choroidal effusions vary among individuals.

Serous. Small, peripheral effusions may be asymptomatic, with minimal to no shallowing of the anterior chamber. Large effusions may cause refractive changes from anterior displacement of the lens-iris diaphragm and resultant myopia, or they may cause significant reduction in visual acuity by encroachment into the visual axis. Patients may experience an absolute scotoma at the site of effusion.

Hemorrhagic. Unlike serous choroidal effusions, which typically develop painlessly, hemorrhagic choroidals generally have an abrupt onset with severe pain and marked reduction in visual acuity. When hemorrhagic choroidals are associated with high IOP, hyperosmotic agents and aqueous suppressants are recommended. The visual outcomes and overall prognosis are worse with hemorrhagic choroidals.

Diagnosis

Choroidal effusions are diagnosed clinically and usually appear elevated in a four-lobed presentation because of firm attachments of the choroid to the vortex veins. B-scan echography helps to differentiate choroidal effusions from retinal detachments. On echography, effusions are notable for their anterior angle and extension to the ora serrata. Ultrasonography offers a method for detecting a small accumulation of fluid in the supraciliary-choroidal space not readily apparent on clinical examination.⁴

Low-lying choroidals may be seen with the use of sulfa-derivative medications, such as topiramate. It is important to document choroidal effusions in these settings because the mechanism of secondary angle closure does not involve a pupillary block mechanism, and laser peripheral iridotomy is ineffective in breaking the acute attack.

Treatment

Choroidal effusions after glaucoma surgery are often managed conservatively, and the approach varies depending on underlying etiology.

Close observation. As IOP rises postoperatively, most effusions resolve spontaneously if they are limited in size and duration and do not affect surgical prognosis or visual outcomes.

Medications. In the presence of inflammation, the eye is treated aggressively with topical or oral steroids. In the presence of overfiltration, steroids are tapered quickly or discontinued to promote bleb scarring; cycloplegic agents are used to deepen the anterior chamber by rotating the ciliary body posteriorly (Figs. 2 and 3).

Other measures. Other treatments for an overfiltering bleb include application of a bandage contact lens, injection of a high-density viscoelastic, or use of compression sutures.

Surgical drainage: indications.

These include a flat anterior chamber, decreased vision, long-lasting choroidal effusions, appositional choroidals (due to the potential for retinal adhesion formation and subsequent visual impairment), and suspected suprachoroidal hemorrhage.

Surgical drainage: technique. The location of maximal fluid accumulation should be noted preoperatively to determine the optimal drainage site. After conjunctival peritomy, a 2- to 3-mm radial incision is made in the sclera about 3 to 4 mm posterior to the limbus. The incision is deepened until the suprachoroidal space is entered and fluid is released. (The fluid is clear and yellowish in serous choroidals or dark red with blood clots in hemorrhagic choroidals.) The incision's edges may be pulled apart by forceps or cautery to facilitate fluid egress. The sclerotomy site is left open with closure of overlying conjunctiva. A second sclerotomy may be needed to drain fluid from another quadrant. Throughout the procedure, the eye should be kept pressurized with injection of balanced salt solution, a viscoelastic substance in the anterior chamber, or an anterior chamber maintainer.

WuDunn and colleagues⁵ reported the largest retrospective series, consisting of 63 eyes, to determine the efficacy and safety of surgical drainage of choroidal effusions following glaucoma surgery. A high success rate was reported with significant improvements in visual acuity and hypotony. The authors noted that, in addition to a drainage procedure, other intraoperative measures, such as additional flap sutures and cataract extraction, might have been responsible for some degree of wound healing and resolution of hypotony. Most patients (51 of 63 eyes) underwent a single drainage procedure, while some required multiple procedures. The most common postoperative complication was cataract formation. However, it was unclear whether cataract progression was secondary to the surgical drainage or to the preoperative presence of a flat anterior chamber and hypotony.

Acute Intraoperative Effusions

In some cases, choroidal effusions may occur during intraocular surgery and can precede an expulsive hemorrhage. An early sign of this phenomenon is loss of the normal red reflex. Proper treatment in this scenario involves immediate closure of the incision. Closure is often followed by a rapid rise in IOP to 80 mmHg or greater with a subsequent normalization of IOP within 15 to 30 minutes, leaving a localized area of effusion.⁶

Prevention

The best strategy for handling acute intraoperative choroidal effusions is to prevent them by minimizing hypotony and inflammation intraoperatively and postoperatively.

During surgery. Good scleral flap architecture and thickness, multiple sutures on the flap to regulate aqueous outflow, use of cautery to achieve hemostasis, judicious use of antimetabolites, placement of anterior sclerotomy to avoid the ciliary body, and meticulous closure of the conjunctiva are all important measures to minimize intraoperative bleeding and hypotony during glaucoma surgery.

Drainage devices. For glaucoma drainage devices, one may opt for valved devices. For nonvalved devices, the tube should be ligated, or two-stage surgery may be performed to allow formation of a fibrous capsule around the plate to avoid excessive filtration in the early postoperative period, minimizing hypotony and its related sequelae.

High-risk patients. Prophylactic sclerotomies at the time of glaucoma filtration surgery may be helpful in high-risk cases with known susceptibility to choroidal effusions, such as patients with prior postoperative choroidal effusions, nanophthalmos, or Sturge-Weber syndrome.

Postoperative care. Topical and systemic aqueous suppressants should be discontinued, and early laser suture lysis should be avoided.

Conclusion

Choroidal effusions may result from various etiologies but are most commonly encountered after glaucoma surgery, especially in the setting of hypotony, inflammation, or both. Meticulous surgical steps and preventive measures may help to reduce the risk of choroidal effusions. While most choroidal effusions resolve spontaneously, surgical drainage may be necessary in some cases to restore normal anatomy and visual function.

1 Bellows AR et al. *Ophthalmology*. 1981; 88(11):1107-1115.

2 Callahan C, Ayyala RS. *Ophthalmic Surg Lasers Imaging*. 2003;34(6):467-469.

3 Sakai H et al. *Am J Ophthalmol*. 2003; 136(3):537-538.

4 Banta JT et al. *Am J Ophthalmol*. 2001; 132(1):112-114.

5 WuDunn D et al. *J Glaucoma*. 2005;14(2): 103-108.

6 Maumenee AE, Schwartz MF. *Am J Ophthalmol*. 1985;100(1):147-154.

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