Managing Secondary Angle-closure Glaucomas

Early recognition and treatment of the cause promote resolution.

By Oluwatosin Smith, MD

Unlike primary angle-closure glaucoma, secondary angle-closure glaucoma (SACG) occurs as a sequel to a wide range of ocular and some systemic conditions. Many forms of SACG have been described at length and are well documented, but some new causative entities have been reported. The occurrence of SACG can be broadly divided into disease in the presence (relative or absolute) or absence of pupillary block leading to an elevation in IOP.

PATHOPHYSIOLOGIC CONSIDERATIONS

Pupillary block can be relative, as in phacomorphic glaucoma, where a large or intumescent cataract pushes the iris forward, closing the angle. Pupillary block also occurs in eyes with pseudoexfoliation and weak zonules, ectopia lentis, Marfan syndrome, and microspherophakia, where the anteriorly subluxated lens can narrow or close the angle. If not carefully evaluated, patients may be misdiagnosed with acute primary angle-closure glaucoma instead of acute angle closure secondary to the lens’ subluxation. The cause of this error includes neglected history or signs of ocular trauma.

Absolute pupillary block may result from the formation of 360° of posterior synechiae between the iris and any structure that lies posterior to it (eg, lens, lens capsule, anterior vitreous phase, and rarely, the IOL), thus trapping aqueous in the posterior chamber. This situation can occur in uveitic eyes or those with inflammation after ocular surgery. Aqueous misdirection syndrome that traps fluid in the vitreous cavity...
can also cause the anterior chamber to become shallow and the angle to close.

In the absence of pupillary block, the angle can close due to the formation of a vascular inflammatory or hemorrhagic membrane, leading to peripheral anterior synechiae (Figure 1). Additional causes are epithelial downgrowth, fibrous ingrowth, or the migration of corneal endothelial cells (ie, iridocorneal endothelial syndrome). SACG can also result from the anterior displacement of the lens-iris diaphragm due to swelling and inflammation of the ciliary body, contracting retrorenal tissue (eg, retinopathy of prematurity), or a scleral buckle; anterior rotation of the lens-iris diaphragm, as induced by topiramate; and choroidal effusion or detachment (eg, uveal effusion syndrome, postoperative finding in nanophthalmic eyes).

DIFFERENTIATION, DIAGNOSIS, AND TREATMENT

Properly managing SACG entails determining the cause of the angle closure, which dictates the specific treatment modality to be instituted. Early detection of the underlying pathology and prompt treatment are therefore imperative for a successful outcome. Except when membrane formation disrupts the angle anatomy, treating the cause of SACG generally leads to its resolution. A carefully obtained history—including a review of systems, past ocular history, past medical history, past surgical history, list of medications, and family history—is sometimes required to elucidate the possible causative or inciting agent.

An assessment of the patient’s general appearance and a thorough eye examination to look for signs of prior trauma, evidence of uveitis, a disparity in the anterior chamber depth between eyes, the presence of choroidal effusion, etc., facilitate a proper diagnosis. An initial approach is to determine whether or not the patient is in pupillary block. Absolute pupillary block with iris bombé and peripheral narrowing of the anterior chamber, angle closure, and elevated IOP can easily be remedied by a laser peripheral iridotomy (LPI). In certain instances, however, the development of chronic angle closure will necessitate additional glaucoma surgery.

The presence of a bilateral secondary angle closure raises the possibility of drug-induced SACG or the presence of uveal effusion syndrome. Early identification of either problem can spare the patient unwarranted surgical intervention. The list of drugs that can induce secondary angle closure continues to grow, but the culpability of sulfa derivatives like topiramate has been well documented. Other causative conditions or medications described include Campylobacter jejuni infections, bupropion HCl (Wellbutrin; GlaxoSmithKline), and methazolamide²,³ (Figure 2). Conservative management with IOP-lowering medication, topical or systemic steroids, and cycloplegics should resolve the problem after cessation of the

Figure 2. Secondary angle closure after use of topiramate.
inducing agent or treatment of the possible cause. Miotics such as pilocarpine can exacerbate SACG caused by medication and uveal effusion syndrome with worsening of the angle closure. Peripheral iridotomy or iridoplasty is not warranted in these cases and should be avoided. In certain instances, drainage of ciliochoroidal effusion may be indicated.

By and large, SACG is managed through the treatment of its causative factors after the physician addresses acute elevations of IOP. Depending on the cause, intervention may involve removing a large intumescent or subluxated lens, treating choroidal swelling or effusion and removing the causative agent, or treating chronic uveitis. An LPI will suffice in some cases. In contrast, neovascular glaucoma, endothelial downgrowth, fibrous ingrowth, and iridocorneal endothelial syndrome are a lot harder to manage; distortion of or permanent damage to the anterior chamber angle requires the creation of an alternate drainage pathway, more commonly a glaucoma drainage device.

**IATROGENIC CONSIDERATION**

The advent of Descemet stripping endothelial keratoplasty introduced new sources of SACG. In these cases, pupillary block can be caused by the presence of air anterior to the iris or, more commonly, by the migration of air behind the iris resulting in extensive iridocorneal adhesions. The IOP usually rises during the first few postoperative days. This spike can initially be managed medically, but a cycloplegic agent may also be required, depending on the clinical scenario. In the presence of extensive and persistent iridocorneal adhesion, medical management can fail to remedy the air posterior to the iris, in which case surgical intervention will be required. In cases of pupillary block, an LPI is warranted to break the attack. Regardless, when indicated, surgical intervention requires reformation of the angle, removal of the air, and a surgical peripheral iridotomy if the causative mechanism was pupillary block.

**CONCLUSION**

SACGs are generally easy to treat if the physician recognizes the causative factor or mechanism early. A thorough history and astute examination facilitate proper diagnosis and, thus, decisions on treatment. Correct diagnosis and appropriate treatment choices early on can spare patients unwarranted procedures and potentially save their sight in the long run.

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