Traumatic Angle-closure Glaucoma

A higher index of suspicion for this diagnosis is warranted when the patient has predisposing risk factors for angle closure.

BY JULIA SONG, MD

Traumatic glaucoma occurs after an ocular injury, whether it be blunt or penetrating trauma. Acute blunt trauma can tear the ciliary body and clog the trabecular meshwork with red blood cells, pigment, or debris. Elevated IOP is usually temporary and can be controlled medically. In the long term, blunt trauma can result in angle-recession glaucoma (for more on this topic, read the article by Quang Nguyen, MD, on page 37). Penetrating intraocular trauma can result in inflammation, bleeding, and elevated IOP. This article focuses on the mechanisms of traumatic angle-closure glaucoma (ACG).

CLASSIFICATION

Trauma resulting in ACG can be divided into two categories: (1) secondary ACG with pupillary block and (2) secondary glaucoma without pupillary block. Previous pupillary block, hyperopia, nanophthalmia, cataract, the presence of a phakic IOL, iris transfixation of a posterior chamber IOL, intracameral air (for the treatment of a Descemet membrane detachment or intracameral migration after vitreoretinal surgery), inflammation, peripheral anterior synechiae, or angle recession can increase a patient’s risk toward developing ACG upon trauma. For example, angle closure in an elderly Asian woman may direct the physician to a preexisting occludable anterior chamber angle, whereas a child who develops angle closure may have ciliochoroidal edema.

It is important to determine the mechanism of the angle closure, because the treatments of the two types of ACG differ. Surgeons generally manage traumatic pupillary-block ACG with a laser peripheral iridotomy (LPI). In contrast, physicians usually treat nonpupillary-block ACG with mydriatics to relax the ciliary body and allow the lens-iris diaphragm to move posteriorly.

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PUPILLARY-BLOCK ACG

As stated, patients with preexisting occludable anterior chamber angles can develop pupillary-block glaucoma after trauma. Tse and colleagues reported ACG in a 52-year-old woman who sustained blunt ocular trauma and a periorbital hematoma. The patient underwent an LPI and was found to have bilaterally shallow anterior chambers. On the other hand, Kutner reported a case of blunt trauma causing ciliary body edema in an 18-year-old that resolved with cyclopentolate. Ultrasound biomicroscopy after blunt trauma displaces the anterior lens, with resultant transient myopia, increased lens thickness (probably due to lens edema), choroidal detachment, and ciliary body edema. Resolution reverses the myopia.

Subluxated lenses or herniated vitreous can also cause pupillary block. Sowka reported the case of a 72-year-old pseudophakic man who, after blunt trauma, suffered a posterior capsular rupture, which resulted in vitreous prolapse, pupillary block, iris bombe, and ACG. He was treated with cycloplegics as well as anti-inflammatory agents and topical glaucoma medications and, subsequently, with a total of three LPIs.
Another cause of pupillary block is iatrogenic trauma from laser treatment. Melamed and colleagues found that, in patients with subluxated lenses, a YAG LPI produced further anterior tilting of the cataract and subsequent ACG. YAG iridotomy can also cause a posterior dislocation of the lens and pupillary-block glaucoma. The mechanism most likely involves a shock-wave effect and zonular rupture. YAG capsulotomy is another reported cause of the IOL’s anterior dislocation, with vitreous herniation and resultant pupillary block. This type of angle-closure attack is broken with pupillary dilation.

Some traumatic forms of ACG require more than cycloplegia or LPI. In a case report by Netland et al., trauma caused the crystalline lens’ dislocation into the anterior chamber, which led to pupillary block that necessitated a lenectomy and anterior vitrectomy. Patients predisposed to anteriorly displaced lenses (Marfan syndrome, spherophakia, Weill-Marchesani syndrome, or homocystinuria) are at increased risk of traumatic dislocation of a phakic lens.

**ADDITIONAL MECHANISMS**

Orbital disorders, particular carotid-cavernous fistulas, and embolization treatments have been associated with ACG. The mechanism involves venous thrombosis and stasis, concomitant exudative retinal detachment and choroidal detachment, intraorbital engorgement, raised episcleral venous pressure, and blood in Schlemm canal on gonioscopy (Figure). Embolization results in increased superior ophthalmal venous pressure, choroidal transudation, the anterior displacement of the lens-iris diaphragm, and nonpupillary-block glaucoma.

Transvenous coil embolization, however, has also been reported to produce pupillary-block glaucoma, causing a cranial nerve III palsy and resultant mydriasis in a patient already predisposed to narrow anterior chamber angles. Penetrating trauma can also cause ACG. In one report, the formation of extensive anterior synechiae after the inadvertent penetration of anesthetic agents into the anterior chamber necessitated the implantation of a glaucoma drainage device.

**CONCLUSION**

Blunt and penetrating trauma to the eye can result in traumatic ACG. The mechanism appears to involve mass effect (vitreous, lens) into the pupillary region, resulting in pupillary block, or choroidal swelling and rotation of the lens-iris diaphragm without pupillary block. A third mechanism involves traumatic mydriasis or associated cranial nerve palsy that results in a dilated pupil and subsequent pupillary-block glaucoma. Clinicians should have a higher level of suspicion for ACG when the patient has predisposing risk factors such as hyperopia, shallow anterior chambers, and a history of surgery.

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