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# Ocular Trauma and Glaucoma

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## ■ Introduction

Development of glaucoma must be considered after any ocular trauma. Transient or prolonged elevations in intraocular pressure (IOP) and damage to the trabecular meshwork and other structures predispose traumatized eyes to the development of glaucomatous optic nerve loss. Each trauma case is unique, but ocular injury tends to occur in certain patterns. An understanding of these patterns allows one to more accurately predict the risk of glaucoma development and the type of glaucoma likely to occur in any given case. It must also be remembered that some of the treatment of ocular trauma cases, such as **steroid therapy or scleral buckling, can cause increases in IOP.** Patients with eye trauma must be carefully counseled on their lifelong risk of glaucoma. Close follow-up is recommended with specific attention paid to any early signs of glaucomatous changes.

## ■ Classification of Ocular Trauma

Ocular trauma can be broadly classified into blunt and penetrating injury. Traumatic glaucoma can also be classified as early or delayed onset. The most intuitive and clinically useful way to approach trauma-related glaucoma is to consider penetrating injury as a separate category and to divide glaucoma related to blunt injury into early and delayed onset. Other settings that warrant mentioning include chemical injury and surgery. Table 1 outlines the different categories of ocular trauma

**Table 1.** *Categories of Ocular Trauma and Glaucoma*


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Blunt injury
Early onset
Iritis
Hyphema
Delayed onset
Angle recession
Ghost cell glaucoma
Penetrating injury
Epithelial/stromal downgrowth
Siderosis
Other situations
Chemical injury
Ocular surgery
Malignant glaucoma
Aphakic glaucoma
Pseudophakia
Penetrating keratoplasty
Vitrectoretinal procedures

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glaucoma and the specific clinical scenarios that will be considered in each.

## ■ Epidemiology

The lifetime prevalence of ocular trauma is estimated to be 19.8% with a 5-year incidence of 1.6%.<sup>1</sup> Approximately 2.4 million ocular injuries occur in the United States each year.<sup>2</sup> **The risk of developing glaucoma after a closed globe contusion was found to be 19% in 1 study.<sup>3</sup> Another study found the corresponding risk with penetrating ocular trauma to be 2.67%.<sup>4</sup>** These data would suggest that blunt trauma make up the majority of trauma-related glaucoma cases. The immediate and dramatic nature of penetrating injury is unmistakable and patients are far more likely to seek care immediately where they are educated about their injury and prognosis. The same cannot be said for most cases of blunt eye injury. It is up to the ophthalmologist to educate patients and primary care providers about the insidious nature of posttraumatic glaucoma and the need for routine surveillance.

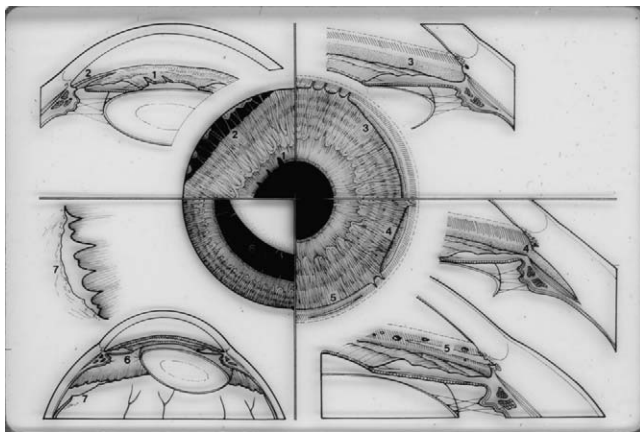
## ■ Blunt Trauma

Blunt eye trauma is a common injury and each case must be considered as a risk factor for the development of glaucoma. Clear cases

such as an iris tear, orbital floor fracture, or layered hyphema tend not to be missed, even in a primary care setting. Ideally, these patients are made aware of their risk for glaucoma and receive the appropriate follow-up. However, deceptively innocuous injuries such as a finger poke corneal abrasion can cause damage that eventually leads to glaucoma. Blunt ocular injury can occur from an airbag and can be overlooked in the context of more severe systemic motor vehicle injuries.

The patterns of injury incurred with blunt trauma are due to equatorial expansion when blunt force indents the anterior aspect of the globe. These injury patterns were described by Campbell<sup>5</sup> as 7 rings (Fig. 1). From anterior to posterior, they are radial tears of the pupillary sphincter, iridodialysis of the iris root, angle recession, cyclodialysis of the ciliary body, trabecular meshwork tear, zonular dehiscence, and retinal dialysis. These injury patterns can be used, even years later, as evidence of blunt injury. In some cases, the injury pattern is invisible to the examiner and the only clues come from a thorough history, with specific attention to any past eye injury, past facial injuries, and history of participating in sports such as boxing.

Glaucoma resulting from blunt eye injury can present early or late in the postinjury period. A transient rise in IOP after injury is not truly glaucoma unless it produces a glaucomatous change in the optic nerve or visual field. This is rare in the early postinjury period unless the IOP is severely elevated or remains elevated for a prolonged period. Certain eyes, however, seem to be more susceptible to glaucomatous changes, even with seemingly mild elevations of pressure.



**Figure 1.** The 7 rings of injury in blunt eye trauma: 1—pupillary tear; 2—iridodialysis; 3—angle recession; 4—cyclodialysis; 5—trabecular meshwork tear; 6—zonular dehiscence; 7—retinal dialysis. With permission from *Eye Trauma*. St. Louis: Mosby Year Book; 1991:118.

### **Traumatic Iritis**

The initial IOP in cases of traumatic iritis is often low. This is thought to be due to decreased aqueous production by a stunned ciliary body. The IOP then tends to rise in the few days after injury owing to clogging of the trabecular meshwork with inflammatory debris. It has been proposed that swelling of the trabecular meshwork itself may contribute to the outflow obstruction.<sup>3</sup> The IOP elevation is usually mild and easily controlled. Recommended treatment for traumatic iritis includes cycloplegia and topical steroids. These agents alone will often decrease the IOP to an acceptable range. If hypotensive agents are used,  $\beta$ -blockers and carbonic anhydrase inhibitors are usually the first line. Prostaglandin analogs should be avoided in this setting as these can exacerbate ocular inflammation. Pilocarpine can also increase both inflammation and the risk for posterior synechiae and is therefore also an inappropriate choice for pressure control.

### **HypHEMA**

Blood in the anterior chamber, whether microscopic or layered, is a common finding in cases of blunt trauma. There is controversy regarding the recommended treatment, which is beyond the scope of this text but some of the debated issues include the need for hospitalization, the use of aminocaproic acid, and whether to instruct patients to discontinue use of blood thinners. Acute elevations in IOP in hypHEMA cases are caused by mechanical obstruction of the trabecular meshwork by blood products and fibrin. Chronic IOP elevation is due to permanent damage to the meshwork, either by direct trauma with or without visible angle recession or rarely by secondary fibrosis and descemetization of the angle.<sup>6</sup> Therefore, unless anterior chamber fibrotic changes are evident on examination and gonioscopy, patients with a history of hypHEMA who go on to develop glaucoma are usually approached as an angle recession patient, even in the absence of discernable recession on examination.

Two subgroups of hypHEMA patients warrant special attention: sickle cell patients and those with a rebleed. The rate of rebleed after a traumatic hypHEMA has been variably reported at anywhere from 3.5% to 38%.<sup>7</sup> When a rebleed occurs, it is often more severe and more damaging than the initial hypHEMA. Sickle cell patients are not only at higher risk for rebleed, but are also more likely to develop glaucomatous nerve damage, even with only moderate IOP elevations.<sup>8</sup>

HypHEMA patients need to be followed daily for 3 to 5 days to monitor for rebleed and for IOP checks. Eye rest and shielding throughout this period is imperative. Pressures over 25 should be treated. Unless there are contraindications,  $\beta$ -blockers are usually used first.<sup>9</sup> Surgical washout of the anterior chamber is usually reserved for

cases with elevated IOP uncontrolled by medical therapy or evidence of corneal blood staining.

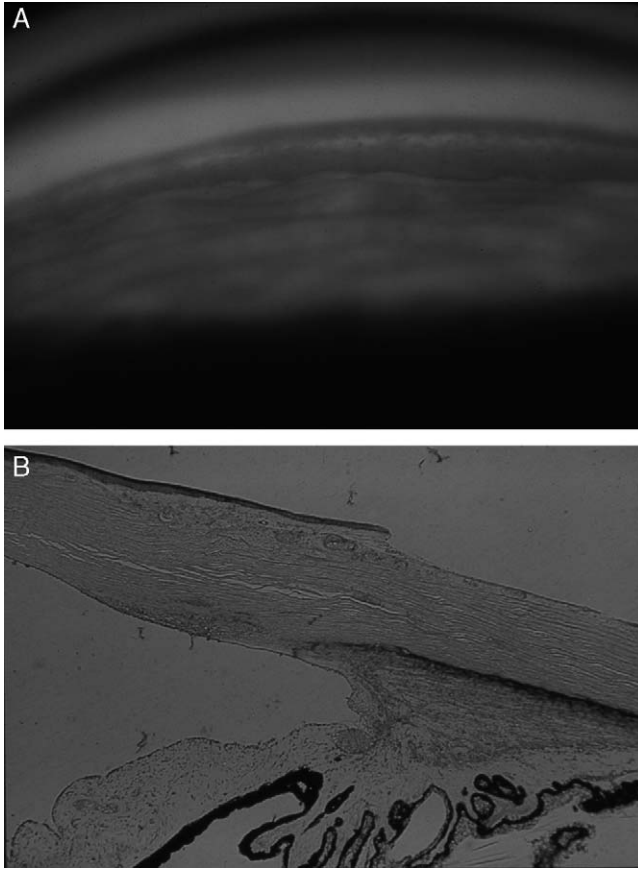
It must be remembered, however, that only the gonioscopic examination can provide the crucial information about the level of permanent damage sustained by the anterior chamber angle and trabecular meshwork. Only a very careful and deliberate gonioscopic examination of both eyes will reveal the sometimes visually unimpressive changes of angle recession. In 1 study of sports-related eye injuries, 55.6% of patients who presented with hyphema were found to have some degree of angle recession.<sup>10</sup> Other studies have found angle recession in over 70% of hyphema cases.<sup>11</sup> **Gonioscopy is typically performed 3 to 6 weeks after the injury to minimize the risk of causing a rebleed.** Regardless of what is observed during this examination, it is an ideal opportunity to educate the patient about the injury the eye has sustained and the lifelong risk for glaucoma. Baseline visual fields and disc photos should be obtained.

### **Angle Recession**

Secondary open-angle glaucoma associated with angle recession represents perhaps the most subtle and yet most devastating form of trauma-related glaucoma. Any patient with blunt eye trauma needs careful gonioscopic examination of the angle of both eyes to look for any evidence of angle recession (Figs. 2A, B). **The angle recession itself is not thought to be responsible for outflow obstruction; rather, it represents a visible marker of invisible damage sustained by the trabecular meshwork.**

Of patients with traumatic angle recession, somewhere between 5% and 20% will go on to develop glaucoma.<sup>12</sup> The higher risk values are typical of patients with greater amounts of recession, typically 180 degrees or more.<sup>13</sup> **Of those patients who do develop glaucoma, up to 50% will eventually develop glaucoma in the fellow eye.**<sup>14</sup> This suggests that these patients **might have a predisposition to glaucoma and that trauma can perhaps initiate the cascade of glaucomatous damage.**

The initial treatment of angle recession glaucoma is medical, and typical open-angle glaucoma drop regimens are employed. Laser trabeculoplasty tends to be relatively ineffective.<sup>15,16</sup> When standard medical therapy fails to lower the IOP to a satisfactory degree, or the glaucoma continues to progress despite maximal medical therapy, filtering surgery is usually the next step. Mermoud et al<sup>17</sup> compared standard trabeculectomy, trabeculectomy with antimetabolites, and Molteno implant surgery for patients with angle recession glaucoma. Trabeculectomy with antimetabolites was associated with the greatest reduction of IOP and the fewest postoperative glaucoma meds. Of note, the rate of bleb infection was also highest in that same study group. Cyclodestructive procedures are reserved for patients with limited visual



**Figure 2.** *A, Gonioscopic view of angle recession. B, Histopathologic finding of angle recession. With permission from Color Atlas of Glaucoma. Baltimore, MD: Williams & Wilkins; 1998:133.*

potential but can offer effective and long-lasting IOP control in refractory cases.<sup>18</sup>

The clinical case outlined below illustrates several important points about glaucoma related to blunt trauma including the unimpressive nature of the original injury, the absence of significant exam findings to suggest trauma or a risk of glaucoma, the rapid development of severe secondary open-angle glaucoma, and the eventual involvement of the fellow eye. The names and dates have been altered to protect patient confidentiality.

MT was 22 years old when his right eye was injured in a playful wrestling match. He was seen by an ophthalmologist and found to have an eyelid abrasion, corneal abrasion, and subconjunctival hemorrhage. The eye healed well and had normal vision and no residual pain. There

was no hyphema detected and no gonioscopic examination was documented at that time. No plan for routine ophthalmologic follow-up was made. One year later, he was noted to have increased eye pressure of 26 mm Hg in the right eye on a routine optometry eye examination. It was not until 3 years later that a referral to ophthalmology was made and the diagnosis of glaucoma was confirmed. Gonioscopic examination at that time was found to be normal, with no visible angle recession. At this point, the patient did have obvious asymmetric cupping and a superior nasal step visual field defect in the right eye. Incrementally, maximal medical management was implemented but despite good IOP control (14 mm Hg), progression of the nerve cupping and field loss continued. The pressure in the uninjured left eye then began to rise. Eight years after the initial injury to the right eye, optic nerve head changes were documented in the left eye, glaucoma was diagnosed, and topical therapy was initiated. Five years later, 13 years after the injury, the patient underwent a trabeculectomy with mitomycin C of the right eye. Short-wave automated perimetry testing performed shortly after this procedure revealed early glaucomatous loss superiorly in the left eye. The trabeculectomy bleb eventually healed with minimal filtration and elevated IOP, despite maximal topical and oral glaucoma medication. A glaucoma drainage device was placed with good resultant IOP control. Follow-up visual fields show a stable defect superiorly in the right eye and a stable superior arc in the left eye (see below) (Figs. 3A–C).

### ***Ghost Cell Glaucoma***

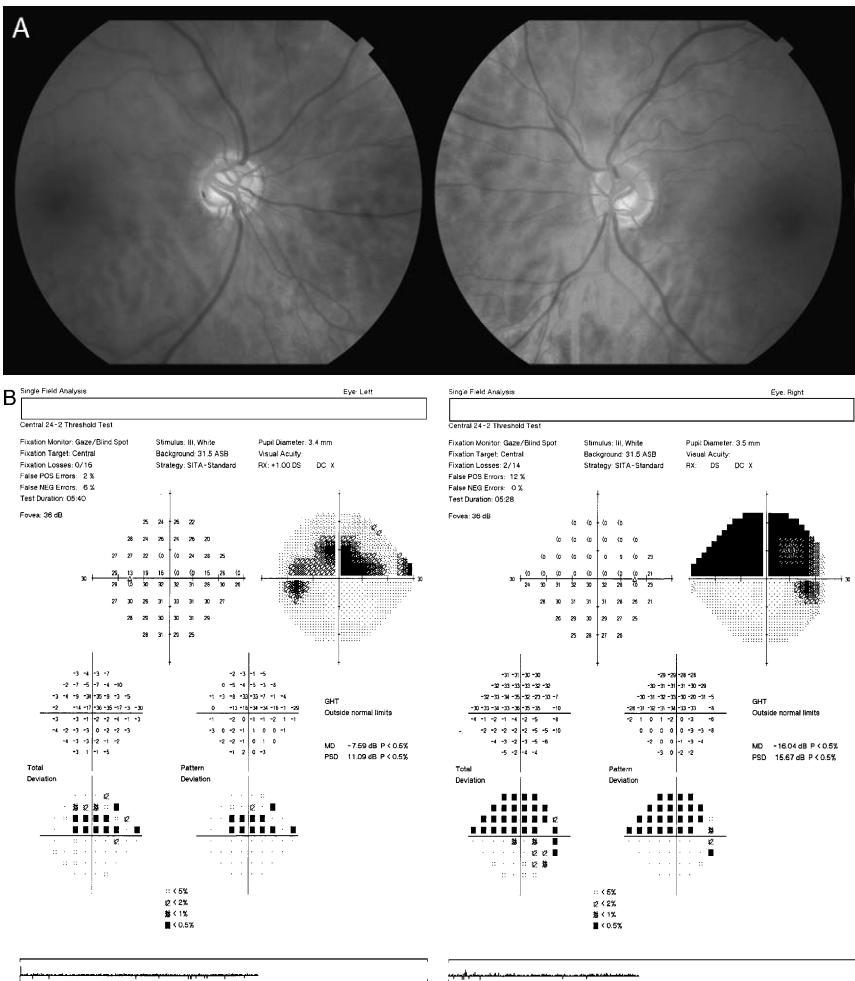
Fresh red blood cells, such as those that are found in an acute hyphema, are pliable and can percolate through the trabecular meshwork. The same is not true of degenerated “ghost cell” erythrocytes. These rigid, khaki-colored, cells form over the course of several weeks and can raise IOP by obstructing the meshwork.<sup>19</sup> This process was classically described occurring **2 or 3 weeks after a vitreous hemorrhage with rupture of the anterior hyaloid face**. It can occur, however, after any intraocular hemorrhage, even if no vitreous hemorrhage or disruption of the hyaloid occurs. The ghost cells can be observed flowing freely in the anterior chamber or as a **tan stripe in a background of red cells, creating the so-called candy-stripe sign** (Fig. 4).

Definitive diagnosis is made by microscopic examination of an anterior chamber specimen.<sup>20</sup> On light microscopy, the ghost cells appear as **rigid spheres** with small dense adherent dots on their surface called **Heinz bodies**. Most cases of ghost cell glaucoma are relatively transient and medical treatment is sufficient to control IOP. Certain cases with dense hemorrhage or pressure not adequately controlled by medical therapy may require **anterior chamber washout or vitrectomy procedures to aid in pressure control**.

## ■ Penetrating Injury

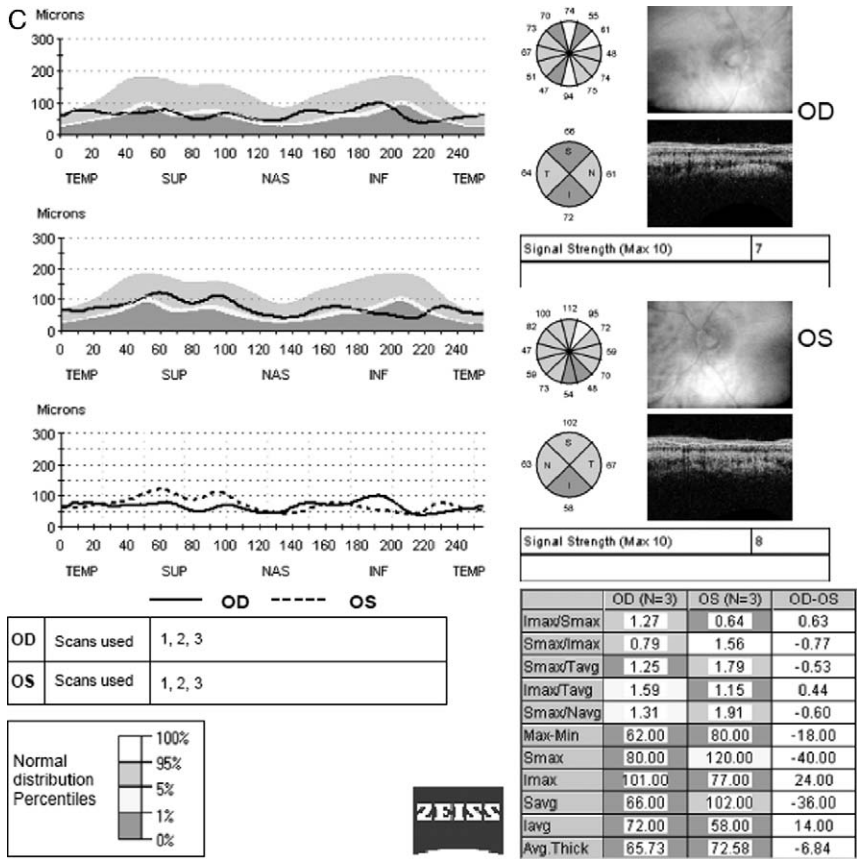
If every case of blunt ocular trauma is unique, penetrating trauma cases are even more so. Unlike blunt trauma, the injuries tend not to follow any predictable patterns and the glaucoma that results is more often owing to multiple factors. Many of the same mechanisms encountered with blunt trauma can also be at play in cases of glaucoma after penetrating injury. Phenomena such as hyphema, angle recession, and ghost cell glaucoma can all occur.

Any penetrating injury can initiate inflammation that eventually leads to uveitic glaucoma. Inflammation must be carefully controlled



(Figure 3. Contd).

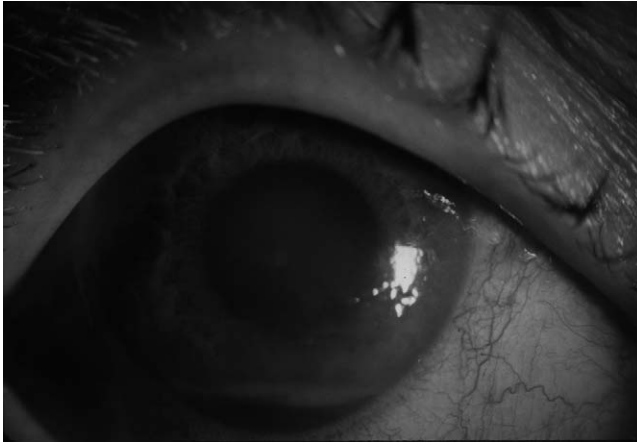




**Figure 3.** A, Disc photographs. B, Humphrey 24-2 SITA visual field. C, OCT retinal nerve fiber layer analysis. OCT indicates optical coherence tomography; SITA, Swedish Interactive Thresholding Algorithm.

and cycloplegia is usually recommended during the acute postinjury phase. Any glaucoma patient with a history of trauma must be examined for peripheral anterior synechiae as a cause of progressive angle closure. Uveitic glaucoma is a separate topic, which will not be considered in detail here but it worth noting that this type of glaucoma can occur after trauma, especially penetrating trauma.

Long-term use of corticosteroids is common in cases of penetrating trauma and this can lead to elevated IOP. The rise in IOP tends to occur 2 to 3 weeks after initiation of therapy and is dose-dependent.<sup>21</sup> Even after cessation of steroid treatment, patients sometimes require lifelong IOP control, perhaps owing to irreversible changes in the trabecular meshwork.<sup>22</sup>



**Figure 4.** “Candy-stripe sign.” With permission from *Color Atlas of Glaucoma*. Baltimore, MD: Williams & Wilkins; 1998:131.

### **Epithelial/Stromal Downgrowth**

Epithelial or stromal growth into the anterior chamber is a rare phenomenon with devastating consequences. Surgery remains the leading cause of both epithelial and stromal downgrowth, with large incision cataract surgery making up most of the cases, followed by penetrating injury and corneal transplant.<sup>23</sup>

Epithelial downgrowth is classically divided into 3 forms: epithelial pearls, epithelial cysts, and epithelial downgrowth.<sup>24</sup> It is the third form, epithelial downgrowth, typified by sheets of epithelial growth, which is the most common and most destructive. Clinical presentation of epithelial downgrowth can occur anywhere from 4 days to 38 years after the inciting event,<sup>26</sup> with glaucoma and retrocorneal membrane being the 2 most common initial findings. A patent fistula may also be seen on presentation, and these cases tend to have a low initial IOP.

Epithelialization of the anterior chamber can lead to glaucoma through several mechanisms including growth over the trabecular meshwork, peripheral anterior synechiae formation, mucus plugging of the meshwork, and pupillary block.<sup>25</sup> These cases tend to be intractable and carry a poor prognosis. Medical IOP management is relatively ineffective and does not treat the other complications such as corneal decompensation owing to retrocorneal membrane formation. Surgical removal of the epithelial membrane with adjunctive cryotherapy is difficult but can offer some limited success.<sup>26</sup>

The incidence of epithelial downgrowth seems to be declining, likely owing to improved surgical techniques with smaller wounds, improved instrumentation, less disruption of ocular surface tissues, and less need for multiple procedures. However, a recent large case series showed

a relatively unchanged incidence of epithelial downgrowth and an increased rate of clinical misdiagnosis.<sup>27</sup> With increases in the number of cataract surgeries being performed and the improved salvage of severely traumatized eyes, the incidence of downgrowth might remain constant despite decreasing rates of this complication. Given the ravaging nature of this process, a high index of suspicion is warranted in any new onset glaucoma case in an eye that has had surgery or trauma. Of note, epithelial downgrowth has almost never been described due glaucoma surgery alone.<sup>28</sup>

Stromal downgrowth is also called fibrous downgrowth and retrocorneal membrane. This process occurs in similar setting as epithelial downgrowth but tends to be less destructive. Cases occur most often after cataract surgery but have been seen after trauma, filtering surgery, and goniotomy.<sup>25</sup> Unlike the retrocorneal membrane of epithelial downgrowth, a fibrous membrane is more often vascularized. Medical management of glaucoma in cases of stromal downgrowth can be successful and drastic surgical interventions are not often required.<sup>29</sup>

### **Siderosis**

Iron is toxic to the epithelial tissues of the eye. Excess iron, either from a retained intraocular foreign body or from chronic intraocular hemorrhage (hemosiderosis) can lead to a pattern of tissue damage in the eye termed siderosis. It has been reported that up to 40% of all open globe injuries retain some amount of intraocular foreign body.<sup>30</sup> Examination findings consistent with siderosis include iris heterochromia with darkening of the iris in the affected eye, a dilated and poorly reactive pupil, rustlike deposits on the corneal endothelium and anterior lens, optic nerve edema, glaucoma, and pigmentary retinopathy.<sup>31</sup> Degenerative changes in the trabecular meshwork including sclerosis and loss of the intertrabecular spaces have been found in siderotic eyes.<sup>32</sup> Any eye with a retained metallic foreign body or long-standing intraocular hemorrhage must be followed closely for glaucoma and for electroretinogram changes consistent with this condition.<sup>33</sup>

### ■ **Chemical Injury**

Both acid and alkali burns can acutely raise the IOP. The mechanism for this is poorly understood but is thought to be due to contraction of the anterior tissues of the eye.<sup>34–36</sup> This is typically followed by a return to normal or subnormal pressure and a slow, progressive increase in IOP. Acutely, topical steroids and aqueous suppressants can be helpful in managing the pressure. Cases that progress to sterile corneal ulceration typically have the most extensive anterior segment damage, and are most likely to develop glaucoma and eventual phthisis.<sup>37</sup>

## ■ Ocular Surgery

### ***Malignant Glaucoma***

Malignant glaucoma, also called ciliary block glaucoma or aqueous misdirection, has been recognized as a consequence of ocular surgery for more than a century. Certain key characteristics distinguish this entity from other conditions such as acute angle-closure glaucoma, including a uniform shallowing of the anterior chamber both centrally and peripherally, an elevated IOP, exacerbation with miotics, and relief with cycloplegics.<sup>38</sup>

Malignant glaucoma was classically described as a complication of incisional surgery for angle-closure glaucoma, reported to occur in up to 4% of cases,<sup>39</sup> but has been recognized as a consequence of many ocular procedures and even as a spontaneous event. Factors that seem to affect the rate of postoperative malignant glaucoma include partial or total angle closure at the time of surgery and a history of acute angle-closure attacks. The type of surgery and the IOP at the time of surgery are not significant factors.<sup>40</sup> The onset of malignant glaucoma is typically early in the postoperative period but has been reported years later. These late-onset cases are often related to cessation of cycloplegic therapy and the initiation of miotic medications.<sup>40</sup>

Shaffer<sup>41</sup> first proposed the role of the vitreous and hyaloid face in the development of malignant glaucoma and although medical management is the first-line treatment, definitive treatment is directed at these structures. Medical management typically includes cycloplegics, hyperosmotics, and aqueous suppressants. Atropine therapy is often used indefinitely after resolution to prevent recurrence. Medical therapy alone can relieve malignant glaucoma in up to 50% cases within 5 days.<sup>42</sup> There is little agreement on the best surgical approach if medical therapy fails. Disruption of the anterior hyaloid face with Nd:YAG laser<sup>43</sup> or shrinkage of the ciliary processes with argon laser photocoagulation<sup>44</sup> have been described. Pars plana vitrectomy is felt by many to be the single definitive therapeutic option. Recently, a multistep procedure has been proposed for malignant glaucoma in phakic eyes. Termed “vitrectomy-phacoemulsification-vitrectomy,” this approach involves an initial core vitrectomy to decompress the pressurized vitreous cavity, standard clear cornea phacoemulsification and intraocular lens implantation, and finally, completion of the vitrectomy with intentional zonulo hyaloidectomy and peripheral iridotomy.<sup>45</sup>

### ***Aphakic Glaucoma***

Malignant glaucoma is well recognized as a possible complication after ocular surgery and aphakia. As discussed previously, aqueous

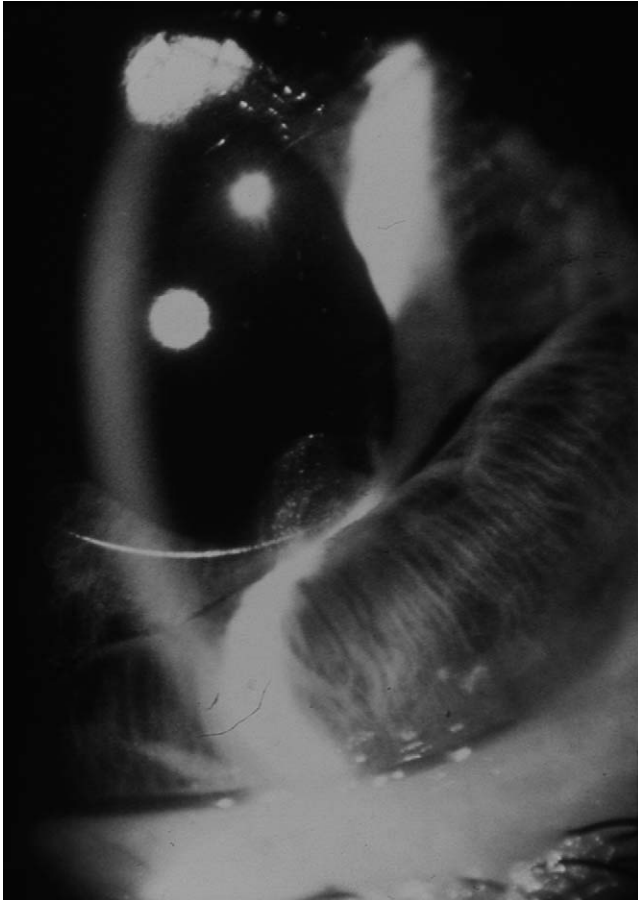
misdirection into the posterior segment can push the anterior hyaloid forward, occlude the pupil, and create a uniform shallowing of the anterior chamber. However, other forms of IOP derangement can also occur. An initial and transient rise in IOP after lens extraction without lens implantation is well recognized.<sup>46</sup> This tends to be of little long-term consequence. More importantly, a tenacious and destructive chronic form of glaucoma can develop in as many as 3% aphakic eyes.<sup>47</sup> The exact mechanism underlying this process is unknown but it has been proposed that the multiple sutures used for closure may distort the angle and lead to outflow obstruction.<sup>48</sup> A treatment algorithm similar to that taken in chronic angle-closure cases would therefore be a logical approach.

### ***Glaucoma and Pseudophakia***

The effects of cataract extraction and pseudophakia on the IOP and anterior chamber angle are complex and case-dependent. Numerous factors such as retained viscoelastic or vitreous in the anterior chamber, lens-related pupillary block (Fig. 5), pigment dispersion, uveitis-glaucoma-hyphema syndrome, and perioperative use of steroids can all create transient or prolonged elevations in IOP. Of note, cataract extraction with posterior chamber implantation can lower IOP in cases of primary angle-closure glaucoma. Nonaka et al<sup>49</sup> have recently used anterior segment ultrasound biomicroscopy to demonstrate an anatomic change including angle widening and posterior shifting of the ciliary processes after cataract extraction in such patients. This was true even in patients who had not demonstrated any anatomic change after laser peripheral iridotomy.<sup>50</sup> A reduction in IOP was also noted after surgery in eyes with previous laser peripheral iridotomy, from 19.3 to 14.8 mm Hg.<sup>50</sup>

### ***Glaucoma and Penetrating Keratoplasty***

A rise in IOP in the immediate postoperative period after a penetrating keratoplasty (PK) is not an uncommon event and has been reported to occur in up to 31% cases.<sup>51</sup> Long-term elevations in IOP and glaucomatous nerve damage occur in approximately 1 of every 5 cases.<sup>52</sup> Factors that seem to be predictive of the development of glaucoma after PK include aphakia, preexisting glaucoma, repeated PK, and an early postoperative IOP elevation.<sup>51,53,54</sup> Post-PK glaucoma is concerning not only owing to the glaucomatous optic nerve damage that can ensue but also because elevated pressures can increase the rate of graft failure.<sup>55</sup> In a recent large retrospective series, Al-Mohaimed et al<sup>56</sup> found that the need for escalation of glaucoma therapy after PK was associated with preexisting glaucoma, advanced patient age, pseudophakia or aphakia, and a surgical indication for PK. Glaucoma was found to be definitively related to an increased graft failure rate and worse visual acuity.



**Figure 5.** *Pupillary block associated with anterior chamber intraocular lens. With permission from Color Atlas of Glaucoma. Baltimore, MD: Williams & Wilkins; 1998:135.*

Theories to explain the cause of glaucoma after PK include changes in the anterior chamber angle<sup>57</sup> and incision-related loss of structural support for the trabecular meshwork.<sup>58</sup> Medical therapy is the first-line option in cases of post-PK glaucoma as incisional drainage surgery and cyclodestructive procedures, although effective in achieving IOP control, are associated with a high rate of graft failure.<sup>59,60</sup> Ramesh et al<sup>61</sup> found no statistically significant difference between mitomycin C trabeculectomy, glaucoma drainage device, and Nd:YAG cyclophotocoagulation in terms of IOP control or rate of graft failure.

## **Glaucoma and Vitreoretinal Surgery**

Elevations of IOP after vitreoretinal procedures are a common occurrence and can be severe. The mechanisms involved vary widely, as do the appropriate treatments, and are beyond the scope of this text. In a series of cases, the IOP within 48 hours after pars plana vitrectomy rose by 5 to 22 mm Hg in 61.3% of eyes and by 30 mm Hg in 35.6%.<sup>62</sup>

A high level of vigilance is therefore warranted and IOP management must be tailored to each individual case.

## ■ **Conclusions**

Glaucoma is a common and often devastating consequence of ocular injury, whether from accidental trauma or intentional surgical interventions. A thorough knowledge of the risk factors for posttraumatic glaucoma and careful examination are required for rapid identification of affected patients. Patient education, careful surveillance, and early intervention provide the posttrauma patient with the best chance for long-term vision preservation.

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The views expressed in this article are those of the authors and do not reflect the official policy or position of the Department of the Navy, Department of Defense, or the United States Government.

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