Early Predictors of Traumatic Glaucoma After Closed Globe Injury

Trabecular Pigmentation, Widened Angle Recess, and Higher Baseline Intraocular Pressure

Ramanjit Sihota, MD, FRCS; Sunil Kumar, MD; Viney Gupta, MD; Tanuj Dada, MD; Seema Kashyap, MD; Rajpal Insan, MD; Geetha Srinivasan, MS

Objective: To prospectively analyze the clinical and ultrasonographic biomicroscopy (UBM) features in eyes with closed globe injury, at the initial examination, that would predict the occurrence of chronic traumatic glaucoma during a 6-month follow-up.

Methods: Forty consecutive eyes with closed globe injury and a chronically elevated intraocular pressure (IOP) of at least 21 mm Hg for a minimum of 3 months were diagnosed as having traumatic glaucoma and compared with 52 eyes with closed globe injury and no evidence of glaucoma.

Results: The median grade of trabecular pigmentation on gonioscopy in eyes with traumatic glaucoma was 3 compared with 2 in eyes without glaucoma (P=.001). On UBM findings, 18 eyes with closed globe injury without glaucoma showed evidence of cyclodialysis, compared with 7 eyes with glaucoma (P=.001). The relative risk of developing traumatic glaucoma was also significantly higher with hyphema, elevated baseline IOP, angle recession of more than 180°, lens displacement, and wider angles on UBM.

Conclusions: Clinically, the presence of increased pigmentation at the angle, elevated baseline IOP, hyphema, lens displacement, and angle recession of more than 180° were significantly associated with the occurrence of chronic glaucoma after closed globe injury. On UBM findings, a wider angle and the absence of cyclodialysis were significant predictors for the subsequent development of traumatic glaucoma.

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TRAUMA IS A COMMON CAUSE of ocular morbidity and occurs most often during childhood or in young adults.1,2 Glaucoma after closed globe injury is a major concern because many cases may go unnoticed and, without close follow-up, are diagnosed many years later as having irreversible glaucomatous optic nerve damage.3,4 Two peak incidences of glaucoma after trauma have been reported, less than 1 year and at least 10 years after trauma.5 A 3.4% incidence of glaucoma after ocular contusion has been reported during a 6-month follow-up6 and up to 10% during the 10 years after trauma.7 Retrospective studies have identified ocular features commonly seen in eyes with traumatic glaucoma, such as poor baseline visual acuity, hyphema, an angle recession of more than 180°, traumatic cataracts, displacement of the lens, and iris injuries.3,6 However, only a few eyes develop glaucoma, despite the presence of traumatic damage such as angle recession. The aim of this study was to evaluate eyes prospectively with closed globe injury from the initial examination, to identify early ocular findings that could be significantly associated with the development of a chronic traumatic glaucoma, and to compare clinical and ultrasonographic biomicroscopy (UBM) findings in eyes that developed and did not develop chronic glaucoma.

METHODS

Consecutive patients initially seeking treatment in the ophthalmic casualty department after concussive closed globe injury during a 1-year period were included for evaluation. Details of the ocular injury—mode of injury, time from trauma to initial visit, and route, dose, and duration of therapy—were recorded. Prior ocular problems and a family history of glaucoma were also noted. Informed consent was obtained from all patients in accordance with the Declaration of Helsinki.

A thorough ocular examination of both eyes was performed, including best corrected

Author Affiliation: Glaucoma Research Facility and Clinical Services (Drs Sihota, Kumar, Gupta, Dada, Insan, and Srinivasan) and Department of Ocular Pathology (Dr Kashyap), Dr Rajendra Prasad Centre for Ophthalmic Sciences, All India Institute of Medical Sciences, New Delhi, India.
visual acuity, slitlamp biomicroscopic examination, fundus examination with a +90-diopter lens, and indirect ophthalmoscopy without indentation. Intraocular pressure (IOP) measurements on at least 3 occasions were recorded by means of applanation tonometry. The zone of injury was recorded and classified according to the location. Zone 1 injuries were superficial injuries limited to the bulbar conjunctiva, sclera, or cornea, including corneal abrasion and subconjunctival hemorrhage. Zone 2 injuries involved structures in the anterior segment up to and including the lens apparatus, the lens zonules, and the pars plicata. Zone 3 injuries were posterior injuries involving the pars plana, choroid, retina, vitreous, or optic nerve.

Patients older than 10 years who were cooperative during the UBM and gonioscopic examinations were included in the study. Exclusion criteria were an open globe injury, primary glaucoma or other preexisting cause of secondary glaucoma, and a history of ocular surgery or laser therapy.

Four weeks after the trauma, gonioscopy with 360° gonioscopy and UBM (UBM P-40; Paradigm Medical Industries, Salt Lake City, Utah) were performed by an experienced glaucoma specialist (V.G.) who was masked to the patient’s history and final diagnosis. On gonioscopic findings, the circumferential extent of angle recession and cyclodialysis were noted and pigmentation was graded as 0 (no pigmentation), 1 (faint), 2 (average), 3 (heavy), or 4 (very heavy). Ultrasound biomicroscopic images were obtained radially every clock hour to find angle recession, cyclodialysis, iridodialysis, and lenticular subluxation or dislocation. The anterior chamber depth, superior and inferior angle measurements in degrees, angle opening distance at 250 and 500 µm, and angle recession area were measured at the widest angle, with note of any other anterior segment abnormalities.

All patients were followed up every month for 6 months and as appropriate thereafter. At each visit, best corrected visual acuity, applanation tonometry, and thorough anterior and posterior segment evaluations were performed. Humphrey field analysis with a 30-2 SITA standard visual field (Humphrey Systems, Dublin, California) was recorded in patients with a best corrected visual acuity of more than 6/60. In the presence of a hyphema, patients underwent evaluation more frequently to monitor the IOP and corneal status.

All patients with concomitant posttraumatic uveitis were treated with corticosteroids for a maximum of 2 weeks. For an elevated IOP, they were treated with β-blockers, brimonidine, or dorzolamide hydrochloride topically and systemic antiglaucoma medications where necessary.

Eyes with an elevated IOP (≥21 mm Hg) and requiring glaucoma therapy for at least 3 months after closed globe injury were diagnosed as having a traumatic glaucoma. In the absence of such a chronically elevated IOP or evidence of glaucomatous optic neuropathy, eyes were diagnosed as having only a closed globe injury without glaucoma.

Histopathological evaluation of the trabeculectomy specimens was performed in eyes that underwent filtering surgery.

We used SPSS statistical software, version 10.0 (SPSS Inc, Chicago, Illinois) for comparing the variables between glaucomatous and control eyes. A binary logistic regression analysis was used to determine the relative risk of developing glaucoma, as evidenced by baseline clinical features and UBM findings. A P < .05 was considered statistically significant. Unless otherwise indicated, data are expressed as mean (SD).

### RESULTS

We reviewed 121 eyes of 121 consecutive patients older than 10 years who had had a recent closed globe injury. Ninety-two patients fulfilled inclusion criteria for the study after exclusion of 13 patients younger than 10 years, 5 with pseudophakia, and 1 who had undergone a recent vitreoretinal surgery.

Forty of the 92 patients (43%) had a persistent elevation of IOP (≥21 mm Hg) for at least 3 months, ie, traumatic glaucoma. The remaining 52 patients (57%) had an IOP consistently less than 21 mm Hg, with no evidence of glaucomatous optic neuropathy; these patients constituted the closed globe injury group. Demographic data are presented in Table 1.

When we classified the closed globe injury group without glaucoma by the zone of injury, 22 (42%) had a zone 1 injury; 20 (38%) had a zone 2 injury; and 10 (19%) had a zone 3 injury. The respective numbers in the traumatic glaucoma group were 1 (3%), 21 (53%), and 18 (45%) (P = .01).

Ocular findings are listed in Table 2. Hyphema, baseline IOP, trabecular pigmentation, angle recession, and lens displacement were statistically more frequent in the traumatic glaucoma group. Four patients with traumatic glaucoma had a recurrence of bleeding within 5 days of the trauma, compared with none of the patients with a closed globe injury.

A mean baseline IOP of 17.3 (5.0) mm Hg was recorded in the closed globe injury group and 35.2 (12.8) mm Hg in the traumatic glaucoma group (P = .001). Gonioscopic examination of the closed globe injury group revealed that 24 eyes had a normal angle structure. In 1 eye, a cyclodialysis cleft was noted, associated with angle recession. Of the remaining 27 eyes, 22 had angle recession of less than 180°; 3, of 180° to 270°; and 2, of more than 270°. On UBM examination, results showed that 22 eyes had a normal angle; 23, an angle recession of less than 180°; 5, an angle recession of 180° to 270°, and 2, an angle recession of more than 270° (Figure 1).

In the traumatic glaucoma group, gonioscopic findings revealed that 3 eyes had normal angles, whereas 37 (93%) had varying degrees of angle recession. Thirteen eyes had an angle recession of less than 180°; 9, of 180° to 270°; and 11, of more than 270°. In 4 eyes, gonioscopic findings revealed a small cyclodialysis cleft associated with angle recession. On UBM examination, 2 eyes had a normal angle. An angle recession was detected in 31 eyes, including 8 with an angle recession of less than

### Table 1. Demographic Data of 92 Patients With Closed Globe Injury

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Closed Globe Injury Without Glaucoma (n = 52)</th>
<th>Traumatic Glaucoma (n = 40)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean (SD), y</td>
<td>22.4 (11.0)</td>
<td>19.4 (9.5)</td>
<td>.74</td>
</tr>
<tr>
<td>Sex, No. M/F</td>
<td>49.3</td>
<td>37.3</td>
<td>.90</td>
</tr>
<tr>
<td>Trauma to presentation interval, mean (SD), d</td>
<td>10.8 (15.1)</td>
<td>9.5 (5.9)</td>
<td>.59</td>
</tr>
<tr>
<td>Type of trauma, No. (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cricket ball</td>
<td>23 (44)</td>
<td>17 (43)</td>
<td>.77</td>
</tr>
<tr>
<td>Firecracker</td>
<td>9 (17)</td>
<td>8 (20)</td>
<td>.90</td>
</tr>
<tr>
<td>Wooden stick</td>
<td>10 (19)</td>
<td>7 (18)</td>
<td>.85</td>
</tr>
<tr>
<td>Other</td>
<td>10 (19)</td>
<td>8 (20)</td>
<td>.88</td>
</tr>
</tbody>
</table>
180°; 10, of 180° to 270°; and 13, of more than 270°. In 7 eyes, a cyclodialysis cleft was detected on UBM examination (Figure 2), but the extent was less than 2 clock hours, and there was evidence of reattachment of the iris to the scleral spur in 5 eyes (Figure 3).

We found good correlation between the extent of angle recession seen by means of gonioscopy and UBM ($r^2=0.66; P<.001$). However, there was no significant correlation between cyclodialysis seen by means of gonioscopy and UBM ($r^2=0.16; P=.44$).

Trabecular pigmentation seen in eyes with traumatic glaucoma (median grade, 3; range, 2-4) was significantly more compared with that in eyes without glaucoma (median grade, 2; range, 1-4) ($P<.001$). Trabecular pigmentation of at least grade 3 was seen in 7 eyes (13%) in the closed globe injury group and in 36 (90%) in the traumatic glaucoma group, correlating significantly with the presence of traumatic glaucoma ($r^2=0.64; P<.001$). Trabecular pigment grades in 18 eyes with closed globe injuries and cleft were 4 in 1 eye, 3 in 5 eyes, and 2 or less in 12 eyes. Thirty-three eyes had traumatic glaucoma with no cleft, of which 15 had a trabecular pigmentation of grade 4, 13, of grade 3, and 5, of grade 2. The extent of pigmentation was not related to the extent of angle injury alone, but was also probably a result of other ciliary body and iris damage.

On UBM, all 4 angle variables were found to be significantly greater in patients with traumatic glaucoma compared with those with closed globe injuries ($P<.001$) (Table 3).

### Table 2. Anterior and Posterior Segment Findings in Eyes With Closed Globe Injury With and Without Chronic Glaucoma

<table>
<thead>
<tr>
<th></th>
<th>Closed Globe Injury (n=52)</th>
<th>Traumatic Glaucoma (n=40)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Visual acuity &lt;6/60 at initial examination, No (%)</td>
<td>12 (23)</td>
<td>25 (63)</td>
<td>.001</td>
</tr>
<tr>
<td>Mean (SD) baseline IOP, mm Hg</td>
<td>17.3 (5.0)</td>
<td>35.2 (12.8)</td>
<td>.001</td>
</tr>
<tr>
<td>Anterior segment features</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hyphema</td>
<td>22 (42)</td>
<td>37 (93)</td>
<td>.001</td>
</tr>
<tr>
<td>Sphincteric tears</td>
<td>18 (35)</td>
<td>17 (43)</td>
<td>.44</td>
</tr>
<tr>
<td>Iridodialysis</td>
<td>4 (8)</td>
<td>6 (15)</td>
<td>.43</td>
</tr>
<tr>
<td>Trabecular pigmentation grade ≥3</td>
<td>7 (13)</td>
<td>17 (43)</td>
<td>.44</td>
</tr>
<tr>
<td>Angle recession &gt;180°</td>
<td>6 (12)</td>
<td>14 (35)</td>
<td>.005</td>
</tr>
<tr>
<td>Angle recession of 360°</td>
<td>1 (2)</td>
<td>9 (23)</td>
<td>.03</td>
</tr>
<tr>
<td>Cyclodialysis on UBM</td>
<td>18 (35)</td>
<td>7 (18)</td>
<td>.001</td>
</tr>
<tr>
<td>Lenticular features</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cataract</td>
<td>7 (13)</td>
<td>10 (25)</td>
<td>.15</td>
</tr>
<tr>
<td>Phacodonesis</td>
<td>8 (15)</td>
<td>14 (35)</td>
<td>.03</td>
</tr>
<tr>
<td>Posterior segment features</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Macular edema</td>
<td>11 (21)</td>
<td>12 (30)</td>
<td>.33</td>
</tr>
<tr>
<td>Choroidal rupture</td>
<td>4 (8)</td>
<td>5 (13)</td>
<td>.67</td>
</tr>
<tr>
<td>Retinal dialysis</td>
<td>6 (12)</td>
<td>8 (20)</td>
<td>.26</td>
</tr>
<tr>
<td>Vitreous hemorrhage</td>
<td>5 (10)</td>
<td>7 (18)</td>
<td>.49</td>
</tr>
<tr>
<td>Macular hole</td>
<td>1 (2)</td>
<td>2 (5)</td>
<td>.41</td>
</tr>
</tbody>
</table>

Abbreviations: IOP, intraocular pressure; UBM, ultrasonographic biomicroscopy.

*Unless otherwise indicated, data are expressed as number (percentage) of eyes.

The relative risk of developing chronic glaucoma in an eye with closed globe injury, based on clinical features seen at presentation, was greatest if there was heavy pigmentation of the trabecular meshwork, an elevated baseline IOP, hyphema, angle recession, and lens displacement with a cataract (Table 4). Findings on UBM...
Glaucoma after blunt trauma appears to have 2 peaks of incidence, at less than 1 year and about 10 years after trauma,7 by which time acute symptoms and signs of trauma have subsided and the patient is unaware of a chronically elevated IOP. It is important to be able to identify eyes at risk for such a chronic traumatic glaucoma and to review them carefully, so that appropriate therapy may be initiated as early as possible.

In our study, the anterior chamber angle was carefully studied by means of gonioscopy and recorded using 360° goniophotography. The presence of heavy trabecular pigmentation (grade ≥3) was a significant predictor of chronic glaucoma, with a relative risk of 20.8. Extensive release of pigment into the eye during trauma could clog the trabecular meshwork, and trabecular endothelial cells that phagocytize the pigment particles could also block the meshwork, directly and through changes induced at the meshwork.8 The degree of pigmentation may be an indicator of the extent of anterior segment damage, but pigmentation of at least grade 3 was present in only 13% of eyes that did not develop a chronic glaucoma, although they also had severe disruption on the anterior segment anatomy (eg, subluxation of the lens and iris injury). The degree of trabecular pigmentation has been correlated with elevated IOP in other conditions such as pigmentary glaucoma or pseudoxfoliation.
tion and after the insertion of piggyback or sulcus-fixed intraocular lenses.\textsuperscript{10-13}

The baseline IOP of eyes developing chronic glaucoma was higher than that of eyes that did not, with a range of 22 to 60 mm Hg compared with a range of 10 to 28 mm Hg. This has not been previously reported because most studies were retrospective. The elevated IOP at baseline probably reflects decreased aqueous outflow due to extensive primary damage, inflammation, and pigment release at the trabecular meshwork.

We noted that a greater extent of angle recession resulted in a greater risk of traumatic glaucoma. Angle recession was present in 93\% of the eyes in our traumatic glaucoma group, and was also seen in 54\% of those without glaucoma. This suggests that there are additional factors involved that produce an elevation in IOP. Girkin et al\textsuperscript{a} reported an incidence of angle recession of 8.6\% in eyes without glaucoma and of 35.8\% in eyes with glaucoma at 6 months. This is much lower than our observations, as is the 19\% to 50\% incidence previously recorded,\textsuperscript{1,15} with incidence increasing in the presence of a hyphema.\textsuperscript{7} This may be because our study was designed to look for such changes, unlike the records reviewed retrospectively by Girkin et al.\textsuperscript{a} Long-term studies of eyes having an angle recession greater than 180\° have shown that only 4\% to 9\% develop late chronic glaucoma.\textsuperscript{5,10,17}

Ultrasonographic biomicroscopy allowed us to objectively identify and delineate angle recession and other features in traumatized eyes. A cyclodialysis was seen by us in only 7 eyes with traumatic glaucoma (18\%) and 18 of those without glaucoma (35\%). The presence of a cyclodialysis was protective against the occurrence of chronic glaucoma on multivariate analysis. A cyclodialysis cleft results from disinsertion of longitudinal fibers of the ciliary muscle from the scleral spur and underlying sclera, allowing direct communication between the anterior chamber and ciliochoroidal space and unrestricted bulk flow of aqueous from the anterior chamber to the supraciliary space. A cyclodialysis is usually associated with a reduced IOP on initial examination, but IOP may increase spontaneously later with closure of the cyclodialysis cleft. In some eyes, iris tissue blocks the cleft and prevents the development of hypotony. To the best of our knowledge, no previous study has looked at the incidence of cyclodialysis in the occurrence of or protection from traumatic glaucoma.

Cyclodialysis was diagnosed by UBM findings and was missed on gonioscopic findings in a large number of eyes. Cyclodialysis clefts may be difficult to detect in recently traumatized eyes because of the presence of hazy media, hypotony, a shallow anterior chamber, or an abnormal anterior segment architecture. Often, cyclodialysis clefts are not apparent on gonioscopic findings, even if disruption of the anterior segment structures is minimal, because the placement of a gonioscope in a hypotonous eye causes a significant indentation of the central cornea, and the convexity of the iris prevents visualization of the scleral spur or the cyclodialysis cleft. Gentile et al\textsuperscript{a} also found UBM to have a greater sensitivity in detecting cyclodialysis after closed globe injury.

Blunt trauma displaces aqueous into the peripheral parts of the anterior chamber and posteriorly toward the lens and vitreous. Earlier retrospective studies have recorded a frequent association of traumatic glaucoma with poor baseline visual acuity, hyphema, an angle recession of more than 180°, traumatic cataracts, dislocation of the lens, and iris injuries.\textsuperscript{3,6,7,10} However, in this prospective study, we found the incidence of iris injuries and cataract to be comparable in eyes with trauma alone and in those with traumatic glaucoma.

The features that were significantly associated with traumatic glaucoma—hyphema, an angle recession of more than 180°, displacement of the lens, and trabecular pigmentation—could all be attributed to ciliary body damage. These ciliary body injuries would lead to an inflammatory response not only at the site of injury, but also throughout the ciliary body and in the contiguous iris and trabecular meshwork. Resolution of uveal inflammation and injury is generally by a fibroblastic response, as seen in the iris or the choroid. Such a reparative process in the ciliary body would necessarily involve the adjoining trabecular meshwork, decreasing aqueous outflow and raising IOP.

A review of data from the US Eye Injury Register found increasing age, poor baseline visual acuity, angle recession, hyphema, and lens injury to be independent risk factors for developing posttraumatic glaucoma.\textsuperscript{6} Posttraumatic glaucoma was recorded at any time within 6 months of the injury, based on the physicians’ opinion alone. This last study, despite its large numbers, is limited by the absence of standardized criteria for diagnosing traumatic glaucoma. There are also limited data cited on IOP, the time when glaucoma was diagnosed, the extent of angle recession, and the use of glaucoma therapy in patients.

The higher prevalence of glaucoma after closed globe injury in our study patients was because our institution is a tertiary referral center and probably examines more severely traumatized eyes. A longer follow-up is necessary to see whether the IOP reduces with time or stays elevated and to see whether eyes in the closed globe injury group would develop glaucoma.

In conclusion, increased pigmentation of the angle on gonioscopic findings, a higher baseline IOP, and the absence of a cyclodialysis cleft on UBM or gonioscopic findings, along with the previously described features of hyphema, angle recession, and lens injury, can assist in the identification of eyes with closed globe injury, predisposed to chronic glaucoma.

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Correspondence: Viney Gupta, MD, Dr Rajendra Prasad Centre for Ophthalmic Sciences, All India Institute of Medical Sciences, Ansari Nagar, New Delhi 110029, India (gupta_v20032000@yahoo.com).

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REFERENCES


Ophthalmological Numismatics

Although Georg Prochaska (1749-1820) considered himself more of an anatomist than an ophthalmologist, he nevertheless achieved recognition as one of the better eye surgeons of his time, having performed no fewer than 3000 cataract operations. Born in Lipsitz (Moravia), he received his medical degree at the University of Vienna in 1776, where he returned in 1791 to serve as professor of anatomy, physiology, and ophthalmology after more than a decade in Prague as chair of anatomy.

In Czechoslovakia in 1949, a commemorative medal by Jan Tomas Fischer 70 mm in diameter was struck in bronze for the bicentennial of Prochaska’s birth. The obverse depicts a clothed bust facing left surrounded by the words “GEORGIUS PROCHASKA MORAVUS 1749-1820.” Within the curve at the lower right, it reads “FISCHER.” The reverse is inscribed in 4 parallel lines: “ARTIS MEDICAE/NOVAM LUCEM ET/FACIEM ELEGANTIOREM/ DABAT.” There is a floral design above and a staff of Aesculapius below, on either side of which reads “1749” and “1949.” Around the coin’s curve it reads “PROFESSOR PHYSIOLOGIAE ANATOMIAE MORB. OCULORUM UNIV. PRAGEN.”

Courtesy of: Jay M. Galst, MD, New York Medical College, and Peter van Alfen, PhD, American Numismatic Society.

Correspondence: Dr Galst, 30 E 60th St, New York, NY 10022.