Early Predictors of Traumatic Glaucoma After Closed Globe Injury

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

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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...
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 photography and UBM (UBM P-40; Paradigm Medical Industries, Salt Lake City, Utah) were performed by an experienced investigator. Intraocular pressure (IOP) was recorded at baseline and 5 days of the trauma, compared with none of the patients with a closed globe injury.

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.

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).
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 ($r^2=0.16$; $P=.44$).

The 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).

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

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**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 $\geq$3</td>
<td>7 (13)</td>
<td>17 (43)</td>
<td>.44</td>
</tr>
<tr>
<td>Angle recession $&gt;180^\circ$</td>
<td>6 (12)</td>
<td>14 (35)</td>
<td>.005</td>
</tr>
<tr>
<td>Angle recession of $360^\circ$</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.
of a large angle opening distance at 250 µm, an angle re-
cession area, and a wider distance from the scleral spur
to the iris root also predicted chronic glaucoma. On the
other hand, the presence of a cyclodialysis was found to
protect against the development of glaucoma (P < .001).

In the closed globe injury group, the mean IOP at the
3-month follow-up was 16.2 (3.2) mm Hg and at 6
months, 14.7 (3.8) mm Hg. In the traumatic glaucoma
group, the mean IOP was 20.4 (4.1) mm Hg and 18.7 (4.8)
mm Hg at 3 and 6 months, respectively. Only 5 patients
in the closed globe injury group required 1 topical anti-

glaucoma medication beyond 3 weeks, and all 5 pa-
tients had ended therapy by 5 weeks after trauma. The
mean number of topical medications at the start of therapy
in the traumatic glaucoma group was 2.5 (1.2) and was
reduced to 1.2 (1.0) at 3 months and 1.1 (0.2) at 12
months. Eleven eyes in the traumatic glaucoma group
required trabeculectomy for control of IOP. Light micros-
copy of the trabeculectomy specimens confirmed the pres-
ence of heavy pigmentation.

At the last follow-up in the closed injury group (mean,
12.0 [4.1] months; range, 9-18 months), 16 patients had
visual acuity of 6/60; 18 patients, of 6/9 to 6/60; and 10
patients, of less than 6/60. In the traumatic glaucoma
group, the respective numbers of patients were 5, 16, and

### Table 3. Ultrasonographic Biomicroscopy Findings
of the Anterior Chamber and Angle Structure in Eyes With
Closed Globe Injury With and Without Chronic Glaucoma

<table>
<thead>
<tr>
<th>Variables</th>
<th>Closed Globe Injury (n=52)</th>
<th>Traumatic Glaucoma (n=40)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior chamber depth, mm</td>
<td>3.0 (0.4)</td>
<td>3.2 (0.4)</td>
<td>.11</td>
</tr>
<tr>
<td>Anterior chamber angle, degrees</td>
<td>28.9 (7.9)</td>
<td>33.7 (1.1)</td>
<td>.004</td>
</tr>
<tr>
<td>Angle recess area, mm²</td>
<td>0.5 (0.2)</td>
<td>0.8 (0.5)</td>
<td>.003</td>
</tr>
<tr>
<td>Reattachment of the iris at the scleral spur, mm</td>
<td>0.4 (0.2)</td>
<td>0.6 (0.4)</td>
<td>.008</td>
</tr>
<tr>
<td>Angle opening distance</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>At 250 µm</td>
<td>0.5 (0.2)</td>
<td>0.6 (0.3)</td>
<td>.002</td>
</tr>
<tr>
<td>At 500 µm</td>
<td>0.6 (0.3)</td>
<td>0.8 (0.4)</td>
<td>.002</td>
</tr>
</tbody>
</table>

a Data are expressed as mean (SD).

### Table 4. Relative Risk of Developing a Chronic Glaucoma
Based on Clinical and UBM Features of All Eyes
After Closed Globe Injury

<table>
<thead>
<tr>
<th>Features Seen ≤4 wk After Injury</th>
<th>Relative Risk (95% CI)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trabecular pigmentation grade ≥3</td>
<td>20.8 (4.5-95.8)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>360° Angle recession</td>
<td>7.5 (1.9-28.6)</td>
<td>.002</td>
</tr>
<tr>
<td>Hyphema</td>
<td>6.9 (2.3-20.6)</td>
<td>.002</td>
</tr>
<tr>
<td>Lens displacement with cataract</td>
<td>3.5 (1.6-7.4)</td>
<td>.005</td>
</tr>
<tr>
<td>&gt;180° Angle recession</td>
<td>3.3 (2.5-25.5)</td>
<td>.004</td>
</tr>
<tr>
<td>Visual acuity &lt;6/60</td>
<td>2.5 (1.5-4.0)</td>
<td>.01</td>
</tr>
<tr>
<td>Baseline IOP</td>
<td>1.3 (1.1-1.5)</td>
<td>.001</td>
</tr>
<tr>
<td>Lens displacement alone</td>
<td>1.7 (1.1-2.7)</td>
<td>.02</td>
</tr>
<tr>
<td>Postsegment involvement</td>
<td>1.4 (0.8-2.3)</td>
<td>.34</td>
</tr>
<tr>
<td>UBM findings</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Angle opening distance at 250 µm</td>
<td>42.4 (4.1-431)</td>
<td>.002</td>
</tr>
<tr>
<td>Angle recess area</td>
<td>30.3 (3.8-238)</td>
<td>.001</td>
</tr>
<tr>
<td>Reattachment of the iris at the scleral spur</td>
<td>18.7 (2.3-150)</td>
<td>.006</td>
</tr>
<tr>
<td>Angle opening distance at 500 µm</td>
<td>16.8 (2.8-99.5)</td>
<td>.002</td>
</tr>
<tr>
<td>Anterior chamber depth</td>
<td>2.3 (0.7-7.5)</td>
<td>.14</td>
</tr>
<tr>
<td>Presence of cyclodialysis</td>
<td>0.2 (0.1-0.5)</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

Abbreviations: CI, confidence interval; IOP, intraocular pressure; UBM, ultrasonographic biomicroscopy.

17 (P = .01). Six patients in the closed globe injury group and 2 in the traumatic glaucoma group were lost to fol-
low-up at 6 months. On follow-up, 7 patients in the trau-
matic glaucoma group were found to have a retinal di-
alysis, compared with 6 patients in the closed globe injury group.

### COMMENT

Glaucoma after blunt trauma appears to have 2 peaks of
incidence, at less than 1 year and about 10 years after
trauma, 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 iden-
tify 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 care-
fully studied by means of gonioscopy and recorded using
360° goniphotography. The presence of heavy trabec-
ular pigmentation (grade ≥3) was a significant predic-
tor of chronic glaucoma, with a relative risk of 20.8. Ex-
tensive release of pigment into the eye during trauma
could clog the trabecular meshwork, and trabecular end-
othelial cells that phagocytize the pigment particles could
also block the meshwork, directly and through changes
induced at the meshwork. The degree of pigmentation
may be an indicator of the extent of anterior segment dam-
age, but pigmentation of at least grade 3 was present in
only 13% of eyes that did not develop a chronic glau-
coma, 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 condi-
tions such as pigmentary glaucoma or pseudoxefolia-

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tion and after the insertion of piggyback or sulcus-fixated intraocular lenses.3-13

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, but 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 al6 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,4,13 with incidence increasing in the presence of a hyphema.5 This may be because our study was designed to look for such changes, unlike the results reviewed retrospectively by Girkin et al.6 Long-term studies of eyes having an angle recession greater than 180° have shown that only 4% to 9% develop late chronic glaucoma.3,10,17

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 al18 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, displacement of the lens, and iris injuries.3,7,16 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.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, predisposing to chronic glaucoma.

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Financial Disclosure: None reported.

REFERENCES


Ophthalmological Numismatics

A lthough 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. OOCULARUM UNIV. PRAGEN.”

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