Traumatic myopia; an ultrasonographic and clinical study

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SUMMARY  Three patients with transient myopia following blunt injury are described with the aid of serial ultrasonography. The cause of refractive change is an increase in the anteroposterior thickness of the crystalline lens, which probably results from oedema of the ciliary body.

Myopia after concussional injury to the globe was first recognised by Kugel in 1870. Subsequent reports describing its transient nature, its association with hypotony, and the possible causes were reviewed by Duke-Elder. We report three cases of marked but transient myopia following blunt ocular injury and show that ultrasonography is useful in its assessment.

Patients and methods

Ocular examination of all patients was performed on the Haag-Streit 900 slit-lamp utilising the Goldmann indirect gonioscopy lens and applanation tonometer. Refraction of all patients was by one of the authors (JKS). Measurements in Tables 1, 2, and 3 were derived by the use of the Stortz A scan biometric ruler. Fixation was on a distant point during measurement to control accommodation. In addition case three was examined by the Kretz A scan, Bronson-Turner B scan, and the combined A and B Ocuscan.

Case reports

CASE 1  A 14-year-old boy ran into the branch of a tree and injured his left eye. When he was first seen, four

<table>
<thead>
<tr>
<th>Date</th>
<th>Axial Length (mm)</th>
<th>A/C depth (mm)</th>
<th>Lens thickness (mm)</th>
<th>Vit. body (mm)</th>
<th>IOP</th>
<th>Refraction and visual acuity (injured eye)</th>
</tr>
</thead>
<tbody>
<tr>
<td>R</td>
<td>L*</td>
<td>R</td>
<td>L</td>
<td>R</td>
<td>L</td>
<td>R</td>
</tr>
<tr>
<td>Day 3</td>
<td>23-9</td>
<td>23-3</td>
<td>4-0</td>
<td>2-6</td>
<td>3-5</td>
<td>4-0</td>
</tr>
<tr>
<td>Day 38</td>
<td>23-6</td>
<td>23-4</td>
<td>4-0</td>
<td>4-1</td>
<td>3-4</td>
<td>3-4</td>
</tr>
</tbody>
</table>

*Injured eye.
Each figure given in Tables 1–3 is the average of at least five readings of the Stortz A scan biometric ruler; mean readings given to one decimal point, but initial readings taken to two decimal points.
ND = not done. CF = counting fingers.

Table 2  Serial measurements on injured and contralateral eyes

<table>
<thead>
<tr>
<th>Date</th>
<th>Axial Length (mm)</th>
<th>A/C depth (mm)</th>
<th>Lens thickness (mm)</th>
<th>Vit. body (mm)</th>
<th>IOP</th>
<th>Refraction and visual acuity (injured eye)</th>
</tr>
</thead>
<tbody>
<tr>
<td>R</td>
<td>L*</td>
<td>R</td>
<td>L</td>
<td>R</td>
<td>L</td>
<td>R</td>
</tr>
<tr>
<td>Day 3</td>
<td>24-7</td>
<td>24-2</td>
<td>3-9</td>
<td>3-4</td>
<td>3-4</td>
<td>4-0</td>
</tr>
<tr>
<td>Day 17</td>
<td>24-6</td>
<td>24-5</td>
<td>4-0</td>
<td>4-0</td>
<td>3-3</td>
<td>3-4</td>
</tr>
<tr>
<td>Day 38</td>
<td>24-8</td>
<td>24-8</td>
<td>4-0</td>
<td>4-0</td>
<td>3-4</td>
<td>3-4</td>
</tr>
</tbody>
</table>

*Injured eye.
See footnotes to Table 1.
hours after the injury, the vision was reduced to hand movements in the left eye with correct projection of light. A total hyphaema was present, and the intraocular pressure was 24 mmHg. By day 3 most of the hyphaema had become absorbed and the intraocular pressure had fallen to 15 mmHg. The pupil reacted normally. The anterior chamber was now noted to be shallow, and the lens appeared thickened, with the iris bowed forward at 12 o’clock. Fundus examination at this stage required a myopic correction; a haemorrhage nasal to the disc was observed. Refraction revealed an emmetropic right eye, while the injured left eye improved from counting fingers to 6/9 with a correction of $-6.25/+1.50$ at $30^\circ$. Accommodation was fixed at approximately 20 cm. Two weeks after the injury the eye was still myopic, but at four weeks the myopia had resolved.

**CASE 2**

A 14-year-old boy was hit on the anterior inferotemporal sclera of the left eye by an airgun pellet fired from a pistol at a distance of approximately 30 feet (9 m). When he was first seen, 22 hours after the injury, the visual acuity was counting fingers with correct projection of light. A moderate hyphaema was present, and the intraocular pressure was 3 mmHg. The pupil was slightly eccentric and reacted sluggishly to light. Funduscopy on the following day revealed a normal disc, slight macular oedema, and an area of commotio retinae temporal to the posterior pole. A small amount of vitreous haemorrhage was present inferiorly. The visual acuity was 6/5 in the right eye but remained at counting fingers in the left. Refraction revealed an emmetropic right eye, while the injured left eye improved to 6/18 with a correction of $-5.50/+0.50$ at $170^\circ$. Accommodation appeared fixed at a distance of approximately 20 cm. Slit-lamp examination revealed a shallower anterior chamber on the left side and the appearance of a thickened lens. On day 3 gonioscopy of the left eye showed an open angle except for a small closed sector round the 3 o’clock meridian. Blood was present in the canal of Schlemm in the superotemporal quadrant. The intraocular pressure was 8 mmHg.

Two weeks after the injury the pupils were equal and normally reactive to light.

**CASE 3**

A 23-year-old man was first seen 15 hours after being struck on the right side of the face by a football. He complained of blurred vision in the right eye. The visual acuity was 6/24, which improved to 6/9 with pinhole. Periocular bruising, a traumatic mydriasis, and cells and flare in the anterior chamber were observed. The intraocular pressure was 20 mmHg. Four days later the vision was 6/60 improving to 6/18 with a pinhole. Five days after the injury a hyphaema was present, the intraocular pressure had fallen to 12 mmHg, and the anterior chamber was first noted as being shallow. Ophthalmoscopy and retinoscopy confirmed the presence of myopia of $-5.50/+0.75 \times 120^\circ$, though an accommodative amplitude of 10 diptres was recorded. Six days after the injury gonioscopy revealed angle recession in all quadrants and a cleft in the superotemporal quadrant, in which the iris was paretic. An ultrasonic B scan showed diffuse swelling of the uveal tract without detachment. Ten days after injury the anterior chamber deepened, the intraocular pressure rose, and the degree of myopia lessened. Funduscopy at this time revealed several choroidal tears in the superotemporal periphery and some macular oedema. Six weeks after injury the right eye was normotensive and had reverted to emmetropia, with a visual acuity of 6/9. Biometry, visual acuity, and intraocular pressure measurements of all three cases are shown in Tables 1–3.

**Discussion**

The three case reports clearly demonstrate that marked transient myopia can follow a variety of non-penetrating ocular injuries. The use of ultrasound to assess the intraocular dimensions allows a further evaluation of the possible explanations outlined by Duke-Elder for this refractive change. No lens opacities were observed which might have caused an index myopia. Lengthening of the globe in these
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subjects was ruled out by ultrasonography, though such a change may occur as the result of blunt injury to the young eye many years later. In all cases there was a large increase in anteroposterior thickness of the lens. As the vitreous cavity remained essentially unchanged in size, it was the forward movement of the anterior lens surface which resulted in shallowing of the anterior chamber. Myopia would result from (1) ciliary spasm, (2) ciliary body detachment, or (3) oedema of the ciliary body, all of which could result in an increase of lens size due to zonular slackening. Duke-Elder considered that spasm of accommodation was probably responsible for the majority of cases of traumatic myopia and it could be reversed by cycloplegia. However, case 2 was treated with cyclopentolate from admission to hospital, and it did not prevent the development of myopia, suggesting that spasm was not the mechanism here.

Transient myopia is known to occur in the presence of uveal effusion. This association is a well documented clinical entity occurring in posterior scleritis, after extensive photoacoagulation, and in Harada’s syndrome. It was postulated by Phelphs in a case report of two subjects who developed angle closure as a result of forward displacement of the iris-lens diaphragm that the mechanism may be an antero-lateral rotation of the ciliary body about its attachment to the scleral spur following the development of choroidal effusion. Our cases do not show a forward displacement of the iris lens diaphragm, but instead a marked anteroposterior thickening of the lens, suggesting that there has been no rotation of the ciliary body, but perhaps only ciliary body oedema. Transient myopia, in the absence of uveal effusion, follows the administration of some drugs. In these hypersensitivity reactions ciliary body oedema is thought to be the mechanism. Ciliary body oedema as a result of increased vascular permeability might occur as a direct result of trauma, perhaps caused by sympathetic paresis, since it has been demonstrated that most of the sympathetic nerve endings in the ciliary body terminate in the walls of blood vessels.

The observation of transient hypotony in two of our cases supports the hypothesis that ciliary body function was compromised temporally. In case 3 B scan ultrasonography clearly demonstrated oedema of the whole uveal tract without detachment, and that is further evidence of ciliary body oedema as the cause of transient traumatic myopia.

We considered that the frequency of traumatic myopia may be underestimated and therefore undertook a small prospective study of 10 patients admitted for hyphaema. We found no significant changes in refraction or intraocular dimension. Thus traumatic myopia is probably not common. However, it is possible that cases are missed, because visual acuity measurement, especially on an injured child’s eye, may be assumed to be inaccurate, and the presence of other signs, such as hyphaema, may be held responsible for diminished vision. The feature which should alert the observer to the possibility of traumatic myopia is shallowing of the anterior chamber in comparison with the uninjured eye.

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References


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