INTRODUCTION

Myopia after concussional injury to the globe was first recognized by Kugel in 1870. Subsequent reports describe its transient nature and association with hypotony. The possible causes of traumatic myopia were reviewed by Duke-Elder (1). Steele and Kraus (2, 3) reported marked but transient myopia immediately after blunt ocular injury.

The range of the myopia varies from 1 to 6 D. In most cases, the refraction returns to normal within a month. Its duration differs greatly from case to case, and varies from days or weeks to years, or may be permanent (1).

To our knowledge, none of the cases reported previously had bilateral ocular involvement. We describe a patient with transient bilateral myopia following blunt ocular trauma to the left eye.

Case Report

A 31-year-old man was struck on the left side of the face by a football. When he was first seen four hours after the injury, he complained of blurred vision in the left eye. The visual acuity was at the level of counting fingers but improved to 20/60 with a correction of -2.75/-1.00x120 in the injured left eye.

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Accommodation was fixed at a distance of approximately 20 cm in both eyes.

Two days after the trauma, the hyphema in the left eye was reduced. The pupil was still dilated moderately. Anterior chamber depth was normal. Intraocular pressure was 20 mmHg. Gonioscopy of the left eye showed no abnormality. Biometric measurements of both eyes are shown in Table I. There was no abnormality in the ocular compartments except for the increased lens thickness in the right eye immediately after the trauma. The myopia in the left eye resolved without cyclopentolate drops within 45 days. By this time the left pupil was moderately dilated and regular in shape. Intraocular pressure had fallen to 14 mmHg and refraction had reverted to emmetropia with a visual acuity of 20/25. Funduscopy revealed retina pigment epithelial irregularity in the area of commotio retina. The patient complained of inferior visual field restriction which was verified with automated perimetry.

**DISCUSSION**

This case shows that asymmetrical transient myopia can occur in both eyes after blunt ocular injuries. Traumatic myopia might result from ciliary spasm, ciliary body detachment, or edema of the ciliary body, all of which could result in an increased lens thickness due to zonular slackening. Duke-Elder (1) considered that spasm of accommodation was probably responsible for the majority of cases of traumatic myopia and could be reversed by cyclopia. The right eye in our case was successfully treated with cyclopentolate, suggesting that the ciliary spasm might be the causative mechanism here.

Transient myopia is known to occur in the presence of uveal effusion. This is a well-documented clinical association occurring in posterior scleritis (4, 5), after extensive photocoagulation (6), and Harada’s syndrome (7). Phelps (8) postulated 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.

Transient myopia in the absence of uveal effusion may be due to drug hypersensitivity reactions (9, 10) and supraciliary edema is thought to be another mechanism (11). Ciliary body edema caused by increased vascular permeability might occur as a direct result of trauma. It can be caused by sympathetic paresis, since most of the sympathetic nerve endings in the ciliary body terminate in the walls of blood vessels (12). The right eye of this patient had a high degree of myopia on manifest refraction and reverted to emmetropia after cyclopia. The diagnosis of spasm of accommodation was supported by the disappearance of pseudo-myopia after cyclopia. Transient myopia observed in his injured left eye supports the theory that ciliary body function may have been compromised temporarily. In both eyes there was no forward displacement of the iris lens diaphragm and anteroposterior thickening of the lens was only seen at two days in the right eye. These findings suggest that the mechanism of the left eye myopia was ciliary body edema caused by sympathetic paresis and increased vascular permeability.

**TABLE I - SERIAL BIOMETRIC AND TONOGRAPHIC MEASUREMENTS IN THE INJURED AND UNINJURED EYE**

<table>
<thead>
<tr>
<th>Date</th>
<th>Axial length (mm)</th>
<th>A/C depth (mm)</th>
<th>Lens thickness (mm)</th>
<th>IOP (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>R</td>
<td>L*</td>
<td>R</td>
<td>L*</td>
</tr>
<tr>
<td>Day 2</td>
<td>23.6</td>
<td>23.7</td>
<td>3.4</td>
<td>3.3</td>
</tr>
<tr>
<td>Day 14</td>
<td>23.8</td>
<td>23.6</td>
<td>3.5</td>
<td>3.4</td>
</tr>
<tr>
<td>Day 45</td>
<td>23.8</td>
<td>23.6</td>
<td>3.5</td>
<td>3.4</td>
</tr>
</tbody>
</table>

IOP = Intraocular pressure; A/C = Anterior chamber; * = Injured eye
To our knowledge, this is the first reported case developing bilateral transient myopia after the blunt trauma to only one eye. It may have been caused by reflex stimulation of the efferent autonomic system. We also found a significant correlation between the degree of traumatic transient myopia and the impairment of accommodation.

REFERENCES