Comment. Contrary to our original hypothesis, we did not find reduced levels of macular pigments in our light-sensitive subjects. Instead, we found that subjects with migraine had significantly higher levels of macular pigments compared with a control group. Although patients with blepharospasm had lower levels of macular pigments, this difference was not statistically significant.

It is not possible to tell from our experiments why migraineurs with chronic light sensitivity accumulate higher levels of macular pigment or whether this accumulation is clinically significant. It is possible that the macula accumulates these compounds in an effort to mitigate light sensitivity. The mechanisms that underlie carotenoid absorption, protein binding, transport, and storage are highly complex. There are numerous points in these processes where carotenoid metabolism could be affected in some light-sensitive individuals.

In conclusion, a deficiency of macular pigments does not appear to be involved in the pathogenesis of photophobia in patients with light sensitivity. Further research will need to be completed to elucidate the pathophysiology of photophobia.

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Author Contributions: All authors had full access to all of the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis.

Financial Disclosure: None reported.

Funding/Support: This work was supported by grant HL07744 from the National Institute of Diabetes and Digestive and Kidney Diseases (Mr Frandsen and Ms Llop), grant EY11600 from the National Institutes of Health (Dr Bernstein), and an unrestricted grant to the Department of Ophthalmology and Visual Sciences, University of Utah Health Sciences Center, from Research to Prevent Blindness.

Previous Presentation: This paper was presented at the 36th Annual Meeting of the North American Neuro-Ophthalmo-Society; March 9, 2010; Tucson, Arizona.

Table 1. Demographic Characteristics and Macular Pigment Optical Density Measurements in 21 Patients With Migraine and 21 Age- and Sex-Matched Control Subjects

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Patients With Migraine</th>
<th>Control Subjects</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean, y</td>
<td>52</td>
<td>54</td>
<td></td>
</tr>
<tr>
<td>Men, No.</td>
<td>4</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Women, No.</td>
<td>17</td>
<td>17</td>
<td></td>
</tr>
<tr>
<td>MPOD, mean (SD)</td>
<td>0.34 (0.15)</td>
<td>0.20 (0.13)</td>
<td>.006</td>
</tr>
</tbody>
</table>

Abbreviation: MPOD, macular pigment optical density.

Table 2. Demographic Characteristics and Macular Pigment Optical Density Measurements in 16 Patients With Blepharospasm and 16 Independent, Age- and Sex-Matched Control Subjects

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Patients With Blepharospasm</th>
<th>Control Subjects</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean, y</td>
<td>59</td>
<td>59</td>
<td></td>
</tr>
<tr>
<td>Men, No.</td>
<td>7</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>Women, No.</td>
<td>9</td>
<td>9</td>
<td></td>
</tr>
<tr>
<td>MPOD, mean (SD)</td>
<td>0.14 (0.12)</td>
<td>0.21 (0.13)</td>
<td>.16</td>
</tr>
</tbody>
</table>

Abbreviation: MPOD, macular pigment optical density.


Rapid Formation and Resolution of Cataracts Following Orthopedic Surgery for a Patient With Charcot-Marie-Tooth Disease

Rapid development and nonsurgical resolution of significant cataracts is extremely rare. Herein, we report an unusual case of bilateral, rapidly developing cataracts following orthopedic surgery for a patient with Charcot-Marie-Tooth disease (CMTD). The cataracts regressed within 45 days of surgery.

Report of a Case. A 45-year-old man was referred for a third opinion regarding vision loss. The vision loss began 2 days after a 4-hour, unremarkable foot surgery for a foot deformity due to type 2 CMTD, an autosomal dominant primary axonal neuropathy. The patient’s medical history was otherwise unremarkable. Preoperative 7-item basic metabolic panel and complete blood cell count findings were unremarkable. Postoperatively, his blood chem-
istry was unremarkable except for hypokalemia, with a potassium level of 3.2 mEq/L (to convert to millimoles per liter, multiply by 1.0). Standard operative medications were delivered, including the following: 2 g of cefazolin, 10 mL of ropivacaine hydrochloride, 0.75%, as a right lower extremity block, 5 mg of midazolam, and 100 µg of fentanyl citrate before induction; 60 mg of lidocaine, 200 mg of propofol, and 50 mg of rocuronium bromide during induction; and 2 mg of cefazolin and 4 mg of ondansetron hydrochloride toward the conclusion of the case. The case was without complication. Postoperatively, the patient's pain was controlled with hydromorphone hydrochloride via a patient-controlled analgesia pump. During his hospital stay, he received nasal mupirocin, docusate sodium, and potassium chloride. The patient was discharged on postoperative day 1 with oxycodone hydrochloride and his usual home medications: α-lipoic acid, 400 mg/d; ascorbic acid, 500 mg/d; coenzyme Q10, 600 mg/d; vitamin B complex daily; and a multivitamin. On ophthalmic examination 29 days after surgery, the patient's best-corrected visual acuity was 20/40 OU while wearing contact lenses with the following prescription: −1.00 + 0.25 × 180 OD and −1.25 + 0.50 × 173 OS. Intraocular pressure measured 15 mm Hg OU. Slitlamp examination results were unremarkable except for significant bilateral posterior cortical cataracts and a Mittendorf dot in the left eye (Figure, A and B). Fundus examination, optical coherence tomography, and fluorescein angiography findings were unremarkable. Follow-up was arranged for cataract extraction evaluation. The patient was seen 16 days later in follow-up, and he described resolution of the vision loss after 1 week of self-initiating the use of an eyedrop containing N-acetylcarnosine, 1%, dosed at 2 drops 4 times daily. His best-corrected visual acuity improved to 20/30 OD and 20/25 OS. Examination revealed complete resolution of the cataracts (Figure, C and D).

Comment. The mechanism of rapid formation of posterior cortical cataracts following orthopedic surgery for CMTD in this case is unknown. Recent eye examination prior to surgery showed no cataracts. His operative and perioperative medications were not found to be linked to cataract formation by PubMed search. Type 2 CMTD is commonly associated with a connexin gap junction mutation as well as several other mutations. Connexins have long been studied in cataractogenesis and are known to be involved in metabolite, ion, and water transport between lens fibers.¹ There are also reports of cataract formation in dynamin-mutated type 2 CMTD,¹,² but there are no reports to our knowledge of a spontaneously regressing cataract as seen in our case. A literature search

Figure. External photographs of the right (A) and left (B) eyes showing cataracts at the initial visit, and photographs of the right (C) and left (D) eyes showing complete resolution of cataracts at follow-up. Arrows indicate the Mittendorf dot in the left eye.
of the other known genes for type 2 CMTD did not reveal any association with cataracts. Reports of spontaneously resolving cataracts have been noted following intraocular surgery. These were thought to arise from several potential mechanisms, one being impaired sodium-potassium adenosine triphosphatase channels with electrolyte imbalance. The feathering sutureal cataracts noted in these studies are similar to those found in our patient. This finding, along with gap junction and dynamin studies, suggests a brief osmotic imbalance as a potential cause. Other spontaneously resolving cataracts have been previously reported, usually associated with significant metabolic disease or intralenticular trauma. These reports offer suggestions of potential resolving mechanisms involving lens epithelial growth and reestablishment of ionic balance. The importance of the patient’s antioxidant home medications and N-acetylcarnosine eye drops in his cataract resolution is unknown. As described by Toh et al, further independent research of clinical effectiveness is still needed to validate research claims of N-acetylcarnosine that have significant conflicts of interest in humans.

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Financial Disclosure: None reported.


COMMENTS AND OPINIONS

The Use of Endocapsular Equator Rings for Preventing Posterior Capsule Opacification

After carefully reading the article by Dr Hara and colleagues, I would offer the following comments and observations and encourage the authors to provide more information. It is still controversial whether the intraocular lens material affects the incidence of posterior capsular opacification. Nevertheless, information regarding the material of the intraocular lens implanted in the treated group was not provided. In addition, it is only stated, but not proved, that the posterior capsule does not touch the lens.

Second, the size of the closed endocapsular ring was fixed, having an outer diameter of 9.5 mm. The authors pointed out that the 9.5-mm ring “fit all eyes.” Indeed, it is well known that there are some manufacturers of intraocular lenses that increase the size of the lenses for myopic patients. In those cases, a greater optical zone, and thus a greater overall size of the lens, allows for a more stable fixation within the capsular bag. It is equally well known that capsular tension rings come in different sizes, with the larger diameter lenses being used in myopic eyes. Given that the ring is “only” 9.5 mm in diameter, it might not be well centered in myopic eyes. Should the ring become decentered, given that the optic size is smaller (only 5 mm), patients may even encounter visual symptoms related to the edge of the lens that is visible in the pupillary area, perhaps in scotopic conditions.

Last, there is a need for “direct observation” of the ring when engaging the intraocular lens’ haptics into their inner groove. This limits its use in patients with narrow pupils or when there is a progressive intraoperative pupil constriction.

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Financial Disclosure: None reported.


In reply

Based on clinical slitlamp observations, the intraocular lens (IOL) optics do not touch the posterior capsule in the eye with the ring. Therefore, we can conclude that the IOL optics do not play any part in preventing posterior capsular opacification (PCO). The main purpose of our study was to determine the effect of the endocapsular equator ring (E-ring) on preventing PCO. We did not need to perform a postoperative posterior capsulotomy in any eye with an E-ring, which is a definitive result. Because of this, we did not investigate the materials and shapes of the IOLs in-