in her son. She had diffuse corneal guttata and a slightly up-drawn pupil in both eyes (Figure 2). Intraocular pressure was normal. Indirect ophthalmoscopy revealed a normal retina in the right eye and several peripheral inferior chorioretinal scars in the left eye. The nature of the chorioretinal scars could not be resolved. She had neurologic signs and symptoms suggestive of multiple sclerosis. There were no other family members with similar ocular abnormalities.

**Comment.** This family presents a unique combination of corneal findings that do not fit into known diseases or syndromes. There is some overlap with Peters anomaly, which is usually a sporadic condition characterized by central corneal opacity and defects in the corneal endothelium, Descemet membrane, and posterior stroma. In our 2 patients, the corneal opacities were superior and peripheral, and there were no visible defects in Descemet membrane. Furthermore, the disease trait appeared to be inherited in an autosomal dominant fashion. Axenfeld-Rieger anomaly is also autosomal dominant and includes posterior embryotoxon, iris stromal hypoplasia, or polycoria; neither had glaucoma. Their corneal opacities were localized and did not progress in surface or density, unlike those of corneal dystrophies or storage diseases. Cornea guttata, such as that in our patients, has not been reported with any of the previously mentioned conditions, to our knowledge.

Optical sector iridectomy is a simple and safe procedure that can improve visual outcome and avoid penetrating keratoplasty. This procedure was highly effective in our younger patient with corectopia and peripheral corneal opacities.

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**Acute Anterior Uveitis and Corneal Edema Associated With Travoprost**

Prostaglandin analogues have dramatically changed many clinicians’ approach to glaucoma treatment. The combination of potency, once-daily dosing, and relatively few side effects make these appealing agents. The most frequently reported adverse reaction with travoprost is ocular hyperemia (occurring with a frequency of 35%–50% in populations studied). Decreased visual acuity, eye discomfort, foreign body sensation, pain, and pruritis are reported to occur with a frequency of 5% to 10%. Herein we report a case of acute iritis and corneal edema after only 5 doses of travoprost.

**Report of a Case.** A 79-year-old white man had a history of atrial fibrillation and bladder carcinoma. The medications he was taking included digoxin, warfarin sodium, and verapamil. In 1983 he underwent planned extracapsular cataract extraction without an implant lens in the left eye. This was followed 1 year later by phacoemulsification with a posterior chamber intraocular lens placed in the right eye and a retinal detachment repair performed in his left eye. He was diagnosed as having open-angle glaucoma in both eyes in 1983. The glaucoma was well controlled medically over nearly 2 decades. Mild corneal guttata were first noted in 1993.

We saw him on January 2, 2002, for an eye examination. The visual acuity was 20/40 OD and 20/230 OS (without aphakic correction). Findings on slitlamp examination revealed clear corneas with 1+ guttata OU. The anterior chambers were quiet. The intraocular pressure was 20 mm Hg OD and 23 mm Hg OS while receiving a therapeutic regimen of 0.5% timolol maleate and bri-
monidine. When presented with the choice to try a new potent medication once a day instead of his current 2 medications, the patient elected to try travoprost once daily beginning on January 3. Two days later, he called complaining of mild redness, discomfort, and blurriness. He was advised of an adjustment period with this eyedrop and told to call back if symptoms did not improve. His symptoms worsened, and by January 8, his visual acuity had dropped to 20/100 OD. Slitlamp examination findings included +2 conjunctival hyperemia, +2+ central corneal edema, and diffuse corneal folds in both eyes. There was 1-2+ “cell and flare” in the anterior chamber in both eyes. The intraocular pressure was 11 mm Hg OD and 13 mm Hg OS. Treatment with travoprost was discontinued and loteprednol etabonate therapy was begun every 6 hours in both eyes. By January 17, the patient’s discomfort resolved and visual acuity had improved to 20/50 OD, the corneal edema was clearing, and the anterior chambers were quiet. Treatment with timolol and brimonidine was restarted, and the loteprednol was tapered and stopped. By February 28 the corneal folds had completely cleared. Central corneal pachymetry measurements on that date were 587 µm OD and 541 µm OS. The endothelial cell count was 661 cells/mm² OD and 708 cells/mm² OS.

Comment. Inflammation has been associated previously with prostaglandin analogues. Latanoprost in particular has been reported to cause uveitis with corneal and macular edema.2-4 Travoprost has been a relatively recent addition to the ocular hypotensive lipid family, and since its introduction there have been relatively few reports of adverse effects. Herein we reported a case of acute anterior uveitis and clinically significant corneal edema associated with the use of travoprost. However, further studies are necessary to confirm this association.

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An Iris Coloboma Preventing Pigmentary Glaucoma

Pigment dispersion syndrome and pigmentary glaucoma result from iridodomal friction causing disruption of the iris epithelium and deposition of iris pigment on anterior segment structures.3 The classic triad of findings includes Kruekenberg spindle, iris transillumination defects, and trabecular meshwork pigment. A posterior bowing of the iris that underlies the iridozonular contact and dispersion of pigment is often noted in these eyes. Usually seen in myopic patients, this concave iris configuration is believed to be due to reverse pupillary block, with the increased axial length allowing for a higher volume or pressure in the anterior chamber compared with the posterior chamber.2-3 Laser iridotomy has been proposed as a therapeutic modality for pigment dispersion syndrome and pigmentary glaucoma by equalizing the pressure between the anterior and posterior chambers, in an analogous manner to the treatment of narrow-angle glaucoma, thereby eliminating the reverse pupillary block.2 This results in a flat iris configuration with reduction or elimination of the iridozonular contact.2-4

Report of a Case. A 48-year-old man was referred for management of elevated intraocular pressure in his right eye. His history was significant for congenital bilateral iris defects. There was no history of ocular trauma. On examination, his visual acuity was 20/25 OU with moderate myopic correction. Pressures were 22 mm Hg OD and 23 mm Hg OS. Biomicroscopy demonstrated a Kruekenberg spindle in the right eye only (Figure 1). Transillumination defects were present in the right eye only (Figure 2). Gonioscopy of the right eye demonstrated an open angle with dense, uniform trabecular pigment; gonioscopy of the left eye showed an open angle without significant pigment (Figure 3). In addition, gonioscopy demonstrated a posterior iris concavity in the right eye and relatively flat iris configuration in the left eye. On funduscopy examination, asymmetry of the optic nerves was noted with a 0.3 cup-disc ratio in the right eye and a 0.1 cup-disc ratio in the left eye. The right eye was diagnosed as having pigmentary glaucoma and a partial iris coloboma. The left eye was diagnosed as having mild ocular hypertension, without pigment dispersion or glaucoma, in association with a complete iris coloboma. Treatment of the right eye was initiated with latanoprost, followed by laser trabeculoplasty, with the pressure stabilizing below 19 mm Hg.

Comment. Pigment dispersion was prevented in the left eye of this patient because the iris coloboma was complete, reaching beyond the edge of the lens, and effectively functioned as would an iridectomy. Therefore, reverse pupillary block was prevented, a flat iris configuration was maintained, and there was no iridozonular contact. In the right eye, since the incomplete iris coloboma did not reach beyond the maximum curvature of the lens, reverse pupillary block remained. As a result, a posterior iris concavity persisted with associated pigment dispersion and glaucoma. Of note, the partial coloboma may have resulted in greater pigment dispersion than would no coloboma at all, as evidenced by the iris concavity and transillumination defects being relatively marked because of the thinner iris tissue in the

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