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.1-5 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.4 This procedure was highly effective in our younger patient with corectopia and peripheral corneal opacities.

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Figure 2. Photograph of the right eye of the patient in case 2 reveals superior corneal opacity and mild corectopia.

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%.1 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 20/250 OS (without aphakic correction). Findings on slitlamp examination revealed clear corneas with 1-2+ 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-
edema. Travoprost has been a relatively recent addition to the ocular hypertension therapeutic modality for pigment dispersion syndrome and pigmentary glaucoma. A posterior bowing of the iris 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. 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. This results in a flat iris configuration with reduction or elimination of the iridozonular contact.

An Iris Coloboma Preventing Pigmentary Glaucoma

Pigment dispersion syndrome and pigmentary glaucoma result from iridozonular friction causing disruption of the iris epithelium and deposition of iris pigment on anterior segment structures. The classic triad of findings includes Krukenberg 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. 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. This results in a flat iris configuration with reduction or elimination of the iridozonular contact.

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Comment. Inflammation has been associated previously with prostaglandin analogues. Latanoprost in particular has been reported to cause uveitis with corneal and macular edema. Travoprost has been a relatively recent addition to the ocular hypertensive 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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