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. Neither patient had glaucoma. One had cataracts and the other had a normal lens. Axenfeld-Rieger anomaly is characterized by central corneal opacities and iris stromal hypoplasia; neither patient had this finding.

optic 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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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 20/230 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-
Pigment dispersion syndrome and pigmentary glaucoma result from iridodugal 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.

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

An Iris Coloboma Preventing Pigmentary Glaucoma

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