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 eye drop 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. Loteprednol 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 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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Comment. 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.1 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.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. Bio- microscopy demonstrated a Krukenberg spindle in the right eye only. A partial iris coloboma was present in the right eye and a complete iris coloboma was present in the left eye (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 funduscopic 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.
region of the partial coloboma. This case serves as a natural prospective example, with its own control, of the potential effectiveness of iridotomy treatment in reducing or preventing pigment dispersion.

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