no recurrence 2 years after treatment; the patient awaits corneal regrafting.

Comment. Although HHV-8 causes neoplastic disease (Kaposi sarcoma, multicentric Castleman disease, and primary effusion lymphoma in an immunocompromised host), HHV-8–related clinical manifestations are not well defined.[2,3,5] To our knowledge, this is the first report of corneal endotheliitis positive for HHV-8 leading to graft failure in an immunocompetent patient.

It is uncertain whether HHV-8 was the cause because polymerase chain reaction detection does not necessarily mean that HHV-8 caused the clinical manifestations of corneal endotheliitis.

Because anti–HHV-8 therapy was unavailable, graft failure occurred with only antiallograft rejection therapy. Expression of HHV-8 DNA in the aqueous was high in the active inflammatory phase (before and 3 weeks after treatment) but not in the stable phase (3 months after treatment). Polymerase chain reaction results for HHV-1 through HHV-7 at 3 weeks and 3 months after treatment indicated no positivity for HHVs without antiviral therapy throughout the therapy course. Because other HHVs[6] cause corneal endotheliitis, HHV-8 may be a candidate. These findings suggest that HHV-8 infection may play a role in corneal endotheliitis leading to graft failure. Investigations about specific anti–HHV-8 therapy or the latency of HHV-8 are needed.

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The 10-Year Incidence of Glaucoma Among Patients With Treated and Untreated Ocular Hypertension

The Ocular Hypertension Treatment Study has shown that medical treatment of ocular hypertension (OHT) reduces the incidence of primary open-angle glaucoma (POAG) at 5 years by more than 50%.[1] The greatest absolute reduction in incidence occurs among those at highest risk based on baseline age, intraocular pressure, central corneal thickness, vertical cup-disc ratio, and pattern standard deviation. In clinical practice, most patients with OHT are followed up for longer than 5 years.[1-3] To help clinicians and patients make decisions about the benefit of early medical treatment and the frequency of examinations, we have calculated the 10-year incidence of POAG under 2 scenarios: treated, ie, medical treatment for 10 years, and untreated, ie, no medical treatment for 10 years.

Methods. The Ocular Hypertension Treatment Study randomized 1636 participants to either medical treatment or observation. After a mean follow-up of 7.5 years, observation participants were then also offered medication. Both groups were followed up for an additional 5.5 years. To estimate the incidence of POAG for the scenario of treated for 10 years, we used data from the medication group. To estimate the incidence of POAG for the scenario of untreated for 10 years, we used untreated data from the observation group. For each scenario, a parametric proportional hazards model was fit while including the 5 aforementioned baseline predictors.[2,3] The baseline hazard function in each model used 4-knot restricted cubic splines, with the knots chosen as the 5th, 33rd, 67th, and 95th percentiles of the uncensored survival times.[4] The 95% CIs were obtained using bootstrap methods from 1000 bootstrap runs resampling the original data with replacement. All analyses were performed using the R version 2.9.2 statistical package (R Foundation for Statistical Computing, Vienna, Austria).

Results. The 10-year incidence of POAG in participants with OHT is reported by baseline levels of risk (tertiles) and by ethnicity (**Table**). Medical treatment for 10 years reduced the incidence of POAG by about 50% at all 3 tertiles of risk. The absolute reduction was greatest in the highest tertile of risk (23%, from 42% to 19%) and lowest in the lowest tertile of risk (3%, from 7% to 4%). Within each tertile of risk, incidences of POAG among African American participants and those who are not African American were similar.

Comment. Topical medication for OHT reduces the 10-year incidence of POAG by 50% at all 3 tertiles of risk for the entire group as well as African American participants.[5] The absolute reduction in incidence of POAG was greatest in the group at the highest tertile of baseline risk. The incidence of POAG in participants with OHT appears to be roughly linear during the first 10 years. It is unknown whether this finding can be extrapolated to 20 years of follow-up or more.

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Table. The 10-Year Incidence of Primary Open-angle Glaucoma Among Participants With Treated and Untreated Ocular Hypertension and Their Baseline Central Corneal Thickness and Vertical Cup-Disc Ratio

	Untreated for 10 y			Treated for 10 y		
Ethnicity	POAG, % (95% CI)	CCT, Mean (SD), µm	VCDR, Mean (SD)	POAG, % (95% CI)	CCT, Mean (SD), µm	VCDR, Mean (SD)
Lowest risk[a]						
All	7 (4-11)	608 (27)	0.27 (0.17)	4 (2-5)	607 (29)	0.30 (0.18)
Not African American	7 (4-11)	609 (27)	0.27 (0.17)	3 (2-5)	607 (30)	0.28 (0.18)
African American	8 (5-12)	599 (30)	0.31 (0.17)	5 (3-8)	606 (23)	0.40 (0.16)
Moderate risk[b]						
All	18 (13-26)	573 (23)	0.39 (0.19)	8 (6-10)	574 (21)	0.41 (0.18)
Not African American	18 (13-26)	575 (22)	0.39 (0.19)	8 (6-10)	575 (21)	0.40 (0.19)
African American	19 (13-29)	563 (25)	0.40 (0.18)	9 (6-12)	572 (23)	0.44 (0.18)
High risk[c]						
All	42 (32-54)	541 (26)	0.49 (0.16)	19 (15-23)	534 (27)	0.46 (0.19)
Not African American	40 (31-52)	546 (25)	0.48 (0.16)	18 (14-22)	538 (23)	0.43 (0.19)
African American	45 (34-59)	535 (27)	0.51 (0.15)	21 (16-26)	528 (31)	0.50 (0.18)

Abbreviations: CCT, central corneal thickness; POAG, primary open-angle glaucoma; VCDR, vertical cup-disc ratio.
[a] Baseline risk of developing POAG was less than 6%.
[b] Baseline risk of developing POAG was 6% to 13%.
[c] Baseline risk of developing POAG was more than 13%.

We have reported that African American individuals have a higher overall incidence of POAG than do individuals who are not African American, but they have similar incidences of POAG within the same tertile of risk. This seeming contradiction is explained by the fact that African American individuals are overrepresented in the highest tertile of risk and underrepresented in the lowest tertile of risk.[6]

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Progressive Synechial Angle Closure From an Enlarging Soemmering Ring

Angle closure in pseudophakic eyes is uncommon and its mechanism varies. Inflammation with posterior synechiae, zonular disruption with vitreous prolapse, and ciliary block with aqueous misdirection may predispose to angle closure and elevated intraocular pressure.

Proliferation of the remaining lenticular epithelial cells after cataract extraction may form a circumferential structure at the level of the lens (Soemmering ring). This typically benign structure has been reported to cause pupillary block, leading to angle closure.[1] We describe a patient who had progressive synechial angle closure without pupillary block, due to an enlarging Soemmering ring after phacoemulsification.

Report of a Case. An 80-year-old man had intermittent blurred vision and colored halos in the left eye. He had a history of exfoliative glaucoma and phacoemulsification with an anterior chamber intraocular lens in the right eye and a posterior chamber intraocular lens in the left eye in 1999. Initial consultation in 2008 revealed a narrow anterior chamber angle with approximately 210° peripheral anterior synechiae (PAS) and a planar iris configuration in the left eye. Intraocular pressure was 20 mm Hg OS, and initial ultrasound biomicroscopy of the left eye confirmed the gonioscopic findings, a lack of pupillary block, and a prominent Soemmering ring, which was larger in the areas of PAS (**Figure**, A and B). Prophylactic laser iridotomy was performed.

Two years later, he returned for reassessment. Intraocular pressure was 45 mm Hg OS. Slitlamp examination revealed a clear cornea, a moderately deep anterior chamber, and a well-centered posterior chamber intraocular lens in the left eye. There was a patent superior iridotomy and exfoliation material at the pupillary mar-

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