

Table. All Consented Patients With Type 1 ROP in at Least 1 Eye

Birth Weight, g	Race	Consented Patients With ROP, No. (N = 2320)	Patients Developing Type 1 ROP, No. (%)
<750	Non-African American	623	186 (29.9)
	African American	315	36 (11.4)
750-999	Non-African American	726	140 (19.3)
	African American	233	18 (7.7)
≥1000	Non-African American	331	25 (7.6)
	African American	92	1 (1.1)
Total	Non-African American	1680	351 (20.9)
	African American	640	55 (8.6)

Abbreviation: ROP, retinopathy of prematurity.

focus on infants particularly likely to develop ROP. However, any study of the effects of β -blocker therapy must address the fragility of the patients to be tested and possible systemic and ocular adverse effects. Nevertheless, if topical β -blockers prove to be effective in preventing some cases of ROP, this opens the door for a more individualized approach to prevention of the disease, eg, using β -adrenergic receptor polymorphisms to guide ROP management.

William V. Good, MD
 Robert J. Hardy, PhD
 David K. Wallace, MD, MPH
 Don Bremer, MD
 David L. Rogers, MD
 R. Michael Siatkowski, MD
 Inge De Becker, MD, FRCSC
 C. Gail Summers, MD
 Rae Fellows, MEd
 Betty Tung, MS
 Earl A. Palmer, MD

Author Affiliations: Smith-Kettlewell Eye Research Institute, San Francisco, California (Dr Good); School of Public Health, University of Texas Health Science Center at Houston (Dr Hardy and Ms Tung); Duke Eye Center, Durham, North Carolina (Dr Wallace). The Ohio State University College of Medicine (Drs Bremer and Rogers) and Department of Ophthalmology, Nationwide Children's Hospital (Drs Bremer and Rogers and Ms Fellows), Columbus; Department of Ophthalmology, Dean McGee Eye Institute, University of Oklahoma, Oklahoma City (Dr Siatkowski); Departments of Ophthalmology (Drs De Becker and Summers) and Pediatrics (Dr Summers), University of Minnesota, Minneapolis; and Casey Eye Institute, Oregon Health and Science University, Portland (Dr Palmer).

Correspondence: Dr Good, Smith-Kettlewell Eye Research Institute, 2318 Fillmore St, San Francisco, CA 94115 (good@ski.org).

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Epithelial Downgrowth After Type 1 Boston Keratoprosthesis Manifesting as Tractional Retinal Detachment and Epiretinal Membrane

Type 1 Boston keratoprosthesis (KPro) is a viable treatment option for corneal disease at high risk for graft failure with traditional penetrating keratoplasty. Postoperative complications of Boston KPro include retroprosthetic membrane, glaucoma, sterile vitritis, infectious endophthalmitis, corneal melt, extrusion, and retinal detachment.¹ To our knowledge, we report the first case of epithelial downgrowth (ED) of the posterior segment after Boston KPro placement.

Report of a Case. A 52-year-old man with a history of penetrating ocular injury to his right eye had open globe repair and cataract extraction in 1974, placement of a secondary anterior chamber intraocular lens in 1992, 2 failed penetrating keratoplasty procedures in 2004 and 2008, Baerveldt glaucoma tube implantation in 2005, astigmatic keratotomy in 2007, and, most recently, intraocular lens removal and type 1 Boston KPro placement in 2010. Histologic examination of the failed corneal graft excised at the time of KPro placement did not demonstrate ED. One week after KPro placement, visual acuity was 20/40 OD. Two months after KPro placement, he had pain and photophobia in the right eye.

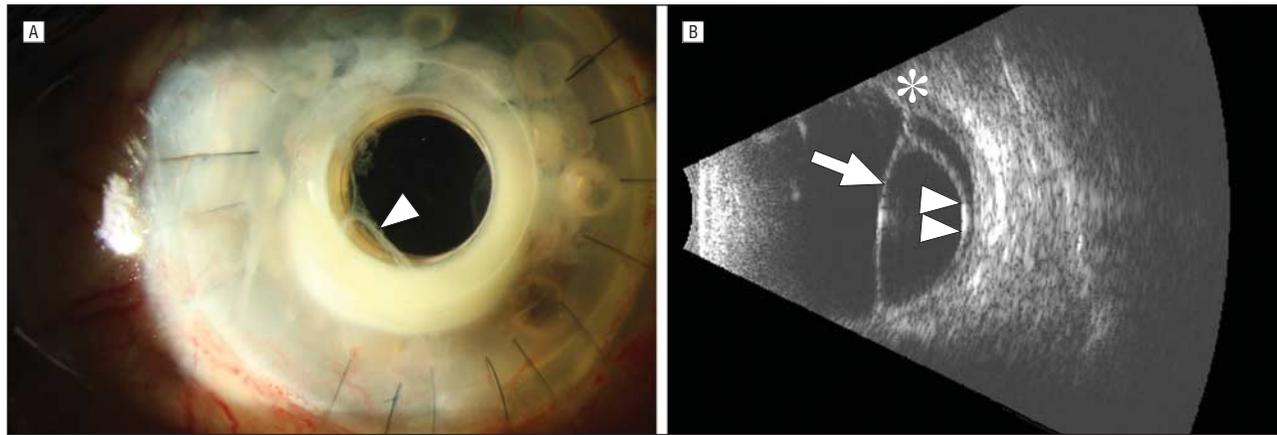


Figure 1. Clinical presentation of epithelial downgrowth. A, Slitlamp examination reveals an intraprosthetic membrane (arrowhead) posterior to the front plate, anterior to the back plate, and extending from the 6-o'clock position to the 10-o'clock position prior to vitrectomy. B, Diagnostic B-scan ultrasonography exhibited an anterior vitreous band (arrow) and tractional retinal detachment due to epiretinal membrane from epithelial downgrowth (arrowheads) and choroidal detachment (asterisk). The tractional retinal detachment induced by the epiretinal membrane was most prominent anteriorly but extended across the posterior pole.

At his visit to us, visual acuity was 8/200 OD and 20/20 OS. No afferent pupillary defect was identified. Intraocular pressure was soft to palpation OD and 14 mm Hg OS. Slitlamp examination revealed an intraprosthetic membrane (**Figure 1 A**). B-scan ultrasonography revealed anterior vitreous bands, tractional retinal detachment (TRD), and a small choroidal detachment (**Figure 1B**). Review of B-scan ultrasonographic images performed 2 months previously, before KPro placement, showed a normal posterior segment. A 23-gauge pars plana vitrectomy was performed to repair the TRD. Pars plana vitrectomy revealed a dense anterior vitreous band contiguous with a TRD and epiretinal membrane (ERM) extending across the macula. Extensive membrane peeling was performed, followed by fluid-air exchange, endolaser, and silicone oil injection. A large ERM specimen from the macula was sent for histopathologic evaluation. Microscopic evaluation of the ERM revealed mucosal epithelium containing goblet cells on hematoxylin-eosin, periodic acid-Schiff base, and Masson-Trichrome staining, consistent with ED (**Figure 2**). Immunohistochemical analysis with cytokeratin showed positive staining, revealing epithelial cells lining one side of the ERM (**Figure 2**). At the patient's 2-month follow-up, slitlamp examination revealed recurrent intraprosthetic membrane and flat retina by indirect ophthalmoscopy and optical coherence tomography.

Comment. Epithelial downgrowth of the posterior segment is rare but can manifest within months after eye surgery. McDonnell et al² reported ED occurring 3 months after ruptured globe repair, lensectomy, and pars plana vitrectomy, wherein TRD and ERM were found to consist of nonkeratinized, stratified squamous epithelium. Our case demonstrates a similar rapid progression of ED into the posterior segment after surgery. B-scan ultrasonography documented a normal-appearing posterior segment before KPro placement. Two months after KPro placement, B-scan ultrasonography showed the large TRD due to ED.

Epithelial downgrowth after KPro placement has been a subject of historical interest. Girard³ observed that ED only occurred in early KPro models previous to 1972. The Boston KPro was approved by the US Food and Drug Ad-

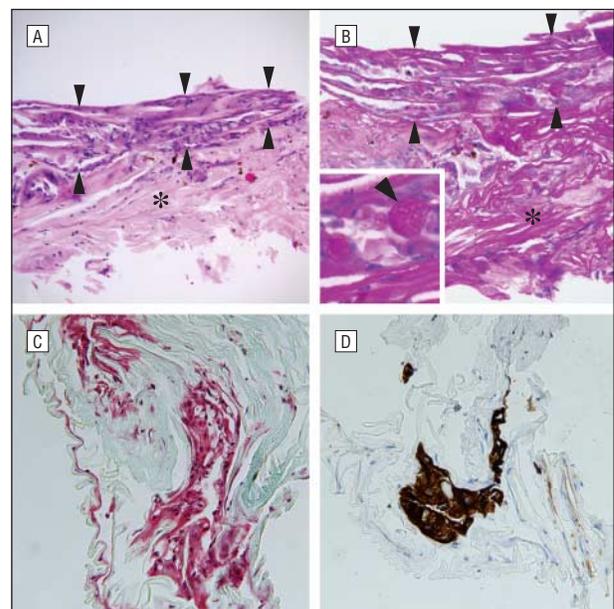


Figure 2. Histopathologic photomicrographs. A, Histologic examination demonstrates epithelial cells (arrowheads) within the fibrous tissue of the epiretinal membrane (asterisk) displayed in a sheetlike configuration (hematoxylin-eosin, original magnification $\times 200$). B, Foci of nonpigmented epithelium (arrowheads) are present within the fibrous tissue of the epiretinal membrane (asterisk) (periodic acid-Schiff, original magnification $\times 100$). Inset, Goblet cell (arrowhead) with mucin at high magnification (periodic acid-Schiff, original magnification $\times 400$). C, Noncollagenous epithelium stains red, sparing mucin-containing goblet cells (Masson trichrome, original magnification $\times 200$). D, Cytokeratin stains positive for epiretinal membrane (immunohistochemical stain [Dako Corp], original magnification $\times 200$).

ministration in 1992 and has demonstrated ED in only 2 cases. Both cases had ED localized to the anterior chamber due to full-thickness tissue melting at the KPro-cornea junction from ocular cicatricial pemphigoid.⁴ To our knowledge, there have been no published reports of ED after Boston KPro placement involving the posterior segment. In our case, histologic review confirmed that ED of the posterior segment occurred after KPro placement.

Histopathologic findings of ED typically consist of nonkeratinized, stratified squamous or conjunctival epithelium extending over the posterior cornea or anterior sur-

face of the iris from an anterior wound site. In phakic or pseudophakic eyes, the posterior lens capsule serves as a barrier preventing further advancement of the epithelium into deeper structures of the eye. Epithelial downgrowth of the posterior segment can occur when this barrier has been disrupted (aphakia, lens luxation or subluxation, iridodialysis) or bypassed (trauma, scleral buckle intrusion).⁵

Many treatments for ED have been described, including cryotherapy, radiation, alcohol, steroids, antimetabolites such as fluorouracil, and complex surgical procedures, each with varying rates of success and recurrence.^{6,7} Although our case demonstrates successful surgical repair of the TRD, the likelihood of ED disease progression remains high. Further study is needed to better understand the etiology, diagnosis, and management of ED in this clinical setting.

Brett P. Bielory, MD
David Jacobs, MD
Eduardo Alfonso, MD
Victor L. Perez, MD
Sander R. Dubovy, MD
Audina Berrocal, MD

Author Affiliations: Bascom Palmer Eye Institute, Miller School of Medicine, University of Miami (Drs Bielory, Jacobs, Alfonso, Perez, Dubovy, and Berrocal) and Ocular Pathology Laboratory, Florida Lions Eye Bank (Dr Dubovy), Miami.

Correspondence: Dr Berrocal, Bascom Palmer Eye Institute, Miller School of Medicine, University of Miami, 900 NW 17th St, Miami, FL 33136 (aberrocal@med.miami.edu).

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Bilateral Uveal Effusion and Angle-Closure Glaucoma Associated With Bupropion Use

Bupropion hydrochloride, an aminoketone antidepressant, is a dopamine reuptake inhibitor with norepinephrine and nicotinic acetylcholine receptor antagonist actions.¹ We report the first case to our

knowledge of uveal effusion and bilateral angle-closure glaucoma associated with bupropion use.

Report of a Case. A 40-year-old healthy white woman with a history of depression had bilateral blurry vision starting the morning of her visit. Her ocular history was significant only for myopia. She reported excellent vision with -6.00-diopter sphere (DS) contact lenses prior to her visit. Her only medications were ibuprofen, last used 1 month prior, and bupropion hydrochloride, 100 mg 3 times a day, which she started 2 weeks prior. Ten years earlier, she had taken an uncertain dose of bupropion for an unknown duration without incident.

At her initial visit, visual acuity was 20/200 OD and 20/400 OS while wearing -6.00-DS contact lenses. Intraocular pressure was 35 mm Hg OU, both pupils reacted to light, and slitlamp examination revealed mild corneal edema and shallow anterior chambers bilaterally (**Figure 1** A and B). Gonioscopy revealed appositional angle closure bilaterally (Figure 1 C and D). Fundus examination showed healthy nerves with a cup-disc ratio of 0.2 OU. A diagnosis of bilateral angle-closure glaucoma was made. Treatment was started in the emergency room with 1 dose of each of the following: pilocarpine hydrochloride, 1%, eye drops; timolol maleate, 2%/dorzolamide hydrochloride, 0.5%, eye drops; brimonidine tartrate, 0.15%, eye drops; latanoprost, 0.005%, eye drops; and oral acetazolamide, 500 mg.

The next day in the Glaucoma Service, intraocular pressure was 22 mm Hg OU. Ultrasound biomicroscopy showed bilateral choroidal effusions causing shallow angles (Figure 1 E and F), and B-scan ultrasonography showed diffuse 360° of suprachoroidal hypoechogenicity consistent with uveal effusions (Figure 1 G and H). Autorefractometry demonstrated a myopic shift to -16.00 DS OU, supporting a diagnosis of bilateral angle-closure glaucoma with myopic shift secondary to uveal effusions. Bupropion, acetazolamide, and pilocarpine were discontinued, and treatments with prednisolone acetate and cyclopentolate hydrochloride eye drops were started.

Two days later, her visual acuity improved to 20/70 OD and 20/100 OS with contact lenses, her intraocular pressures normalized, and her angles were open. All treatments with eye drops were stopped. At 1 week, her examination findings normalized (**Figure 2** A-D) and repeat ultrasound biomicroscopy (Figure 2 E and F) and B-scan ultrasonography (Figure 2 G and H) showed complete resolution of the uveal effusions. One month later, her visual acuity was 20/20 OU while wearing -6.00-DS contact lenses. She started treatment with escitalopram oxalate for depression. Nine months later, her examination findings remained stable without effusions.

Comment. Drug-induced uveal effusions with resultant bilateral angle-closure glaucoma and myopic shift are uncommon but have been reported with a variety of medications, most notably sulfa-based medications such as topiramate.² The mechanism for drug-induced uveal effusions is unclear. Some cases appear to be dose dependent as lower doses of the inciting medication may not