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).  

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. 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.

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. 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 1A and B). Gonioscopy revealed appositional angle closure bilaterally (Figure 1C 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 1E and F), and B-scan ultrasonography showed diffuse 360° of suprachoroidal hypoechogenicity consistent with uveal effusions (Figure 1G and H). Autorefracti on 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 2A-D) and repeat ultrasound biomicroscopy (Figure 2E and F) and B-scan ultrasonography (Figure 2G 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. 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
trigger recurrence. If our patient were on a lower dose previously, this may explain why effusions did not develop during her prior exposure.

Bupropion, a non–sulfa-based dopamine reuptake inhibitor with a 10-hour half-life, is a different class of medication than previously associated with this syndrome. This

Figure 1. Findings at the initial visit. Shallow anterior chambers with mild corneal edema are seen in the right (A) and left (B) eyes. Gonioscopy shows appositional angle closure in the right (C) and left (D) eyes. Ultrasound biomicroscopy shows ciliary body swelling with angle closure in the right (E) and left (F) eyes. B-scan ultrasonography shows diffuse uveal effusions in the right (G) and left (H) eyes.

Figure 2. Findings 1 week after the patient’s initial visit. Deeper anterior chambers are seen in the right (A) and left (B) eyes. Gonioscopy shows open angles in the right (C) and left (D) eyes. Ultrasound biomicroscopy shows open angles and resolution of ciliary body swelling in the right (E) and left (F) eyes. B-scan ultrasonography shows resolution of uveal effusions in the right (G) and left (H) eyes.
case suggests that choroidal vasodilation could play an inciting role as increased levels of dopamine have been shown to cause choroidal vasodilation. The time course of effusion development in this case is similar to that with topiramate. This suggests that if bupropion use were causative, one of its major active metabolites, hydroxybupropion or theobupropion (both with half-lives similar to that of topiramate), may be responsible. Both metabolites inhibit dopamine and norepinephrine reuptake; thus, norepinephrine and serotonin reuptake inhibitor. Bilateral effusions have been reported with venlafaxine hydrochloride, a norepinephrine and serotonin reuptake inhibitor.

Bupropion is a common medication, and it is unclear why other cases have not been reported. If bupropion use were causative in this case, the absence of other similar reports may reflect underreporting; alternatively, this patient may harbor a rare, private polymorphism that causes bupropion or one of its metabolites to become a particularly potent choroidal vasodilator.

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Small Dose of Rituximab for Graves Orbitopathy: New Insights Into the Mechanism of Action

Rituximab has been used in the treatment of several autoimmune diseases. Preliminary studies from our laboratory, recently confirmed, have shown that 2 cycles of 1000 mg of rituximab induced peripheral CD20+ cell depletion and significant clinical improvement of active Graves orbitopathy (GO). We report an unexpectedly rapid therapeutic effect of 100 mg of rituximab observed in 3 patients as early as 1 to 7 days after therapy, with persistent inactivation of inflammation and total depletion of CD20+ and CD19+ B cells. Immunohistochemistry of orbital tissues from patients treated with rituximab has shown early recruitment of type 2 macrophages, which may be involved in rituximab-induced phagocytosis of B-cell targets in orbital tissues.

Methods. Patients with GO underwent rituximab infusion after premedication with acetaminophen, chlorpheniramine maleate, and 100 mg of hydrocortisone sodium succinate. Peripheral cell subpopulation analysis was performed at baseline, at the time of the acute reaction (60 minutes), and weekly thereafter by flow cytometry (Figure 1 C). Orbital tissues after treatment with 100 mg of rituximab, treatment with steroids, and no treat-