brain and regional lymph nodes. Generally, involvement of the surgical margin by tumor cells on histopathologic examination is an indication to repeated surgical resection with wider margins, especially in immunosuppressed patients.

In summary, we described a 56-year-old patient with liver transplantation who developed aggressive conjunctival squamous cell carcinoma that invaded the brain and led to death despite orbital exenteration. Physicians should be aware that conjunctival squamous cell carcinoma in five patients: the 1998 Pan American Lecture. Ophthalmic Plast Reconstr Surg. 1999;15:153-160.


Angle-Closure Glaucoma Associated With Ciliary Body Detachment in Patients Using Topiramate

Topiramate (Topamax; Ortho-McNeil Pharmaceutical, Raritan, NJ) is a sulfamate-substituted monosaccharide that is used primarily as an antiepileptic medication and also demonstrates preliminary efficacy in the treatment of bipolar disorders and pain control of migraine. Recently, cases of acute angle-closure glaucoma (AACG) presumably associated with topiramate have been reported.1,2 However, the causative role of topiramate in producing angle closure in these cases was confounded by concomitant use of other drugs, notably selective serotonin reuptake inhibitors (SSRIs), which have also been reported to cause AACG.

We describe 2 cases of bilateral AACG associated with topiramate use and with ultrasound biomicroscopic signs of ciliochoroidal effusion. The patients were not using any other drugs previously reported to be associated with glaucoma.

Support was provided by the Eye Tumor Research Foundation, Philadelphia, Pa (Drs C. Shields and J. Shields), the Paul Kayser International Award in Retina Research, Houston Tex (Dr J. Shields), the Macula Foundation, New York, NY (Dr C. Shields), and the Noel T. and Sara Simmons Endowment for Ophthalmic Pathology, Wills Eye Hospital (Dr Eagle).

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Report of Cases. Case 1. A 44-year-old male psychiatrist was examined in the emergency department complaining of severe ocular pain and decreased visual acuity in both eyes. He had had pain in both eyes for 3 days before our examination. He denied any remarkable ocular history or any symptoms related to previous episodes of angle closure. His medications included topiramate (which he started 5 days before initial examination and used for 3 days), niacin, glutamine, and chromium supplement.

On initial examination, the patient’s visual acuity was 20/400 OU, and it improved with pinhole examination to 20/100 OD and 20/80 OS. Slitlamp examination disclosed similar findings in both eyes with diffuse conjunctival hyperemia, corneal edema, and a shallow anterior chamber peripherally and centrally. The pupils were nonreactive to light, and the iris contour was slightly convex. Intraocular pressure measured by Goldmann applanation tonometry was 60 mm Hg OU. The funduscopic examination showed normal optic discs and no signs of choroidal effusion. On gonioscopy, there was appositional angle closure in both eyes.

A diagnosis of bilateral angle-closure glaucoma was made and the patient was given 0.5% timolol maleate, dorzolamide hydrochloride, brimonidine tartrate, bimatoprost, and 500 mg of oral acetazolamide. An ultrasound biomicroscopic examination was performed and demonstrated closed angle, shallow anterior chamber, and suprachoroidal effusion with forward displacement of the ciliary body in both eyes (Figure 1). A diagnosis of secondary angle-closure glaucoma associated with ciliary body detachment was made and the patient was treated with 1% atropine and 1% prednisolone acetate in both eyes.

Twenty-four hours after initial examination, the patient was more comfortable. The anterior chamber had deepened centrally but was still shallow in the periphery of both eyes. The intraocular pressure was 26 mm Hg OU. On the fourth day of follow-up, the visual acuity was 20/25 OU with a -2-diopter (D) myopic correction. The anterior chamber was deep and quiet in both eyes and the intraocular pressure was 14 mm Hg OD and 17 mm Hg OS. The pupils were normally reactive, and gonioscopic examination showed open angles throughout 360°. All medications were discontinued, and 1 month later the visual acuity was 20/20 OU with intraocular pressure of 21 mm Hg. With A-scan ultrasound, the axial lengths were 24.57 mm OD and 25.38 mm OS. The anterior chamber depths were 3.56 mm OD and 3.79 mm OS.

Case 2. A 42-year-old woman came to the emergency department complaining of blurred vision in both eyes for 1 day. She had started using topiramate 10 days earlier for chronic headache. Her ocular history included only a myopic refractive error of −3.50 D OD and −5.25 D OS. On initial examination, her visual acuity was 20/80 OU, and it improved with pinhole examination to 20/25 OD and 20/30 OS. Slitlamp examination showed clear corneas with a shallow anterior chamber in both eyes. The intraocular pressure was 32 mm Hg OU. Gonioscopy showed appositional angle closure in both eyes. Funduscopic findings were normal.

An ultrasound biomicroscopic examination was performed and showed a shallow anterior chamber and suprachoroidal effusion with forward displacement of the ciliary body in both eyes (Figure 2). A diagnosis of bilateral angle closure secondary to ciliary body detachment was made. The topiramate was discontinued and the patient was treated with timolol, brimonidine, and 500 mg of oral acetazolamide. We administered 1% atropine to the right eye only. On the following day, the intraocular pressure was reduced to 16 mm Hg OD and 18 mm Hg OS, and the anterior chamber deepened in both eyes. With slitlamp biomicroscopy, we determined that the anterior chamber was deeper in the right eye (grade 4) than in the left eye (grade 2). Two weeks after discontinuation of all medications, the visual acuity was 20/30 OD with intraocular pressure of 18 mm Hg OD and 2 mm Hg OS. With A-scan ultrasound, we found that the axial length was 24.96 mm OD and 24.97 mm OS. The anterior chamber depth was 3.79 mm OD and 3.68 mm OS.

Comment. Topiramate is part of a new generation of antiepileptic drugs that have been increasingly prescribed throughout the world. A syndrome consisting of acute myopia associated with angle-closure glaucoma has been reported in some patients using this drug. Sankar et al reported on 2 cases of uveal effusion and angle-closure glaucoma presumably associated with topiramate use. In one
Although SSRI drugs have not also been reported in the literature after paroxetine administration have closure to ciliary body edema and FORWARD DISPLACEMENT OF THE LENS. Although these mechanisms seem to be related to the antiepileptic properties of topiramate, the mechanism of choroidal effusion associated with this drug remains unclear. Fluid movement in choroidal effusion could be related to drug-induced membrane potential changes. However, the finding of effusion in only a few patients taking this drug suggests a possible idiosyncratic reaction.

In conclusion, a history of topiramate use should be sought in patients presenting with bilateral AACG. In suspected cases, an ultrasound biomicroscopic examination can help to confirm the diagnosis.

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Uvea effusion is the presence of uveal effusion in both cases returned to normal values in 24 to 48 hours.

In another report, Rhee et al described a case of bilateral AACG in a patient using topiramate. They attributed the mechanism of angle closure to ciliary body edema and forward displacement of the lens. Although the authors did not comment on the presence of uveal effusion, review of the patient’s photographs suggests that supraciliary fluid was present. Also in this case, the patient was using an SSRI, paroxetine. Cases of bilateral AACG after paroxetine administration have also been reported in the literature.14 Although SSRI drugs have not been implicated in causing uveal effusion or ciliary body edema, a possible contribution of these drugs in causing or aggravating the glaucoma in those reported cases cannot be excluded.

Both of our patients were receiving topiramate, and neither one had used an SSRI. Of their medications, topiramate was the only drug possibly associated with glaucoma. Ultrasound biomicroscopic examination in both cases showed supraciliary choroidal effusion. We believe that fluid accumulated in the supraciliary space with ciliary body detachment is the main factor producing the anterior rotation of the ciliary body. This rotation pushes the iris anteriorly and closes the angle. Secondary angle-closure glaucoma related to ciliary body detachment has been well described in the literature.1,8 As the mechanism of angle closure does not involve pupillary block, peripheral iridectomy and miotics are not useful in the treatment of this condition. In fact, miotics can possibly aggravate it by pushing the lens-iris diaphragm further forward. Our patients were treated solely on the basis of topical and oral hypotensive medications associated with cycloplegia and topiramate discontinuation. The intraocular pressure in both cases returned to normal values in 24 to 48 hours.

Figure 2. Ultrasound biomicroscopic image of the left eye showing ciliary body detachment from fluid accumulated in the supraciliary space (arrow). The ciliary body is anteriorly positioned and the angle is closed.

**Figure 2.** Ultrasound biomicroscopic image of the left eye showing ciliary body detachment from fluid accumulated in the supraciliary space (arrow). The ciliary body is anteriorly positioned and the angle is closed.
Epithelial Downgrowth Following Insertion of an Ahmed Glaucoma Implant

Epithelial downgrowth has been reported as a complication of various forms of ocular surgery, including trabeculectomy, cataract surgery, and penetrating keratoplasty. It can result in visual compromise and is difficult to treat, often requiring aggressive surgical means. To the best of our knowledge, epithelial downgrowth has not been reported as a complication of a Seton implant. We report a case of histopathologically documented epithelial downgrowth following insertion of an Ahmed glaucoma implant.

Report of a Case. A 68-year-old white woman was first seen in our office in October 1991. Her ocular history was significant for myopic degeneration with posterior staphyloma and primary open-angle glaucoma in both eyes treated with twice-daily 0.5% timolol maleate. Corrected visual acuity was 20/30 OD and 20/40 OS. She had undergone extracapsular cataract extraction and left aphakic approximately 30 years earlier. She required 180° laser trabeculoplasty in her right eye in 1996, followed by a second 180° trabeculoplasty in 1997. She subsequently required a trabeculectomy with mitomycin in the right eye in November 1997, followed by an Ahmed glaucoma implant in April 1998 for uncontrolled intraocular pressure from 30 to 40 mm Hg. Her visual acuity had decreased to counting fingers due to a combination of myopic degeneration, glaucoma, and corneal edema from elevated intraocular pressure. Her visual acuity subsequently improved to 20/80 following surgery. Four months after surgery, a retrocorneal membrane was first noted. Six months after implantation, rapid progression of the retrocorneal membrane was noted. In April 1999, 1 year after implantation, she developed band keratopathy, which decreased her visual acuity to counting fingers, and she underwent EDTA chelation therapy in June 1999. Due to progressive corneal opacification resulting in visual acuity of hand motions, the patient required penetrating keratoplasty in January 2000. An iris biopsy involving the retrocorneal membrane was also performed for diagnostic purposes. Histopathologic examination of the corneal specimen showed a bilayer of cells on the endothelium with round nuclei typical of epithelial cells (Figure 1). The iris specimen revealed obvious epithelial downgrowth (Figure 2). In July 2000, the retrocorneal membrane recurred. No further diagnostic or therapeutic intervention has been attempted thus far. The patient’s intraocular pressure has remained controlled at around 12 mm Hg, but her visual acuity has remained at hand motions because of a recurrence of...