All patients were followed for six months. Mean FT \pm SD was 408 \pm 77 μ m at baseline, 425 \pm 83 μ m at two months, 453 \pm 97 μ m at three months, and 454 \pm 101 μ m at six months (P = .172, Friedman test). A decrease by \geq 20% in FT was observed in none of the eyes at both three and six months (Table 2).

Mean Early Treatment Diabetic Retinopathy Study (ETDRS) letter scores \pm SD were 59 \pm 15 (20/80) at baseline, 58 \pm 12 (20/80) at one month, 60 \pm 14 (20/63) at two months, 59 \pm 16 (20/80) at three months, 57 \pm 15 (20/80) at six months (P = .398, Friedman test). VA improved by \geq 1 line in four eyes (36.4%) at three months and in one eye (9.1%) at six months (Table 3). Distribution of changes in VA (improvement or impairment of VA by one or two ETDRS lines) from baseline to months one, two, three, and six were not statistically significant (P = .406, Chi-square test). No clinically significant complication was encountered in any eye.

In this small, retrospective series, there was no change in VA and FT in the short-term after intravitreal bevacizumab for DME in previously vitrectomized eyes. This may be attributable to rapid clearance of intravitreal bevacizumab from the vitreous cavity and thus insufficient sustained therapeutic levels in vitrectomized eyes. It may also be attributable to individual systemic factors that may affect macular edema such as type and glycemic control of diabetes, age, blood pressure, serum lipid levels, and nephropathy. In addition, it is not known whether or not continued injections for six to 12 months might have improved the outcomes. Further studies with larger series of patients, eventually with measurement of the bevacizumab levels in the vitrectomized vitreous cavity, are required for a more reliable conclusion.

THE AUTHORS INDICATE NO FINANCIAL SUPPORT OR FInancial conflict of interest. Involved in design and conduct of study (A.Y.); collection of data (A.Y., B.A.); management, analysis and interpretation of data (A.Y.); preparation of the manuscript (A.Y., B.A., F.H.); and review and approval of the manuscript (A.Y., B.A., F.H.). Informed consent was obtained in accordance with Helsinki Declaration from all patients. No Institutional Review Board approval was required for this study.

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Peripheral Retinopathy and Maculopathy in High-dose Tamoxifen Therapy

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PURPOSE: To describe the clinical, angiographic, and optical coherence tomography (OCT) features of highdose tamoxifen retinopathy in three male patients. DESIGN: Observational case series.

METHODS: A review of history, clinical examination, and findings on fluorescein angiography (FA) and optical coherence tomography (OCT) was conducted.

RESULTS: Three male patients receiving high-dose tamoxifen therapy sought treatment for vision loss and a crystalline maculopathy. Crystalline deposits were noted in the peripheral retina of two patients. All the patients showed macular leakage by FA, but cystoid macular edema (CME) on OCT was detected in two patients. Inner retinal hyperreflective deposits were identified by OCT in all the patients.

CONCLUSIONS: High-dose tamoxifen therapy may result in peripheral crystalline retinopathy in addition to perifoveal opacities. Angiographic evidence of macular edema may not unanimously correlate with presence of CME on OCT in these cases. (Am J Ophthalmol 2007;144: 126–128. © 2007 by Elsevier Inc. All rights reserved.)

T AMOXIFEN IS AN ORAL ANTIESTROGEN DRUG MOST commonly used in low dosages (20 mg daily) for the adjuvant treatment of breast cancer. High-dose tamoxifen chemotherapy also may be used for the treatment of brain malignancies. Retinal toxicity with tamoxifen therapy is dose related, and cumulative doses of 100 g or more have been reported to cause refractile perifoveal opacities, and in more severe cases, cystoid macular edema (CME) and retinal pigment epithelial abnormalities.¹ The pathophysiology may be the result of the formation of retinal deposits and lipid– drug complexes that lead to axonal degeneration.² This article describes the clinical, fluorescein angiography (FA), and optical coherence tomography (OCT) findings of tamoxifen retinopathy in three male patients with brain malignancy receiving high-dose tamoxifen chemotherapy (200 mg/day).

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• PATIENT 1: A 32-year-old man who received a cumulative dose of 876 g tamoxifen after surgical resection, irradiation, and chemotherapy of astrocytoma of the brain. Snellen visual acuity was 20/60 in the right eye and 20/80 in the left eye. Fundus examination disclosed annular crystalline maculopathy in both eyes (Figure 1, Top). Peripheral retinal examination revealed white, superficial crystalline deposits (Figure 1, Middle). FA showed marked late leakage consistent with florid macular edema. OCT imaging confirmed severe CME associated with numerous hyperreflective inner retinal deposits in both eyes (Figure 1, Bottom).

• PATIENT 2: A 45-year-old man who received a cumulative dose of 219 g tamoxifen after irradiation and chemother-



FIGURE 1. Peripheral tamoxifen retinopathy and maculopathy with cystoid macular edema (CME). (Top) Color fundus photographs of (left) the right eye and (right) the left eye showing tamoxifen retinopathy with annular refractile deposits. (Middle) Peripheral color fundus photograph of the left eye showing white refractile deposits in the temporal periphery (arrow). (Bottom) Horizontal optical coherence tomography (OCT) scan confirming severe CME in both eyes. Numerous inner retinal hyperreflective deposits are noted.



FIGURE 2. Peripheral tamoxifen retinopathy and maculopathy without cystoid macular edema (CME). (Top) Peripheral color fundus photographs of the left eye demonstrating crystalline deposits in the temporal periphery (arrows). (Middle) Latephase fluorescein angiography (FA) image showing mild fluorescein leakage in the left eye. (Bottom) Horizontal optical coherence tomography (OCT) scan denoting multiple hyperreflective inner retinal deposits without evidence of CME.

apy of glioblastoma of the brain. Visual acuity was 20/80 in the right eye and 20/40 in the left eye. Fundus examination showed crystalline maculopathy in both eyes. Superficial crystalline deposits were scattered in the periphery in both eyes (Figure 2, Top). Late leakage consistent with mild CME was observed in the left eye by FA (Figure 2, Middle). OCT failed to detect CME or macular thickening, but showed multiple hyperreflective inner retinal deposits (Figure 2, Bottom).

• PATIENT 3: A 60-year-old man who received a cumulative dose of 730 g tamoxifen after surgical resection and irradiation of mixed oligoastrocytoma of the brain. Visual acuity was 20/50 in the right eye and 20/70 in the left eye.

Fundus examination showed annular crystalline maculopathy in both eyes. Additional deposits were not present in the periphery. The FA showed late leakage consistent with CME in both eyes. Presence of macular edema and numerous hyperreflective inner retinal deposits were noted on OCT in both eyes.

The presented occurrence of peripheral retinal crystals associated with tamoxifen maculopathy is a novel clinical finding. Histopathologic studies have identified tamoxifen crystals in the ciliary body, and crystals also may be detected in the lens and cornea.^{2,3} The predilection of crystalline deposition on the macula in part may relate to its greater blood supply. However, we suspect that peripheral crystals may not be an uncommon finding in patients with high-dose tamoxifen retinopathy if sought after with meticulous peripheral examination.

One of the patients in the series showed late leakage on FA without CME on OCT. Gualino and associates⁴ recently described two patients with tamoxifen retinopathy who did not have signs of macular edema or retinal thickening on OCT, but showed changes consistent with atrophy. Although our cases failed to delineate macular atrophy on OCT with clarity, the macular edema seen in two of our patients is a well-documented association of severe tamoxifen maculopathy.^{1,5} In these cases, OCT may prove to be an integral tool in the assessment of CME and its response to possible treatment. Furthermore, OCT imaging confirmed the location of the crystalline deposits to the inner retina, which has been described previously with histopathologic analysis.²

Although most patients with tamoxifen retinopathy are women with breast cancer, this diagnosis should be considered in male patients with crystalline maculopathy and a history of tamoxifen therapy. Peripheral retinal examination may reveal crystalline deposition in the mid peripheral and anterior retina. FA and OCT imaging may aid in the diagnosis of angiographic and clinical CME, respectively.

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Comparison of Fibrin Glue and Sutures for Conjunctival Closure in Pars Plana Vitrectomy

Ruth Mentens and Peter Stalmans

PURPOSE: Evaluating whether fibrin glue causes less postoperative pain, discomfort, and sick leave in conjunctival closure following 20-gauge pars plana vitrectomy than sutures. DESIGN: Retrospective study.

METHODS: A questionnaire was sent in 2006 to 506 patients who underwent 20-gauge pars plana vitrectomy in 2004 at University Hospital, Leuven, Belgium. Postoperative pain, eye discomfort, and sick leave duration were determined.

RESULTS: The patients in the glue group had a shorter duration of eye redness (P = .0471), eye discomfort (P = .0376), and ointment use (P = .0105). The patients in the glue group used less ointment (P = .0038), had shorter sick leave with independent workers (P = .0292), and experienced less pain on the first postoperative day after vitrectomy without cerclage (P = .0340).

CONCLUSIONS: Fibrin glue causes less postoperative pain, discomfort, and sick leave for closure of conjunctival wounds in 20-gauge pars plana vitrectomy than sutures, and therefore, appears in our hands to be a better alternative to sutures. (Am J Ophthalmol 2007;144: 128–131. © 2007 by Elsevier Inc. All rights reserved.)

T ISSUCOL (BAXTER; BIOSCIENCE BIOSURGERY, DEERfield, Illinois, USA) is used in closing conjunctival wounds in glaucoma surgery,¹ strabismus,² and intraocular lens implantation.³ One study investigated 130 procedures with conjunctival wounds of which 20-gauge pars plana vitrectomies were performed.⁴ All of these studies concluded that the use of fibrin glue caused significantly less postoperative pain, less discomfort, and shortened surgery time. None of the publications showed postoperative adverse or allergic reactions, bacterial infections, inflammation, or delayed healing.¹⁻⁶

This retrospective study determines whether fibrin glue causes less postoperative pain, discomfort, and sick leave in conjunctival closure after pars plana vitrectomy than sutures.

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