proved to be genetically identical by DNA sequencing methods.[7]

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Optical Coherence Tomography Findings in Tamoxifen Retinopathy

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PURPOSE: To describe optical coherence tomography (OCT) findings in two cases of typical tamoxifen retinopathy.

DESIGN: Observational cases report.

METHODS: Two patients with tamoxifen retinopathy were imaged with fluorescein angiography and OCT 3.

RESULTS: Fluorescein angiography showed foveolar hyperfluorescence. OCT revealed a foveolar cystoid space with focal disruption of the photoreceptor line. There was no evidence of macular edema or thickening.

CONCLUSIONS: In both cases, OCT findings are not consistent with previous descriptions of tamoxifen retinopathy, based on fundus examination and fluorescein angiography, which include a description of macular edema. This new imaging suggests that tamoxifen maculopathy may include a foveolar cystoid space different from macular edema. (Am J Ophthalmol 2005;140: 757–758. © 2005 by Elsevier Inc. All rights reserved.)

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OCULAR TOXICITY FROM TAMOXIFEN IS CHARACTERized by bilateral superficial white refractile deposits located in the inner layers of the retina and punctate gray lesions in the outer retina and retinal pigment epithelium.[1] Loss of central vision may occur and is usually thought to be a complication of cystoid macular edema, observed on fundus examination or fluorescein angiography, or both.[1,2] This report describes the optical coherence tomography (OCT) appearance of two patients with typical tamoxifen retinopathy.

A 64-year-old woman had taken a cumulative dose of 34.2 g of tamoxifen after breast cancer surgery. Snellen visual acuity was 20/50 in the right eye and 20/40 in the left eye. Fundus examination disclosed tiny refractile deposits in the macula of both eyes, typical of tamoxifen retinopathy (Figure 1, top). Fluorescein angiography showed early foveolar hyperfluorescence (Figure 1, middle), which remained stable throughout the entire sequence. There was neither pooling nor leakage of dye on late frames. OCT was performed using Stratus OCT 3

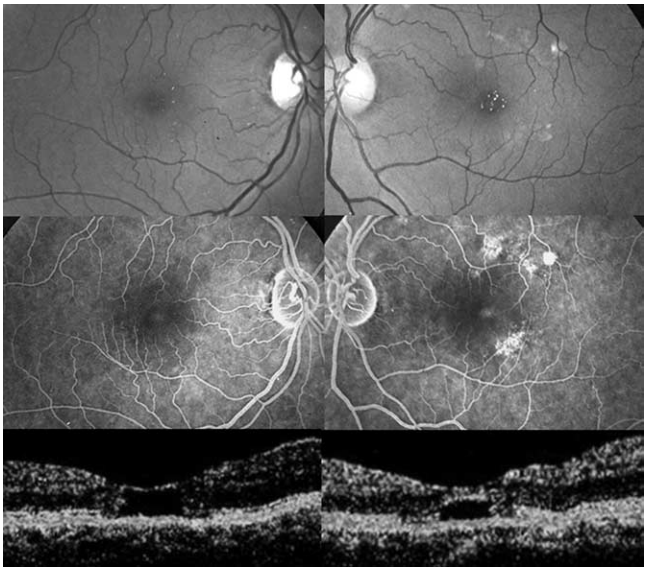

FIGURE 1. Tamoxifen retinopathy. Case 1. (Top) Red-free photograph of the right eye (left) and the left eye (right) of a 64-year-old woman showing tamoxifen retinopathy with tiny refractile crystalline deposits in the macula. (Middle) Fluorescein angiography shows foveolar hyperfluorescence. Several patchy areas of hyperfluorescence are also present around the macula. (Bottom) A horizontal OCT scan showed a foveolar cystoid space, combined with focal disruption of the photoreceptor line. Macular thickness was measured with calipers at 175 μm at the center.

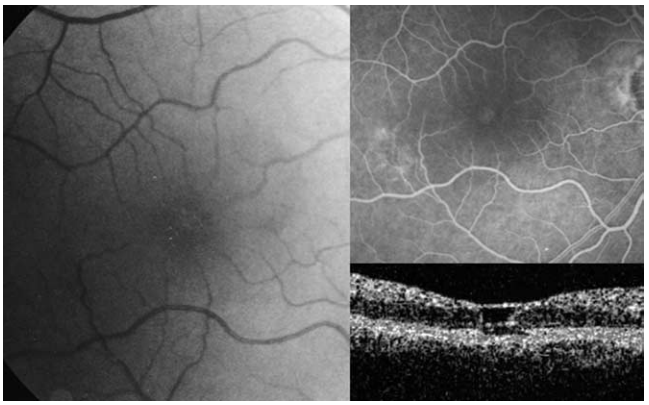

FIGURE 2. Tamoxifen retinopathy. Case 2. (Left) Red-free photograph of the right eye showing tiny refactile deposits. (Top right) Fluorescein angiography shows central hyperfluorescence. (Bottom right) A horizontal OCT scan shows a foveolar cystoid space with interruption of the photoreceptor layer.

(Carl Zeiss Meditec, Dublin, California). Fluorescein angiography and OCT were performed with informed consent given by the patient. OCT disclosed an image of foveolar cystoid space combined with focal disruption of the photoreceptor line (Figure 1, bottom). There was no macular thickening. Central foveolar thickness was measured with calipers at 175 μm.

A 72-year-old woman had taken a cumulative dose of 30.6 g of tamoxifen. Visual acuity was 20/50 in the right eye and 20/25 in the left eye. Fundus examination of the right eye showed deposits typical of tamoxifen retinopathy (Figure 2). Fluorescein angiography and OCT were performed with informed consent given by the patient. On fluorescein angiography, mild foveolar hyperfluorescence was observed, without dye pooling. OCT revealed a foveolar cystoid space also combined with focal disruption of the photoreceptor line, but no macular thickening (Figure 2). Foveolar thickness was measured with calipers at 167 μm. Fundus examination of the left eye showed no foveal deposits, but fluorescein angiography revealed slight foveolar hyperfluorescence. OCT was normal.

In these two cases, OCT revealed a foveal cystoid space, which occupied most of the foveolar thickness. There was no evidence of macular thickening. These findings are not consistent with previous descriptions of tamoxifen retinopathy, which include a description of cystoid macular edema,[1,2] but, on the contrary, tend to indicate some degree of atrophy of the retinal tissue in the foveolar center, as shown also by the disruption of the photoreceptor line.[3]

Pathogenesis of tamoxifen toxicity is unknown. However, it may act, in vivo, as an antagonist of glutamate transporters in retinal pigment epithelium cells, as already demonstrated in vitro.[4] We suggest that tamoxifen leads to an increase in glutamate, which would explain the axonal degeneration observed histopathologically[1] and the crystalline deposits corresponding to degenerative products observed clinically. Müller cell impairment may follow retinal neuron injury[5] and generate atrophy and formation of an intraretinal foveolar cyst, observed here thanks to OCT. Further experimental and clinical studies are needed to describe the typical OCT pattern of tamoxifen retinopathy accurately and to improve our understanding of the pathogenesis of the disease.

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Transient Serous Retinal Detachment in Classic and Occult Choroidal Neovascularization After Photodynamic Therapy

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PURPOSE: To quantify transient serous retinal detachment in classic and occult choroidal neovascularization (CNV) after photodynamic therapy (PDT).

DESIGN: Prospective consecutive case series.

METHODS: Consecutive patients with classic and occult CNV were examined by optical coherence tomography before PDT and at 2 and 7 days after PDT.

RESULTS: In classic CNV (n = 6), retinal elevations increased from 217 (SD 42) μm before PDT to 626 (SD 157) μm 2 days after PDT and decreased to 240 (SD 36) μm 7 days after treatment. In occult CNV (n = 4), the mean retinal elevation of 266 (SD 41) μm before PDT

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