and off-label uses, ophthalmologists should anticipate an increasing number of topiramate-associated BAACG cases and perform ALPI if discontinuation of topiramate and ocular hypotensive therapy fail to reverse this sight-threatening disease.

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Cystoid Macular Edema Secondary to Albumin-Bound Paclitaxel Therapy

Cystoid macular edema (CME) without capillary leakage is a rare subcategory of CME recently associated with the taxane drugs docetaxel (Taxotere; Sanofi-Aventis US LLC, Bridgewater, New Jersey) and paclitaxel (Taxol; Bristol-Meyers Squibb Co, Princeton, New Jersey).1-3 Protein-bound paclitaxel (Abraxane; Abraxis BioScience Inc, Los Angeles, California) is an albumin-stabilized nanoparticle formulation of paclitaxel reported to be more effective and better tolerated than standard paclitaxel.4 We report for the first time to our knowledge a case of profound CME with minimal fluorescein leakage secondary to treatment with the newer albumin-bound paclitaxel, which resolved on discontinuation of the drug.

Report of a Case. A 56-year-old white woman had a 2-month history of decreased vision in both eyes with no other associated symptoms. She had been receiving protein-bound paclitaxel for approximately 2.5 years at a dosage of 400 mg every 3 weeks concomitant with trastuzumab (Herceptin; Genentech Inc, South San Francisco, California) for stage IV breast cancer, with the last intravenous infusion 3 weeks prior to her initial visit. Visual acuity corrected to 20/80 OD and 20/60 OS. Anterior segment examination revealed no inflammation and 1+ nuclear sclerosis in both eyes. Dilated fundus examination showed marked CME bilaterally. Spectral-domain optical coherence tomography (Carl Zeiss Meditec Inc, Dublin, California) determined the central retinal thickness to be 630 µm OD and 585 µm OS (Figure 1). Profound CME with well-defined septa was evident in the outer plexiform layer, with cystic changes clearly visible throughout all layers. Fluorescein angiography results were normal until late frames, where minimal leakage could be discerned centrally (Figure 2). The patient discontinued protein-bound paclitaxel therapy immediately but continued to receive trastuzumab.

On follow-up 4 weeks later, the patient noted marked improvement in her visual acuity (20/60 OU uncorrected) and ophthalmic examination confirmed dramatic reduction of the CME. Spectral-domain optical coherence tomography documented the central retinal thickness in the right eye to be 352 µm, a reduction of 278 µm. Similarly, the left eye reduced by 291 µm to a central retinal thickness of 294 µm. Only small cystic

Figure 1. Spectral-domain optical coherence tomographic imaging reveals profound cystoid macular edema at the initial visit with intraretinal cysts primarily in the outer retinal layers of the right eye (A), and the patient exhibited complete resolution at the 3-month follow-up (B). The left eye had similar findings.
changes remained. At the 3-month follow-up, the patient’s best-corrected visual acuity had improved to 20/20 OU and spectral-domain optical coherence tomography revealed complete resolution of CME with central retinal thickness of 252 µm OD and 261 µm OS (Figure 1).

Comment. Our findings demonstrate that albumin-bound paclitaxel, like other taxanes, can result in CME, which is reversible on cessation of protein-bound paclitaxel therapy. Interestingly, there has been another documented case of CME in a patient receiving trastuzumab concurrent with taxane therapy. However, it does not appear to be related to the cause of CME in our case as our patient’s condition improved despite continued trastuzumab therapy.

The pathogenesis of CME without capillary leakage remains unclear. The slight amount of leakage on late fluorescein angiographic frames in our case implies a partial compromise of the blood-retinal barrier. With a partial compromise, smaller molecules and proteins may diffuse out more readily than the larger fluorescein molecules. Fluorescein would diffuse into the extracapillary space at a much slower rate, resulting in the findings of no or very slight leakage on late fluorescein angiographic frames. A previous report suggested that the mechanism of drug-induced CME without capillary leakage is similar to that of fluid retention syndrome. Our patient, however, had no systemic evidence of fluid retention. There is no other explanation for why taxane therapy caused CME in this patient, especially after 2.5 years of treatment.

It was hoped that this new nanoparticle protein-bound formulation of paclitaxel would have fewer adverse effects than the non–protein-bound version. However, it appears that all types of taxane therapy can cause angiographically atypical CME. If the association is recognized, the condition appears to be reversible on discontinuation of therapy.

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Inconsistencies and Gaps in Evidence Concerning Vitamins and Risk of Cataract

The results of this study are limited in their interpretation because of the following: (1) The study did not measure cataract development in a standardized fashion according to a standardized grading system. (2) Given that cataract was the main outcome measure, there should have been qualified examinations at entry and exit to identify all cataract cases. (3) The study was observational and thus can strongly sug-