Cystoid macular edema (CME) is thought to result from disruption of the normal blood-retinal barrier. Leakage from paravascular capillaries is demonstrated on fluorescein angiograms as a classic petalloid pattern in typical CME. Expansion of the intracellular fluid space may also lead to CME. Accumulation of fluid in the intracellular space may lead to CME without evidence of leakage on fluorescein angiograms. We report a case in which CME was secondary to paclitaxel (Taxol; Bristol-Meyers Squibb Co, New York, NY) use without evidence of leakage at angiography.

Report of a Case. A 63-year-old woman reported gradual decreased vision in both eyes. The patient’s medical history was significant for metastatic breast carcinoma with previous radiation therapy to the brain. Her chemotherapeutic regimen consisted of trastuzumab (Herceptin; Genentech Inc, South San Francisco, Calif) and paclitaxel (175 mg/m² for 10 months). At the initial ophthalmologic examination, the best-corrected visual acuity was 20/80 OU. Anterior segment examination yielded normal findings. Dilated fundus examination revealed no evidence of vitreitis. Cystoid macular edema was clinically noted in both eyes. Fluorescein angiograms exhibited normal filling of the choroidal and retinal vessels and an intact paravascular capillary net. Late frames of the angiograms did not show any significant leakage. Optical coherence tomography scans of both eyes revealed CME with a foveal thickness greater than 500 µm (Figure). Findings from the examination were consistent with CME in both eyes without any evidence of fluorescein leakage. Niacin maculopathy, Goldmann-Favre syndrome, and congenital X-linked retinoschisis were ruled out on the basis of history and clinical examination. Literature review yielded 1 case report of CME without fluorescein leakage.

**Paclitaxel Maculopathy**

Cystoid macular edema (CME) is thought to result from disruption of the normal blood-retinal barrier. Leakage from paravascular capillaries is demonstrated on fluorescein angiograms as a classic petalloid pattern in typical CME. Expansion of the intracellular fluid space may also lead to CME. Accumulation of fluid in the intracellular space may lead to CME without evidence of leakage on fluorescein angiograms. We report a case in which CME was secondary to paclitaxel (Taxol; Bristol-Meyers Squibb Co, New York, NY) use without evidence of leakage at angiography.

**Figure 6.** Fundus color photograph of the right eye shows prominent whitish choroidal granulomas (A) with diminution following treatment (B).
leakage secondary to docetaxel use. Both paclitaxel and docetaxel are mitotic inhibitors with similar mechanisms of action. In our patient, CME was thought to be secondary to paclitaxel use. Paclitaxel treatment was discontinued and capecitabine (Xeloda; Hoffman-LaRoche Inc, Nutley, NJ) was prescribed by the patient’s oncologist.

Six weeks after cessation of paclitaxel treatment, the patient’s best-corrected visual acuity improved to 20/40 OU. Fundus examination findings and optical coherence tomography scans (Figure) revealed resolution of CME.

Comment. Paclitaxel is an antimitotubule agent that inhibits normal reorganization of the microtubule network within cells. Toxic effects to bone marrow are the predominant dose-limiting adverse effect of this agent. Ophthalmic adverse effects include decreased vision, scintillating scotomas, and abnormal visual evoked potentials. Cystoid macular edema most often develops in association with inflammation or after cataract extraction. Typical CME is manifested with leakage as seen on fluorescein angiograms. Radiation retinopathy may also result in CME. Although our patient’s history was significant for previous radiation therapy, the lack of accompanying funduscopic findings and absence of leakage at angiography makes radiation therapy an unlikely etiologic factor. There has been 1 reported case of CME without leakage on fluorescein angiograms associated with docetaxel therapy. The pathophysiology of angiographically negative CME is unclear. Toxicity to Muller cells with subsequent intracellular fluid accumulation and subclinical leakage of extracellular fluid have been proposed.

We report a case in which paclitaxel use was associated with angiographically negative CME. Modification of the patient’s chemotherapeutic regimen resulted in resolution of CME and improvement in visual acuity.

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Optic Disc Tubers

Tuberous sclerosis is an autosomal-dominant disorder characterized by enhanced proliferation of neural and astrocytic precursors. It is caused by mutations in either TSC1 or TSC2, with loss of hamartin or tuberin function. Affected patients exhibit a specific constellation of neurologic, cutaneous, visceral, and retinal lesions. The classic triad of epilepsy, adenoma sebaceum, and mental retardation is found in less than one third of cases diagnosed by current criteria.

Unilateral optic disc elevation in tuberous sclerosis is usually attributable to an astrocytic hamartoma on the surface of the optic disc. These phakomas may gradually calcify but generally do not enlarge. We document preservation of vision despite massive enlargement of a tumor situated within the optic disc in a child with tuberous sclerosis.

Report of a Case. A 19-month-old girl was found to have swelling of the right optic disc. She had developed seizures at 6 weeks of age, which were controlled with carbamazepine. At 6 months of age, she had been diagnosed with tuberous sclerosis when magnetic resonance imaging disclosed multiple subependymal nodules and cortical tubers without ventriculomegaly. She had a hypopigmented macule and mul-

Figure. Optical coherence tomography scans of the right and left eyes. A and B, Marked cystoid macular edema at the initial examination. C and D, Resolution of edema noted 6 weeks after cessation of paclitaxel therapy.