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 antimicrotubule 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.

Optic Disc Tuber

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-
Multiple ash-leaf spots on her legs and trunk. At 7 years of age, she developed a trace afferent pupillary defect in the right eye with no corollary change in her vision or optic disc appearance (Figure A).

At 12 years of age, her seizures were controlled with carbachol, and she was making excellent grades in school.
school. Visual acuity was 20/20 OU. The patient was able to identify all Hardy-Rand-Ritter color plates using either eye. Both pupils responded briskly to light, and she retained a 1+ afferent pupillary defect in the right eye. Retinal examination disclosed circumferential enlargement of the optic disc tumor and increased smooth elevation of the peripapillary retina with no obscuration of the major retinal vessels (Figure B). Tiny surface drusen were now visible, and the optic disc was blanketed by a lattice-like arteriovenous network of filigree vessels. The left optic disc appeared normal.

Early-stage fluorescein angiography showed surface vascularity with mottled hyperfluorescence within the tumor (Figure C). The late-stage angiogram showed diffuse nodular staining of the tumor surface (Figure D). Visual field testing showed isolated blind spot enlargement with a normal mean deviation (Figure E). B-scan ultrasonography showed calcifications within the optic nerve head. Optical coherence tomography showed an intrinsic optic disc tumor extending into the peripapillary subretinal space, with surface drusen that produced shadowing within the tumor (Figure F).

Comment. In tuberous sclerosis, unilateral optic disc swelling is usually caused by an astrocytic hamartoma situated on the surface of the optic disc. The early lesion appears as a focal elevated mass of whitish, gray, or yellowish tissue that obscures visualization of the underlying retinal vessels. These optic disc hamartomas may gradually calcify into raised tumors with a “mulberry-like” appearance that resembles optic disc drusen in consistency. Fluorescein angiography shows a prominent network of fine blood vessels in the superficial portion of the mass during the venous phase, and intense late staining of the tumor. Although these lesions only rarely enlarge, Shields et al recently documented visual loss secondary to massive enlargement of astrocytomas of the retina and optic disc in 4 patients with tuberous sclerosis.

Our patient’s intrapapillary tumor was characterized by progressive enlargement of the optic disc, and gradual expansion into the peripapillary subretinal space, suggesting the possibility of a low-grade intrapapillary astrocytoma. Despite its impressive enlargement and increasing surface vascularity, this lesion has remained visually inconsequential during 10 years of observation.

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Lymphomatoid Granulomatosis Associated With Bilateral Exudative Retinal Detachments

Lymphomatoid granulomatosis (LYG) was first described by Liebow et al in 1972. It is a necrotizing lymphoproliferative disorder primarily affecting the lungs. The skin, kidneys, and nervous system may be involved, but ocular involvement is unusual. We present the case of a 39-year-old man who developed bilateral exudative macula detachments 16 years following onset of LYG. Prompt management with systemic steroids resulted in complete resolution with no long-term sequelae.

Report of a Case. A 39-year-old man attended the ophthalmology department with a 3-day history of bilateral blurring of vision with no other ocular symptoms. There was no ophthalmological history.

He had been examined because of cough, dyspnea, and a skin rash 16 years previously and was subsequently diagnosed with LYG. Chest x-ray film had shown bilateral infiltrates with bronchoscopic biopsy yielding granulomatous material with perivascular lymphoid infiltrates. A skin biopsy helped confirm the diagnosis of LYG. At the time of his initial ophthalmological examination, he was taking 20 mg of prednisolone daily to control the pulmonary symptoms of LYG.

On examination, his visual acuities were 20/60 OD and 20/40 OS. Fundus examination revealed bilateral dome-shaped smooth elevations of the macula with subretinal fluid visible, consistent with exudative retinal detachments. No retinal breaks, vasculitis, or vitritis was present. Fluorescein angiography demonstrated bilateral multifocal pinpoint leakage throughout the macula and extensive subretinal fluid (Figure).

The patient’s dose of prednisolone was decreased to 60 mg daily. Within 4 weeks, visual acuities had returned to 20/20 OU with normal Amsler test results. Fundus examination now revealed complete resolution of the subretinal fluid and flat retinæ bilaterally with no obvious sequelae of the exudative detachments seen.

The dose of prednisolone was thereafter reduced in 5 mg increments, and 5 months later, with the dose back at 20 mg, vision had remained stable with no recurrence of the subretinal fluid. Two years of follow-up have shown no further ocular involvement, but further skin lesions and arthralgia in the patient have also responded to systemic steroid therapy.

Comment. Lymphomatoid granulomatosis is an uncommon granulomatous inflammatory syndrome closely related to Wegener granulomatosis and to malignant lymphoma. It requires histopathological diagnosis because its clinical features are often difficult to distinguish from those of other granulomatoses, so it is frequently misdiagnosed as sarcoidosis. Clinical