

eye. The patient would not consent to laser treatment at any time during his treatment course. Unfortunately, further bleeding occurred, which resulted in a dense vitreous hemorrhage.

The left eye was monitored conservatively for several months, but showed no sign of clearing. Visual acuity in the right eye improved during this time to 20/70. On Aug. 28, 1991, the patient underwent pars plana vitrectomy in the left eye. The surgical procedure proceeded favorably, but marked preretinal fibrosis, retinal pigment epithelial atrophy, and metaplastic subretinal tissue were evident in the macula. Early preoperative cataract demonstrated progression that ultimately required extracapsular cataract and anterior chamber intraocular lens implantation, which were performed on Dec. 11, 1991.

On Jan. 23, 1992, best-corrected visual acuity was R.E.: 20/40 and L.E.: 20/300. There was no clinical sign of hemorrhage in the left macula, but there was distortion caused by preretinal fibrosis and metaplastic subsensory retinal tissue.

Focal arterial wall damage is the likely precursor of retinal artery macroaneurysms. The rarity of emboli as the cause of this damage is evident from previous reports of macroaneurysms. Some additional cases in which an embolus is noted near a macroaneurysm could have a similar origin. Palestine, Robertson, and Goldstein³ noted that only one of ten cases had an embolus nearby. Thus, it is likely that most are not caused by embolic disease. Of course, most macroaneurysms are detected because of symptoms of loss in visual acuity. The appearance of the retinal artery before the onset of the symptoms is not known in most cases, and the potential for an undetected embolus to have preceded the macroaneurysm cannot be excluded. Emboli have been demonstrated to be the definite cause of a retinal artery macroaneurysm in three cases, including the present one.^{1,5} The lack of macroaneurysms as a subsequent development in the large series of retinal artery emboli described by Arruga and Sanders⁴ implies that most macroaneurysms are attributable to other causes.

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Nonbacterial Thrombotic Endocarditis and Cortical Blindness

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A 73-year-old man had a two-week history of stepwise painless progressive visual loss in both eyes. No focal nonvisual neurologic symptoms occurred; however, he experienced four weeks of headaches and two weeks of mild confusion. His medical history was unremarkable.

Visual acuity was R.E.: 20/200 and L.E.: counting fingers at 2 feet. He identified none of the ten City University color plates with either eye. Pupillomotor responses were brisk without afferent defect in both eyes. Results of the examination of the ocular fundi were normal bilaterally. Computed perimetry showed absolute superior altitudinal field loss with involvement of fixation, along with moderate generalized depression inferiorly in both eyes. Results of a left superficial temporal artery biopsy and bilateral carotid ultrasonograms were normal. Cranial magnetic resonance images showed a lesion in the left occipital lobe of mixed signal, suggesting hemorrhage, and mild gyral enhancement of the right occipital cortex (Fig. 1). Progressive confusion and weight and appetite loss resulted in hospitalization and death 11 days later. A chest x-ray film disclosed a left upper lobe infiltrate found on autopsy study to be a mucin-secreting pulmonary adenocarcinoma. Nonbacterial thrombotic endocarditis was present on the aortic and mitral valves, as well



Fig. 1 (Rush). T₂-weighted image shows the round hypointense lesion in the left occipital lobe and mild gyral enhancement of the right occipital cortex.

as in both posterior cerebral arteries, and caused multiple systemic infarctions, including those imaged on magnetic resonance scans (Fig. 2).

Occipital stroke is insufficiently recognized as a cause of visual loss in patients with cancer in whom nonbacterial thrombotic endocarditis develops. As many as 85% of patients with nonbacterial thrombotic endocarditis have underlying tumors, especially mucin-secreting adenocarcinomas of the lung and pancreas.¹ Undetectable by cardiac auscultation and conventional echocardiography because of their smaller size, the friable fibrin-platelet excrescences frequently embolize to the middle cerebral arteries and cause nonvisual stroke, which might be the initial clinical symptom of cancer.² Homonymous hemianopsia with ipsilateral sensory³ or motor⁴ deficits has been reported, but occipital visual loss without focal nonvisual

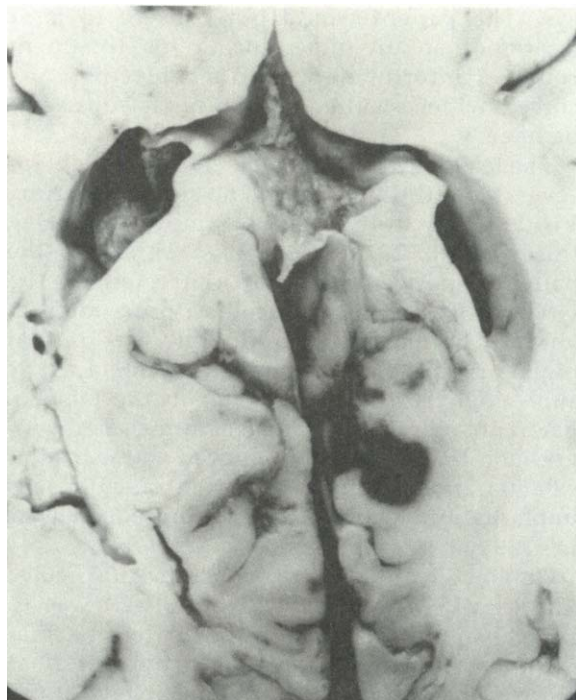


Fig. 2 (Rush). Axial autopsy section through occipital cortex shows round area of hemorrhage in the left occipital lobe and bilateral calcarine cortical infarctions.

neurologic symptoms, as in this case, might be the earliest symptom.

Nonbacterial thrombotic endocarditis should be considered in any stroke patient in whom a source of emboli cannot be found. As a remote effect of cancer on the visual system, its early detection might lead to life-prolonging anticoagulation.⁵

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Visual Recovery After Radiation Therapy of Orbital Lymphoma

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An 84-year-old white man with glaucoma had a history of transitional cell carcinoma of the urinary bladder, disseminated well-differentiated lymphocytic follicular lymphoma, and squamous metaplasia of the prostate.

On Dec. 5, 1991, the patient had visual acuity of R.E.: 20/25 and L.E.: 20/30 (ascribed to nuclear sclerotic cataracts). On Jan. 6, 1992, he returned because of decreased vision in his left eye. Ophthalmic examination then disclosed visual acuity of R.E.: 20/30 and L.E.: no light perception. Applanation tensions were 16 mm Hg in both eyes. His pupils were miotic and nonreactive as the result of pilocarpine therapy. Both exotropia and exophthalmos were noted in his left eye. Hertel exophthalmometry showed 5 mm of proptosis at 16 mm in the right eye and 21 mm in the left eye, with a base diameter of 95 mm. Slit-lamp biomicroscopy disclosed edema of the left upper eyelid.

Computed axial tomography of the left orbit showed an ovoid area of soft tissue 3.0 cm long and 18 mm wide surrounding the left optic nerve and extending to the orbital apex (Fig. 1). Intratumoral microcalcifications were noted.

Because of the patient's desire to have prostate surgery, he was first examined in consulta-

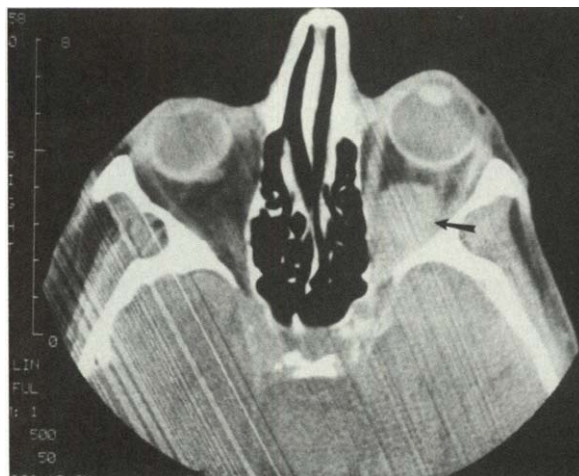


Fig. 1 (Moshfeghi and associates). Computed axial tomography shows a 3.0 × 1.8-cm tumor surrounding the optic nerve in the left eye (arrow).

tion on Jan. 30, 1992. Visual acuity was R.E.: 20/40 and L.E.: no light perception. Results from an ultrasonically guided fine-needle aspiration biopsy of the left orbital tumor were consistent with small cell malignant orbital lymphoma (Fig. 2).

Fifty-one days after documentation of the patient's loss of light perception in his left eye, external beam radiotherapy was initiated. Through a paired wedge technique, a 4 × 5-cm field, and 6-MEV photons, our patient received a total dose of 36 Gy (18 fractions of 200 cGy over a period of 24 days).

Two weeks later, visual acuity was L.E.: 20/40. The patient had normal ocular motility and decreased eyelid edema. Indirect ophthalmoscopy showed no evidence of maculopathy, vasculopathy, or optic neuropathy. Three months after treatment Hertel exophthalmometry showed symmetry at 16 mm in both eyes with a base diameter of 95 mm. Though the patient's visual acuity had stabilized at 20/40 in both eyes, relative optic nerve pallor was noted in the left eye.

Orbital lymphomas are the most common malignant orbital tumors and account for 10% of tumors of the orbit and adnexa.^{1,2} Though these tumors are relatively radiosensitive (as was illustrated by our case),^{3,4} it was remarkable that our patient started treatment 51 days after the first documentation of no light perception in the involved eye, and recovered to 20/40.

This case suggests that when orbital lymphoma-