

and that older age, presence of glaucoma or diabetes mellitus (DM), larger radiation doses, larger tumor size, lack of rectus muscle disinsertion, and anterior tumor location may be associated with larger IOP elevations.

Methods. This was a retrospective record review of all patients undergoing Collaborative Ocular Melanoma Study–style brachytherapy at the Eye Institute of the Medical College of Wisconsin between January 1, 1996, and December 31, 2007. Institutional review board approval was obtained. Patients were identified by billing record search. Exclusion criteria included being younger than 18 years, having nonmelanotic tumors, and having incomplete medical documentation. The following data were gathered: preoperative IOP, age, sex, history of DM or glaucoma, tumor location and height, radiation dose, plaque diameter, daily IOP during brachytherapy, muscle disinsertion, and use of topical antiglaucoma medications.

Results. Of 113 patients identified as having uveal melanoma, 40 were excluded owing to incomplete records. There were 33 men and 40 women. The mean (SD) age was 61.9 (15.7) years. One patient had open-angle glaucoma, no patients had ocular hypertension, and 12 patients had DM.

Three IOP variables were constructed: a preoperative IOP composite for each patient, defined as the average of preoperative IOP measurements (≤ 3 measurements); delta IOP (Δ IOP), or the change in IOP as measured on a perioperative day minus the preoperative composite; and the maximum Δ IOP (Δ IOP_{max}) for each patient.

The mean preoperative IOP composite was 16.0 mm Hg. This was statistically different from the mean IOP_{max} of 24.3 mm Hg during brachytherapy ($P < .001$) but not statistically different from the mean IOP of 15.5 mm Hg 1 day after plaque removal ($P = .24$) (**Figure**). The mean (SD) Δ IOP_{max} during plaque therapy was 8.5 (6.0) mm Hg. This was not statistically different for tumors at the posterior pole, midperiphery, and ciliary body ($P = .15$). Using a linear regression model, there was no relationship between tumor height and Δ IOP_{max} ($P = .53$) or between the mean total radiation dose and Δ IOP_{max} ($P = .46$). There was limited correlation between plaque diameter and Δ IOP_{max} using a linear regression model ($r^2 = 0.07$; $P = .02$). The Δ IOP_{max} for patients with DM was not statistically different from the Δ IOP_{max} for those without DM ($P = .40$). Eighteen patients with extraocular muscle disinsertion did not have statistically different Δ IOP_{max} compared with 55 patients without extraocular muscle disinsertion ($P = .72$).

Comment. This study demonstrated a statistically significant trend of increased IOP of 8 mm Hg in eyes treated with Collaborative Ocular Melanoma Study–style iodine 125 brachytherapy that persisted while the plaque was in place but resolved by the first postoperative day. Ten patients (14%) experienced IOP increases of 15 mm Hg or more, and 23 (32%) required topical hypotensive therapy. These medications may minimize the IOP elevation associated with brachytherapy. Factors such as age, DM, plaque size or location, tumor height, and radiation dose do not serve as reliable indicators of who will experience these marked elevations in IOP. Given

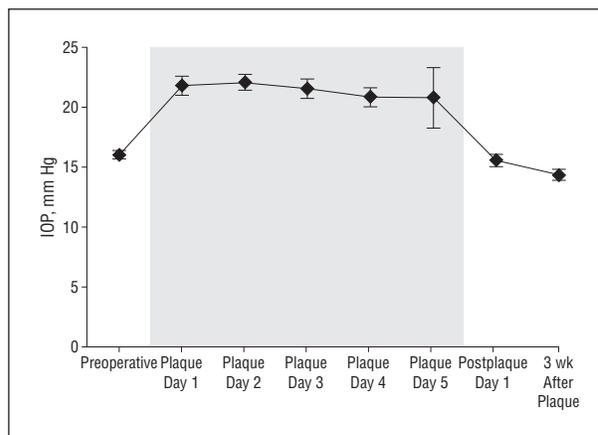


Figure. Variation in intraocular pressure (IOP) from preoperative measurement throughout brachytherapy and postoperative measurements. Error bars indicate standard errors of the means.

the number experiencing moderate IOP elevation and our inability to identify these patients preoperatively, regular IOP monitoring may be advisable for patients while undergoing iodine 125 brachytherapy.

Sandeep K. Bhatia, MD
Douglas J. Covert, MD, MPH
William J. Wirostko, MD

Author Affiliations: Retina Service of the Eye Institute, Medical College of Wisconsin, Milwaukee.

Correspondence: Dr Wirostko, Retina Service of the Eye Institute, Medical College of Wisconsin, 925 N 87th St, Milwaukee, WI 53226 (wirostko@mcw.edu).

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Photoreceptor Recovery Following Laser Photocoagulation and Albendazole in Diffuse Unilateral Subacute Neuroretinitis

Diffuse unilateral subacute neuroretinitis is a rare condition that typically causes significant vision loss. Herein, we describe spectral-domain optical coherence tomographic (OCT) findings correlating with vision recovery following treatment of a patient with diffuse unilateral subacute neuroretinitis.

Report of a Case. A 45-year-old man reported having progressive visual decline, floaters, and photopsias in the left eye for 2 months. One month prior, he was noted to have

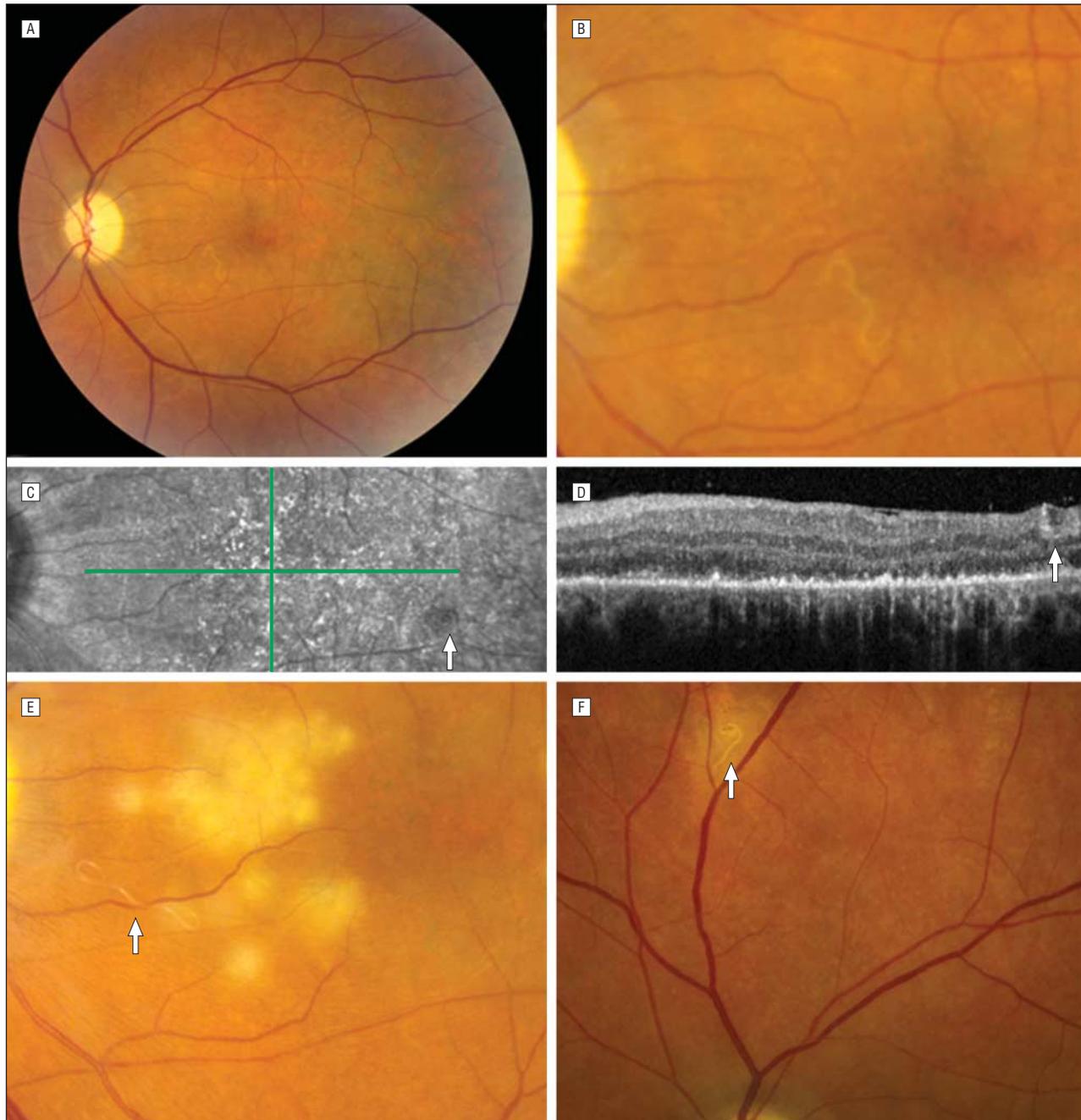


Figure 1. Fundus photographs, infrared photograph, and spectral-domain optical coherence tomographic image. A, Fundus photograph demonstrates mild optic nerve pallor, arteriolar attenuation, retinal pigment epithelial mottling, and a nematode in the papillomacular region. B, Higher-magnification fundus photograph shows the nematode. C, Infrared photograph demonstrates diffuse retinal pigment epithelial mottling in the macula and shows the nematode temporal to the fovea (arrow). D, Spectral-domain optical coherence tomographic image of a horizontal section inferior to the fovea shows widespread outer retinal disruption and a cross-section of the nematode located in the inner retina (arrow). Fundus photographs show the appearance of the nematode (arrow) following initial (E) and subsequent (F) laser photocoagulation.

anterior segment inflammation. Treatment with topical and oral steroids did not halt his vision loss. He reported an unremarkable medical and ocular history. Review of systems revealed recent travel to Mexico and frequent outdoor activity in the northern United States.

Visual acuities measured 20/20 OD and 20/160 OS. A 1.2-log unit relative afferent pupillary defect was present in the left eye. Anterior segment examination findings were unremarkable. Dilated examination of the left eye demonstrated rare vitreous cells, optic nerve pallor,

arteriolar attenuation, diffuse retinal pigment epithelial mottling, and a motile 1500- μm nematode in the papillomacular region (**Figure 1** A and B). Spectral-domain OCT demonstrated widespread outer retinal disruption, the nematode located in the inner retina, and an elevated epiretinal membrane (Figure 1C and D and **Figure 2**A). Photocoagulation of the worm was performed, avoiding the fovea (Figure 1E). The patient began treatment with a 30-day course of albendazole, 400 mg/d. Three days later, the worm had moved into the su-

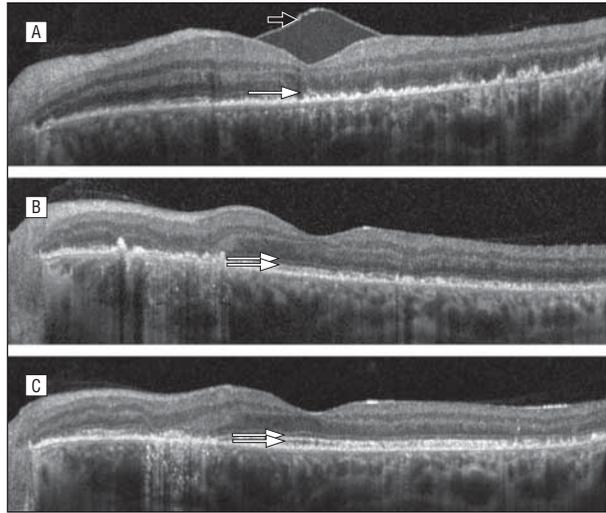


Figure 2. Spectral-domain optical coherence tomographic images. A, A horizontal section through the fovea at the initial visit shows widespread outer retinal disruption (white arrow) and an elevated epiretinal membrane over the fovea (dark arrow). Images 1 month (B) and 3 months (C) following treatment demonstrate disruption of the photoreceptors in the papillomacular region secondary to prior photocoagulation and progressive restoration of the inner segment/outer segment junction (upper arrow) and photoreceptor architecture (lower arrow) in the fovea.

perior retina and was still motile. More aggressive photocoagulation was applied and the worm was successfully killed (Figure 1F).

The patient reported significant subjective improvement, and his visual acuity measured 20/40 OS 1 month and 20/25 OS 3 months following the initial visit. Spectral-domain OCT showed disruption of the photoreceptors in the papillomacular region secondary to prior photocoagulation but progressive restoration of the normal inner segment/outer segment junction and photoreceptor architecture in the fovea (Figure 2B and C).

Comment. Diffuse unilateral subacute neuroretinitis was initially characterized by Gass et al¹ and further described by Gass and Braunstein.² Typically, patients initially have unilateral ocular inflammation, optic disc swelling, and chorioretinitis. This condition is caused by at least 2 separate nematodes of different sizes. It has been suggested that the smaller nematode, measuring approximately 500 μm in length, is *Ancylostoma caninum* and the larger nematode, measuring 1500 to 2000 μm in length, is *Baylisascaris procyonis*.²⁻⁴ Progression to optic atrophy, diffuse arteriolar attenuation, pigmentary degeneration, and significant vision loss occur if the nematode is not killed. The size of the nematode in this case is consistent with the larger worm.

Both antiparasitic medication and thermal laser photocoagulation can halt progression and may lead to visual improvement in some patients with diffuse unilateral subacute neuroretinitis.^{5,6} The treatment of choice is photocoagulation if the worm can be identified. At the initial laser treatment, the worm demonstrated rapid movement across the macula, propelling itself with a whipping movement. It was presumed to have been killed when movement ceased. However, 3 days later the worm migrated to the superior retina. At the second photocoagulation, there was minimal movement and more aggres-

sive laser was applied. It is possible that albendazole treatment resulted in impaired motility or that the initial laser injured the nematode.

Spectral-domain OCT demonstrated the initial inner retinal location of the nematode, associated diffuse disruption of the outer retinal architecture, and an elevated epiretinal membrane. Following treatment, progressive restoration of the inner segment/outer segment junction and outer retinal architecture suggests photoreceptor recovery. Persistent optic nerve pallor and visual field loss are likely the result of permanent damage to the ganglion cells, nerve fiber layer, and retinal pigment epithelium. The foveal epiretinal membrane was not seen on subsequent spectral-domain OCT and may have represented an inflammatory change that resolved. These findings coincided with improved visual acuity and support evidence that early killing of the nematode can allow visual improvement correlating with anatomical changes seen on spectral-domain OCT.

Ryan M. Tarantola, MD
Kori A. Elkins, MD
Christine N. Kay, MD
James C. Folk, MD

Author Affiliations: Vitreoretinal Service, Department of Ophthalmology and Visual Sciences, University of Iowa, Iowa City.

Correspondence: Dr Tarantola, Vitreoretinal Service, Department of Ophthalmology and Visual Sciences, University of Iowa, 200 Hawkins Dr, Iowa City, IA 52242 (ryan-tarantola@uiowa.edu).

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Peripheral Retinal Nonperfusion in Septo-optic Dysplasia (de Morsier Syndrome)

Septo-optic dysplasia, also known as de Morsier syndrome, includes the association of bilateral optic nerve hypoplasia (ONH), absence of the septum pellucidum, and pituitary maldevelopment with as-