cal steroids. At 3 weeks of follow-up, his best-corrected visual acuity in the left eye was counting fingers at 3 m, with a quiet anterior chamber and a large scar seen temporal to the fovea.

Comment. Cysticercosis is the most common ocular parasitic infection in humans, caused by the larvae of the tapeworm *Taenia solium.*2 Humans and pigs act as the intermediate host by ingesting eggs or gravid proglottids (body segments). Once the eggs are ingested, oncospheres (larvae inside the egg that each have a ring of 6 hooks) are liberated. The oncospheres penetrate the intestinal wall, enter into portal vessels or the mesenteric lymphatic system, and finally reach the systemic circulation. They are filtered out into the muscular tissues where they ultimately settle down and develop into the cysticerci (the resting stage of larva). Besides striated muscle, cysticerci may be seen in the eye and brain. The life cycle of the parasite is completed when humans ingest undercooked pork containing the cysticerci. In the human intestines, the cysts evaginate, and the scolex (head) anchors to the gut wall by means of its suckers and develops into an adult worm by gradual strobilization (the process of producing or growing new proglottids by asexual reproduction).2 The adult tapeworm resides in the small intestines for many years, and the only extraintestinal manifestation reported previously was in the spinal cord.3

In the present series, we report juvenile strobilate tapeworm presenting as fibrinous uveitis in 2 patients. Fibrinous anterior uveitis has been reported in cysticercosis cel lulose infections.4 In the present series, the 48-year-old man living in the Himalayan foothills of North India (case 1) had severe fibrinous uveitis with an abscess in the anterior chamber. There was no cyst seen clinically. A histopathologic examination of the iridectomy specimen showed the abscess wall attached to the iris. Because there were no parts of the tapeworm seen attached to the wall, it was reported as an abscess wall. However, because the eye subsequently developed phthisis bulbi, it is possible that there was a cyst in the eye that ruptured, causing severe inflammation and evagination of the juvenile worm. For the 38-year-old man living in Nepal (case 2), we performed a pars plana vitrectomy, and the subretinal cyst was removed in toto. However, this patient had severe postoperative fibrinous uveitis and was subsequently found to be harboring a juvenile tapeworm in the anterior chamber. In this case, there could possibly have been another small cyst/larva present behind the iris or in the angle of the anterior chamber that was missed initially and that later evolved into a juvenile worm in the anterior chamber.

Just how the adult tapeworm can survive in the anterior chamber is unclear. The best postulate is that the fluid-filled cyst of a scolex ruptures and that the evaginated scolex then attaches with hooks to the iris (like the intestinal wall) and grows thereafter.

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**Response to Ranibizumab Following Tachyphylaxis to Bevacizumab in a Patient With Radiation Maculopathy Following Stereotactic Fractionated Radiotherapy for Optic Nerve Meningioma**

A 38-year-old man presented to our eye clinic 21 months after radiotherapy for optic nerve meningioma in the left eye. At initial assessment, he had a visual acuity (VA) of 6/36, a central retinal thickness of 531 µm, and minimal disruption of the foveal avascular zone. The decision was made to treat his condition with intravitreal injections of bevacizumab (Avastin; Roche). His left eye’s initial response to bevacizumab was very encouraging, with VA improving to 6/12 after 3 injections. However, this response was transient; after 6 injections, he had a VA of 6/18 and a central retinal thickness of 475 µm in the left eye. His treatment was then switched to intravitreal injections of ranibizumab (Lucentis; Novartis), and he experienced a complete resolution of his cystoid macula edema, with a final VA of 6/6 and a central retinal thickness of 264 µm in the left eye. This is an interesting case of tachyphylaxis to bevacizumab in a patient with radiation maculopathy, with full resolution following intravitreal injections of ranibizumab.

**Report of a Case.** A 38-year-old man presented to our eye clinic 21 months after stereotactic fractioned radiotherapy (50 Gy) for optic nerve meningioma in the left eye. He was initially under the care of the oculoplastics team and neurologists from September 2003 following diagnosis of the meningioma, with proptosis, a VA of 6/4, and a constricted visual field. After 4 years of follow-up, in spite of the VA remaining stable, imaging revealed progression of the meningioma and further constriction of the Goldmann visual field; as a result, he was referred for radiotherapy. Twenty-one months after radiotherapy, his VA remained stable at
6/5. Ophthalmoscopy, however, revealed intraretinal hemorrhages and cotton-wool spots, and a diagnosis of radiation retinopathy was made. Six months later, his VA had deteriorated to 6/36. Optical coherence tomography revealed cystic intraretinal fluid with a central retinal thickness of 531 µm (Figure 1). Fundus fluorescein angiography revealed an enlarged foveal avascular zone (Figure 2).

Methods. This is a retrospective case report based on the patient’s response to therapy. After explaining the risks, benefits, and treatment options, he consented to anti–vascular endothelial growth factor (VEGF) injections.

All of the injections were given in an operating room, under strict sterile conditions. The eye was anesthetized with topical proxymetacaine and prepared with povidone-iodine (10% on the eyelid and periorcular region and 5% on the ocular surface). The anti-VEGF treatment was then drawn up using a 1-mL syringe with a 30-gauge needle (for ranibizumab) if not already in a pre-prepared syringe (for bevacizumab). Both drugs are stored at between 2°C and 8°C and are used before their expiration date. Following insertion of the speculum in the eyelid, further topical anesthesia was administered with a pledget over the injection site. Finally, an injection was given through the pars plana in the inferotemporal quadrant, 4 mm from the limbus. Optic nerve perfusion was assessed with indirect ophthalmoscopy, and topical chloramphenicol, 0.5% (4 times a day for 5 days), was prescribed.

All injections were given off-label bevacizumab monthly for 6 consecutive months before being switched to 0.05 mg of ranibizumab.

Results. The patient’s initial response after 3 injections of bevacizumab was observed, and his VA improved to 6/12. However, with subsequent injections, there was no further improvement: his left eye’s VA and central retinal thickness worsened to 6/18 and 475 µm, respectively (Figure 3).

He was switched to ranibizumab, and after 7 injections given on a pro re nata regime, his VA improved to 6/6, and he had a final central retinal thickness of 264 µm. There was no evidence of maculopathy (Figures 4 and 5).

Comment. Ever since its description by Stallard in 1933, radiation retinopathy has been known as a clinical entity in its own right. It occurs after irradiation for tumors or for inflammation of the choroid, retina, orbit, or paranasal sinuses. Although there is no definitive threshold, the risk of radiation retinopathy is related to the dose administered, with incidences increasing at doses greater than 45 Gy.2 Comorbidities and the use of other agents (such as chemotherapy) increase the risk of developing radiation retinopathy.2 From a morphological perspective, it can be characterized by retinal changes seen in any insult to the blood vessels and by ensuing hypoxia (more specifically, by microangiopathy, intraretinal hemorrhage, cotton-wool spots, cystoid macular edema, and neovascularization). This disease can cause irreversible loss of vision if the...
macula is involved; one study suggested that rates of radiation maculopathy are up to 23% from plaque radiotherapy.

Recent advances have revolutionized the traditional modalities of treating radiation retinopathy. Previously, nonsteroidal anti-inflammatory drugs, laser photoagulation (both focal macular and scatter), hyperbaric oxygen, and steroids delivered by different methods had been used, all with limited success. The theory behind anti-VEGF therapy is to inhibit the formation of new vessels, which will go on to leak and to decrease vascular permeability by reducing endothelial cell fenestrations through upregulating occludin. In 2007, Finger and Chin showed that bevacizumab was effective in treating radiation maculopathy. They produced a consecutive case series of 6 patients using a mean number of 3 bevacizumab injections and analyzed response at a mean follow-up of 4.7 months. They noted a marked improvement in leakage from vessels, stable or improved VA, and almost complete resolution of macular edema. It was shown that younger patients with new-onset maculopathy responded better. Mason et al treated 10 patients with bevacizumab, and these patients were observed for 4 months after treatment. They found that the mean foveal thickness decreased from 482 to 449 µm; however, there was only a modest improvement in VA. Indeed, Wen and McCannel published a review in 2009 in which they analyzed treatments for radiation retinopathy. They exclusively looked at bevacizumab in the anti-VEGF agents group and surmised that the improvements in VA after injections appeared to be minimal, with the exception of the results achieved by the Finger and Chin. Wen and McCannel suggest that the role of bevacizumab was limited.

Our case differs from those already mentioned in that, initially, there was some benefit seen with the injections of bevacizumab. It is well recognized that some patients with age-related macular degeneration seem to have a decreased response after repeated treatments and that others seem to have no response at all. Tachyphylaxis is a rapidly decreasing therapeutic response to a pharmacologically active substance following the initial doses. Eghøj and Sørensen observed that, in 976 patients (1076 eyes), 2% developed tachyphylaxis on reactivation of previously treated choroidal neovascularization.

In the eye, cellular mechanisms play a predominant role in this type of drug tolerance. For instance, a possible cause for this tachyphylaxis...
laxis response is the development of circulating antibodies against bevaciuzumab. Forooghian et al\textsuperscript{15} showed that levels of neutralizing antibodies increase with repeated injections and that the median time to develop tachyphylaxis is 100 weeks. Another suggested cause is a greater production of VEGF by local macrophages to negate the effect of the agent.

An alternative theory proposed by Schaal et al\textsuperscript{16} focuses on other mediators playing a role in propagating disease. They performed a small study whereby 15 eyes were treated with bevacizumab, 11 eyes were treated with triamcinolone, and 17 eyes were treated with both drugs, for subfoveal choroidal neovascularization secondary to age-related macular degeneration.\textsuperscript{16} In the bevacizumab group, approximately 3 injections were required for the efficacy of the treatment to decrease to 50% of the initial response; conversely, in the combination group, the mean number of injections was 5.1.\textsuperscript{16} Schaal et al\textsuperscript{16} suggested that other signaling pathways, such as fibroblast growth factor, could compensate for the blocked activity of VEGF when using bevacizumab alone.

Interestingly, there are reports in the literature of patients who develop tachyphylaxis to one anti-VEGF agent but respond to another. Hoffman and Taylor\textsuperscript{17} described this as one of the methods to reduce or avoid tachyphylaxis. Gasperini et al\textsuperscript{18} described 10 patients with age-related macular degeneration (3 classic lesions and 7 occult lesions) who were switched to ranibizumab after an average of 7 injections for apparent tachyphylaxis. Eight of these 10 patients had a positive therapeutic response after switching agents (4 after only 1 injection with ranibizumab). Two patients with classic lesions and 2 patients with occult lesions had complete resolution of subretinal fluid.\textsuperscript{18}

As can be appreciated, all the reports related to anti-VEGF treatment and tachyphylactic responses concerned patients with age-related macular degeneration, and, as such, they had pathogeneses that were different from the pathogenesis in our patient with radiation maculopathy. It is our understanding that there is nothing in the literature with respect to tachyphylaxis for anti-VEGF treatment of radiation maculopathy. There is, however, evidence to advocate the role of open-label ranibizumab (Lucentis) in treating radiation maculopathy. Finger and Chin\textsuperscript{19} performed a consecutive case series to analyze the effect of ranibizumab with respect to safety profile, change in VA, and resolution of maculopathy. All 5 patients had either 8 or 9 injections of ranibizumab. Their 8-month analysis showed a mean 35% reduction in foveal thickness and a mean improvement in best-corrected VA of 6 letters.\textsuperscript{19}

Yuan and Singh\textsuperscript{20} described their experience in treating a 49-year-old woman for metastatic breast cancer after she underwent brain radiation therapy. She received dual therapy, initially with panretinal photocoagulation and then intravitreal ranibizumab, because there was no improvement in VA after laser treatment. After 2 injections, her VA improved in the left eye by 4 lines, which did not correspond to improvement in foveal thickness, unlike the reports from Finger and Chin.\textsuperscript{19} For our patient, we had been monitoring his progress and basing our decisions for further injections partly on the optical coherence tomographic foveal thickness measurement; the data from Yuan and Singh\textsuperscript{20} suggest that perhaps one should not give too much credence to this parameter but should rather be more concerned with the VA.

Future studies are required to address the long-term course of the disease after anti-VEGF treatment. For instance, when should these patients be followed up? How many injections should they have, and over what time interval? When should a different form of management be considered? To our knowledge, the literature has hitherto been unable to answer these questions. In summary, we present a case of a 38-year-old man with radiation maculopathy who responded to intravitreal injections of ranibizumab after developing tachyphylaxis to intravitreal injections of bevacizumab.

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Figure 5. Fundus color photograph showing resolution of cotton-wool spots and retinal hemorrhage after ranibizumab therapy.
this work and should be considered as co-first authors.

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**Bietti Crystalline Retinopathy: Report of Retinal Crystal Deposition in Male Adolescent Siblings**

Bietti crystalline dystrophy (BCD) is a genetically determined disorder characterized by progressive chorioretinal degeneration, nystagmus, visual field constriction, and multiple intraretinal yellow-white crystalline deposits. Similar crystals can be seen at the corneoscleral limbus on slitlamp examination and in circulating lymphocytes on histological examination. Retinal crystals are observed predominantly at the posterior pole and in the superficial and deep retinal layers. They are associated with multiple, sharply demarcated areas of atrophy in the retinal pigment epithelium and loss of choriocapillaries; the crystals are less obvious as the disease advances. Bietti crystalline dystrophy is inherited as an autosomal recessive trait and is associated with mutations in the CYP4V2 gene (cytochrome P450, family 4, subfamily V, polypeptide 2). The CYP4V2 gene maps to chromosome 4q35, is expressed in a wide variety of tissues (including the retina and the cornea), and encodes an enzyme with a selective fatty acid α-hydroxylase activity.1,2

The age at onset of BCD is typically after the second decade of life, and to our knowledge, only 2 pediatric cases with fundoscopic lesions in keeping with BCD have been reported3,4; neither had a confirmed molecular diagnosis. In our report, we present the clinical find-

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**Figure 1.** Color fundus photographs of patient 1 (A; the proband) and patient 2 (B) showing multiple crystalline deposits that are more numerous at the posterior pole and peripapillary region than at the retinal periphery, which is relatively spared. The corresponding fundus autofluorescence images of patient 2 (C) are also shown.