A 62-year-old man with a large ciliochoroidal melanoma developed early-onset scleral necrosis with tumor extrusion within 1 month of epibulbar iodine I 125 plaque radiotherapy. The eye was enucleated. Pathologic study revealed nonmicrobial scleral necrosis with extrusion of histologically intact and necrotic uveal melanoma cells. The patient has been followed up for 15 months without clinical recurrence. We discuss possible mechanisms to explain the early development of scleral necrosis after plaque therapy in this patient.


Scleral necrosis is an uncommon but occasionally reported sequela of episcleral plaque radiotherapy for malignant intraocular tumors. In most reported cases, the scleral necrosis occurred either in the ciliary body region following unshielded plaque treatment of large ciliochoroidal melanoma or in the equatorial region posterior to the insertion of one of the rectus muscles. Most such cases occurred several months to years after plaque therapy. The following case report describes a patient with a ciliochoroidal melanoma who developed a large necrotic scleral defect within 1 month after shielded iodine I 125 plaque radiotherapy.

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REPORT OF A CASE

An asymptomatic 62-year-old white man was found to have a mass lesion of the choroid and ciliary body on routine eye examination. At initial evaluation, visual acuity was 20/15 OU. Slitlamp examination revealed prominent episcleral blood vessels superiorly in the right eye. Indirect ophthalmoscopy showed a solid, dark brown, mushroom-shaped superior ciliochoroidal mass associated with a localized extra-macular serous retinal detachment (Figure 1). The tumor measured 15 × 12 mm in basal diameter × 9.5 mm in maximal thickness. Apically, the tumor extended through Bruch’s membrane and invaded the retina, resulting in visible superficial intraocular hemorrhage. Ultrasonography confirmed the solid nature of the mass but showed no extrascleral tumor extension. Our diagnosis was primary ciliochoroidal melanoma in the right eye.

Baseline systemic testing revealed no evidence of metastatic disease. The patient was treated by surgical implantation of an 18-mm-diameter iodine I 125 radioactive plaque. During the procedure, the superior rectus muscle was disinserted to allow proper plaque placement. Inspection of the superior sclera at the time of surgery showed no thinning or abnormal pigment. The radioactive plaque delivered a total dose of 37 400 cGy to the tumor base and 8000 cGy to the tumor apex during 93.8 hours. The plaque was removed without complications, at which time the superior rectus was reinserted with a double-armed 5-0 polyglactin-910 suture.

Thirteen days later, the patient returned with pain, redness, and swelling of the right eye and upper eyelid. Topical antibiotic and corticosteroid drops were prescribed. One week later, the patient’s symptoms were unchanged. On indirect ophthalmoscopy, the intraocular tumor appeared smaller and the secondary retinal detachment appeared less extensive. Our presumptive diagnosis was periocular in-
flammation from the 2 recent surgical procedures. The patient was started on a course of oral prednisone (40 mg/d).

After 1 week of this therapy, the patient’s pain was less but the redness and swelling of his right eye and upper eyelid persisted. Examination revealed a dark epibulbar mass superiorly, suggestive of either a subconjunctival hematoma or transcleral extension of the ciliochoroidal tumor (Figure 2). Visual acuity in the affected eye was decreased to 20/400. Indirect ophthalmoscopic findings again suggested shrinkage of the tumor but revealed a more extensive retinal detachment and peripheral choroidal detachment. Magnetic resonance imaging of the eyes and orbits suggested scleral necrosis with partial extrusion of the tumor at the site of recent plaque therapy. On the basis of this evidence, enucleation of the right eye was recommended.

At enucleation, inspection of the globe confirmed an irregular defect in the sclera superiorly, through which dark tumor tissue extruded. The tumor was grossly cohesive, and no obvious pigmented tissue was left behind in the orbit. Pathologic study of the enucleated globe confirmed the presence of a ciliochoroidal melanoma (Figure 3) that protruded through a necrotic defect in the sclera. The tumor was composed largely of epithelioid melanoma cells. The cells filling the perforation and composing most of the base of the tumor were totally necrotic. The inflamed sclera adjoining the perforation was diffusely thickened and edematous and contained foci of largely necrotic polymorphonuclear leukocytes. Special stains for bacteria, fungi, and acid-fast organisms disclosed no microorganisms. The disrupted scleral fibers surrounding the necrotic defect were infiltrated by foamy histiocytes. No epithelioid histiocytes, inflammatory giant cells, necrobiotic collagen, or zonal granulomatous inflammation consistent with rheumatoid scleritis was observed. An intense infiltrate of well-differentiated lymphocytes and plasma cells was present on the surface of the globe. Several vessels were cuffed by lymphocytes, but there was no convincing evidence of systemic vasculitis.

The patient has been followed up for 15 months since enucleation. He has not developed either orbital tumor recurrence or metastasis to date. He also has not developed evidence of any rheumatic disease.

**COMMENT**

Iodine I 125 plaque radiotherapy is currently one of the most widely used conservative methods for treating primary choroidal and ciliochoroidal melanomas. Multiple complications of this treatment, including
radiation-induced scleral necrosis, have been reported. In virtually all reported cases of scleral necrosis after plaque radiotherapy, the interval between treatment and development of the scleral defect has been several months to several years. Our patient is unusual in that scleral necrosis developed within 1 month of plaque therapy.

Several possible mechanisms may explain the early development of the scleral defect in our patient. First, a direct necrotizing effect of radiation on the sclera is possible; however, the scleral dose of radiation in this patient was not appreciably different from that delivered in a typical case of this type in our center, and such a dose is generally well tolerated by the sclera. Second, indirect effects of treatment on the sclera may have occurred because of local ischemic inflammation related to disinsertion of the superior rectus muscle, tumor necrosis, or both. Third, an occult systemic autoimmune vasculitic disease may have precipitated the postoperative scleral necrosis. Fourth, either the direct or indirect effects of surgery mentioned above and the presence of an occult systemic autoimmune vasculitic disease could have been synergistic. Finally, focal weakening of the sclera because of unrecognized perforation during disinsertion or reinsertion of the superior rectus muscle cannot be excluded. Although the precise cause of the scleral necrosis in this patient cannot be established, we take consolation from the observation that this complication is extremely uncommon.

Accepted for publication September 22, 1998.

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REFERENCES


From the Archives of the ARCHIVES

A look at the past . . .

R. AXENFELD (Breslau). On the Tubercular histology of sympathetic ophthalmitis. The writer urges anew the occurrence of epithelioid and giant cells in eyes suffering from uveitis which has caused sympathetic ophthalmia. He shows specimens in which these have arranged themselves into typical tubercles. They had not become caseous, nor were bacilli present. In spite of the histological resemblance to tuberculosis the author does not think that the latter disease was present, for if so we should find tuberculosis oftener in cases of sympathetic ophthalmia. In order to decide the question more scientifically he recommends the transplantation, from freshly enucleated eyes, of portions of the choroid into the peritoneum of the guinea-pig.

Reference: Arch Ophthalmol. 1898;27:344