Compared with patients with classic GCD, those with the superficial variants have more opacities, leading to an earlier age of decreased visual acuity, generally during the second decade of life, and therein an earlier need for surgical intervention.2 Our patient claimed that her vision began to diminish when she was a teenager. The distinction between the superficial and classic forms of GCD has important clinical therapeutic implications because the superficial variants can be managed successfully by a superficial keratectomy, as in our patient, or by excimer laser phototherapeutic keratectomy; however, recurrences can occur and there are limits to the amount of acceptable corneal thinning.

Superficial GCD represents 1 of 2 well-characterized dystrophies that involve the Bowman layer. Ultrastructurally, the other dystrophy (Thiel-Behnke dystrophy) can be readily distinguished from SGCD because of the presence of subepithelial “curly” fibers.10 Because of the confusing nomenclature, the term CDB has been proposed for both of these dystrophies: CDB type I for SGCD and CDB type II for Thiel-Behnke dystrophy.10

Our failure to detect a mutation in exon 4 or 12 of the BIGH3 gene indicates that either this gene is not responsible for this case of SGCD and for the amyloid in our patient or a different part of the gene is mutated. Regardless of which of these 2 possibilities is true, the present case indicates that SGCD with amyloid deposition can occur without a mutation in either of the 2 exons in the BIGH3 gene that are known to be hot spots for mutations.

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This study was supported in part by research grant R01 EY 12712 from the National Eye Institute, Bethesda, Md.

We thank the Duke University Medical Center DNA Analysis Facility, Durham, for performing DNA sequencing and denaturing high-performance liquid chromatography.

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tion showed persistent, widespread macular edema, and fluorescein angiography confirmed continued, diffuse macular leakage. With a visual acuity of 20/60 and no response to laser therapy, he underwent intravitreal injection of 4 mg of triamcinolone acetonide in 0.1 mL, 4 mm posterior to the limbus, using a 27-gauge needle and a 1-mL tuberculin syringe. The patient was reclined in an examining room chair and the eye was prepped using topical povidone-iodine. Four weeks after the treatment, the patient went to his ophthalmologist with a painless decrease in visual acuity to hand motions in the treated eye, a hypopyon, and no view to the fundus. He urgently underwent vitreous tap and injection of intravitreal vancomycin (1 mg/0.1 mL) and ceftriaxone (2 mg/0.1 mL) for presumed endophthalmitis.

When the patient did not improve after the initial intravitreal antibiotics, and acid-fast bacilli were identified growing from the vitreous cultures, he was referred for further evaluation and treatment. With final species identification and susceptibilities still pending, the patient underwent repeated vitreous tap and injection of ceftazidime (2.25 mg/0.1 mL), vancomycin (1 mg/0.1 mL), and amikacin (0.4 mg/0.1 mL). He subsequently underwent pars plana lensectomy and vitrectomy and received an additional injection of vancomycin (1 mg/0.1 mL) and amikacin (0.4 mg/0.1 mL). Vitreous cultures grew M chelonae abscessus sensitive to amikacin and clarithromycin. Susceptibilities were determined by minimum inhibitory concentration (MIC); the MIC for amikacin was 16 µg/mL and the MIC for clarithromycin was 0.5 µg/mL. The patient was treated aggressively with both topical amikacin (8 mg/mL) and oral clarithromycin (500 mg by mouth twice daily). Over the following 3 months, he underwent vitreous cavity tap and injection of vancomycin (1 mg/0.1 mL) and amikacin (0.4 mg/0.1 mL) on 5 separate occasions in addition to the topical and oral treatments. Despite the intensive therapy, the intraocular culture results continued to be positive for organisms, the patient’s visual acuity dropped to no light perception, he had persistent pain in the eye, and a hypopyon redeveloped. Three months after the initial examination, the patient underwent enucleation of the involved eye.

Gross examination of the globe showed a 3-mm hypopyon with the remainder of the anterior chamber and vitreous cavity filled with white-tan fibrous tissue (Figure 1). Microscopic examination disclosed the vitreous cavity to be filled with a dense granulomatous infiltrate consisting of lymphocytes, histiocytes, and plasma cells with foci of giant cells. Examination of the infiltrate with a modified Ziehl-Neelsen stain disclosed numerous acid-fast bacilli consistent with the Mycobacteria species (Figure 2). Growth of the species M chelonae abscessus was confirmed on Lowenstein-Jensen culture media.

Comment. Macular edema is a common cause of visual compromise in patients with diabetes. Recently, several small case series and case reports have described encouraging visual and anatomic results with the use of long-acting intravitreal corticosteroids in the treatment of macular edema resistant to conventional laser therapy.1,2

Mycobacteria species are a rare cause of ocular infections and an even rarer cause of endophthalmitis; the species M chelonae has been described as causing keratitis, most recently in association with laser in situ keratomileusis as well as endophthalmitis.4,7 These reports detail the deleterious effect corticosteroids can have on mycobacterial infections. In a review of systemic infections due to M chelonae, Wallace and colleagues8 described the importance of prior corticosteroid therapy in the development of infections secondary to this organism. Additional reported cases of cutaneous and osseous infections caused by the M chelonae species have identified concurrent long-term corticosteroid therapy as a risk factor for developing such infections.9,10 In the current case, treatment with a long-acting intravitreal corticosteroid may have increased the susceptibility of this patient to infection with M chelonae abscessus. Innoculation of the organism into the vitreous may have occurred at the time of triamcinolone acetonide injection. Alternatively, the patient may have had a disseminated M chelonae abscessus infection with a distant focus, and an endogenous endophthalmitis may have developed in the setting of local ocular immunosuppression.8,10

Although encouraging results have been reported with the use of

**Figure 1.** Gross photograph of the pupilloptic nerve section of the globe with an anterior chamber hypopyon and a white-tan fibrous tissue present in the vitreous cavity.

**Figure 2.** Ziehl-Neelsen stain displaying numerous acid-fast bacilli consistent with Mycobacteria species (original magnification ×40).
intravitreal triamcinolone acetone in cases of diabetic macular edema refractory to focal grid laser photocoagulation, caution and close follow-up care is advised in these patients with a locally immunosuppressed intraocular status. In cases of endophthalmitis occurring after intravitreal injection of a long-acting corticosteroid, atypical Mycobacteria species should be considered in the differential diagnosis.

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Surgical Removal and Histopathologic Findings of a Subfoveal Neovascular Membrane Associated With Choroidal Osteoma

Choroidal osteomas are rare benign tumors that typically arise in young women who are otherwise healthy. Approximately 80% are unilateral, although Gass describes patients with unilateral osteomas who later develop bilateral disease. Ophthalmoscopic features include a well-defined, slightly elevated, white-to-orange lesion in the peripapillary or macular choroid. Histopathologically, these tumors are composed of cancellous bone and lie between an altered choriocapillaris and the outer choroidal layers. There is thinning and atrophy of the overlying retinal pigmented epithelium (RPE).

Many patients with choroidal osteomas are asymptomatic because vision in the affected eye may be remarkably well preserved. However, visual loss and metamorphopsia can occur due to geographic changes involving the central fovea or serous and hemorrhagic detachment of the macula; the latter is commonly the result of choroidal neovascularization.

No therapies currently exist to eradicate a choroidal osteoma. The subretinal neovascularization that complicates some cases is sometimes observed without treatment, particularly when the membrane extends beneath the fovea. In some cases, good vision can be maintained with spontaneous resolution of subretinal neovascularization. Other cases have been treated with laser photocoagulation, with variable results. Multiple treatments may be necessary to close the membrane, presumably because of the scarcity of melanin in the atrophic RPE.

A subfoveal location is considered to be a relative contraindication to laser photocoagulation because central vision may be immediately reduced. In recent years, surgical excision of subfoveal neovascular membranes has been described in association with various conditions, including age-related macular degeneration, presumed ocular histoplasmosis syndrome, punctate inner choroidopathy, and traumatic choroidal rupture. We report a case of surgical removal of a subfoveal neovascular membrane in a patient with a choroidal osteoma.

Report of a Case. In May 1994, a 30-year-old woman sought treatment at the Retina Service, Massachusetts Eye and Ear Infirmary, Boston, for blurred vision in her right eye of 2 days' duration. Examination revealed visual acuities of 20/25 OU, normal anterior segments, and a normal left fundus. In the right eye, peripapillary subretinal hemorrhage was present inferiorty from the 3- to the 9-o’clock position (Figure 1A). A neovascular membrane appeared to extend toward the fovea, and a large area of hypopigmentation of the RPE was present in the posterior pole concentric to the optic disc. Fluorescein angiography confirmed the presence of a neovascular membrane (Figure 1B), and B-scan ultrasonography revealed significant acoustic shadowing consistent with a choroidal osteoma (not shown). The neovascular membrane temporal to the disc was treated with argon laser photocoagulation. The subretinal hemorrhage resolved during the following 3 months, and the patient’s visual acuity remained at 20/25 in the affected eye.

In July 1995, 14 months after the laser treatment, visual acuity was 20/20–1 OD. However, the subretinal hemorrhage had recurred (Figure 1C), and fluorescein angiography revealed diffuse leakage that was more prominent inferiorly. Another laser treatment was discussed, but the patient did not wish to proceed.

The patient was followed up without treatment for another 16 months, during which time her visual acuity gradually decreased to 20/100 OD and the subretinal membrane grew beneath the fovea (Figure 1D and E). In December 1996, the membrane was surgically removed. After vitrectomy, a temporal retinotomy was created, and the neurosensory retina was elevated with balanced salt solution. The membrane...