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Glaucoma Care in a Patient With Previous Anterior Ciliary Sclerotomy and Scleral Expansion Procedure

Presbyopia is a gradual decrease of accommodation that becomes clinically significant during the fifth decade of life. Its pathophysiological changes remain uncertain and controversial. In recent years, Schachar and associates suggested that presbyopia occurs because of growth in the equatorial diameter of the lens, and the ciliary muscle contraction can no longer tense the zonule and expand the lens coronally. Based on this theory, scleral expansion by making radial relaxing incisions in the sclera or implanting plastic bands intrasclerally to expand the scleral ring were postulated to restore the accommodation. Although the clinical efficacy of these surgical techniques remains to be proven, they are being offered widely as a means to correct the inevitable ocular afflication of presbyopia. We report an unusual case of glaucoma care in a patient with previous anterior ciliary sclerotomy and scleral expansion procedure.

Report of a Case. A 59-year-old white man had ocular discomfort and evidence of bleb leakage in the left eye. Two years before consultation, he underwent anterior ciliary sclerotomy for presbyopia in both eyes. Six months later, his left eye required glaucoma medication for increased intraocular pressure (IOP). He subsequently underwent a scleral expansion procedure (SEP) for his left eye in an attempt to restore the accommodation and reduce the IOP. Nonetheless, his IOP remained uncontrolled, and he required a trabeculectomy without antimetabolite.

Examination of his left eye revealed best-corrected visual acuity of 20/100 and IOP of 10 mm Hg. A small superior conjunctival bleb was noted to be thin, leaking, and extending 3 mm anteriorly onto the superior cornea. The surrounding conjunctiva was hyperemic, scarred, and retracted. Deep conjunctival scars were associated with the insertion sites of 4 silicone expansion bands. Two bands were exposed, and 1 was extruded (Figure 1).

Bleb revision was performed by excision of the anterior extension onto the cornea and the leaky avascular portion of the bleb, with mobilization of the surrounding conjunctiva to cover the trabeculectomy site. The conjunctiva was secured to the limbus with interrupted 9-0 polyglactin sutures at 2 wings. The conjunctiva overlying the expansion bands was dissected, and the bands were removed. Four weeks after surgery, visual acuity improved to 20/30, with the IOP controlled at 12 mm Hg (Figure 2).

Figure 6. Electron microscopy examination discloses an unremarkable Bruch membrane covered by retinal pigment epithelium measuring 8.3 mm in height. The pigment epithelium has normal-appearing melanin pigment granules and numerous lipofuscin granules (arrows) (original magnification ×8000). BM indicates Bruch membrane; CC, choriocapillaries.
Comment. Anterior ciliary sclerotomy involves radial scleral incisions overlying the ciliary body in the 4 oblique quadrants to restore lost zonular tension, thereby improving accommodation according to the controversial Schachar theory.1,2 In SEP, silicone bands are inserted at the depth of the sclerotomy or tunneled into the sclera to counteract rapid regression of the surgical effect of anterior ciliary sclerotomy.2,3 There are conflicting reports that SEP may have an effect on reducing the IOP.2,3 In a prospective, nonrandomized study6 of patients with glaucoma uncontrolled with medication, a median IOP reduction of 7 mm Hg was reported. However, in another prospective small case series, IOP was not modified after SEP.3 The proposed mechanisms of IOP reduction include creation of a localized ciliochoroidal detachment, expansion of the anterior chamber angle, and facilitation of aqueous outflow.2,3 The decreased scleral rigidity from multiple scleral incisions may also result in an artificially lowered IOP by applanation. However, scarring and hardening of sclera or closure of the proposed ciliochoroidal space may ultimately reverse the initial effect.

In our patient, 3 implanted bands were exposed or extruded. Erosion of a foreign body through the conjunctiva is common when the implant is close to the limbus. Even with additional protection with scleral or pericardial grafts to cover the extraocular portion of glaucoma drainage devices near the limbus, glaucoma specialists frequently observe thinning of the overlying graft and conjunctiva over time.

In this case, the conjunctival reaction and foreign body reaction associated with prior peritomies and implanted expansion bands led to aggressive scarring of tissue surrounding the filtration bleb. This tight scarring limits the aqueous filtration and is frequently associated with progressive thinning and bleb leakage. In patients who have undergone a previous SEP, the expansion bands should be removed to eliminate the continuous foreign body reaction in the limbal quadrants and to improve the chance of success with glaucoma surgery.

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West Nile Virus Infection

Uveitis Associated With West Nile Virus Infection

In a recent case report, vitritis and chorioretinitis were described in a patient with presumed West Nile virus infection. We describe herein a patient in an outpatient neuro-ophthalmology clinic with iritis and vitritis and a confirmed acute West Nile virus infection. The 56-year-old woman developed blurry vision, floaters, malaise, and myalgias within a few weeks of mosquito exposure. Examination revealed bilateral nongranulomatous anterior uveitis and vitritis. Serological tests for West Nile virus revealed markedly elevated IgM and slightly elevated IgG. Plaque reduction neutralization testing by the Centers for Disease Control and Prevention (Fort Collins, Colo) confirmed West Nile virus infection. St Louis encephalitis virus test results were negative. Our findings demonstrate that nongranulomatous uveitis can occur in the setting of confirmed acute West Nile virus infection.

West Nile virus was first identified in a Ugandan patient in 1937. However, the first reports of the virus in the Western hemisphere did not surface until a meningoencephalitis outbreak in the New York City metropolitan area in 1999. The virus has continued to spread, and the large outbreak in the midwestern United States in 2002 resembled the outbreak of St Louis encephalitis in 1975. Although most patients infected with West Nile virus remain asymptomatic or develop only minor symptoms, potentially fatal meningitis or encephalitis occurs in approximately 1 in 150 infected persons.

The ophthalmologic characteristics consist of eye pain, photophobia, conjunctival hyperemia, and papilledema. Recently, a case report documented vitritis and chorioretinitis in a patient with possible West Nile virus infection. We describe herein a woman with nongranulomatous uveitis and concomitant acute West Nile virus infection, confirmed by plaque reduction neutralization testing.

Report of a Case. A 56-year-old woman with a medical history significant for hypertension, coronary artery disease, Graves disease, type 1 diabetes mellitus, hepatic and pancreatic insufficiency, seizure disorder, and mitochondrial myopathy complained of blurry vision in both eyes and black spots in the lower field of her left eye for 4 days. She denied flashing lights, eye or neck pain, fever, chills, or new neurologic deficit. Several mosquitoes had bitten her in the previous 3 weeks. She admitted to nausea, extreme malaise, muscle weakness, and soreness.

On examination, she was alert, calm, oriented, and cooperative. Her uncorrected visual acuities were 20/25 OD and 20/30 OS. Intraocular pressure was 17 mm Hg in each eye. Pupils were round, equal, and reactive, without relative afferent pupillary defect. Findings on slitlamp examination were unremarkable except for mild cataracts. Dilated fundus examination revealed bilateral mild nonproliferative diabetic retinopathy and no evidence of choroidal infiltrates. Her automated visual fields were normal in the right eye, with a new inferior arcuate in the left eye from previous perimetry. Her neurologic examination results were unremarkable.

She returned to the emergency department 5 days later with progressive blurring of vision in both eyes and black spots also now in the right eye. Magnetic resonance imaging of the brain yielded normal findings. Visual acuities were 20/25 OD and 20/40 OS. Her intraocular pressure was elevated to 23 mm Hg in the right eye and 32 mm Hg in the left eye. Slitlamp examination and dilated fundus examination demonstrated trace conjunctival hyperemia, bilateral nongranulomatous keratic precipitates, and moderate anterior chamber and anterior vitreous inflammation. Laboratory investigations showed normal results on complete blood count, chemistry panel (including blood glucose), thyroid function test, Westergren sedimentation rate, C-reactive protein, and Cryptococcus antigen test. West Nile virus serologies were positive by enzyme-linked immunosorbent assay with an IgG result of 1.3 (normal, <2.00), and an IgM result of 6.38 (normal, <2.00). Plaque reduction neutralization testing (performed at the Centers for Disease Control and Prevention) confirmed a positive infection with West Nile virus and excluded an infection with St Louis encephalitis virus. She was treated with 1% prednisolone acetate in each eye on an hourly basis for a week. One week later, her vision had improved to baseline. The keratic precipitates were gone, and the inflammation had improved. Her systemic symptoms and the uveitis improved during the next 3 weeks. The patient refused to have a lumbar puncture.

Comment. West Nile virus is a single-stranded RNA virus that belongs to the Japanese encephalitis serogroup of flaviviruses, including St Louis, Kunjin, and Murray Valley encephalitis. Identification of IgM in serum or cerebrospinal fluid by IgM antibody-capture enzyme-linked immunosorbent assay suggests a preliminary diagnosis of infection in humans. However, because of cross-reactions between antibodies to West Nile and St Louis encephalitis viruses in this assay, results must be confirmed by plaque reduction neutralization testing, a virus-specific assay. Our patient had virus-specific IgM and IgG antibody responses to West Nile virus demonstrated by IgM antibody-capture enzyme-linked immunosorbent assay and confirmed by plaque reduction neutralization testing.

Recently, the finding of vitritis and chorioretinitis in a patient with positive IgM antibody to West Nile and St Louis encephalitis viruses was reported. These authors did not present evidence of convalescence titers or plaque reduction neutralization testing confirmation of West Nile virus infection to definitively prove the diagnosis and thus acknowledged that their case was presumptive. Their patient could conceivably have been infected with St Louis encephalitis virus, with symptoms mimicking West Nile virus infection. Nevertheless, their case report suggests that flaviviruses in general may cause uveitis.