**Bacillus cereus Endophthalmitis Secondary to Self-inflicted Periocular Injection**

Endophthalmitis is an ocular emergency that can have a devastating outcome. The poor prognosis is often related to rapid progression of the disease process and a relative delay in diagnosis due to the wide array of clinical symptoms and signs.\(^1\) Although endophthalmitis is most often related to surgical intervention, endogenous sources are identified in 2% to 15% of cases.\(^1\) Intravenous drug-related endophthalmitis is most commonly caused by *Bacillus cereus*.\(^2,3\) We report a case of *B. cereus* endophthalmitis secondary to periorbital drug injection that resulted in spontaneous lens subluxation. This case underscores the difficulty in making an expeditious diagnosis when there is an uncertain medical history.

**Report of a Case.** A 29-year-old male prison inmate had a 12-hour history of vomiting associated with pain, redness, and impaired vision of the left eye. The patient denied trauma and drug abuse. Medical history was remarkable for active interferon therapy for hepatitis C. The right eye was normal. The left eye had light perception vision, 360° perilimbal injection, mid-dilated pupil, and dull red reflex. There were no signs of penetrating injury. The angle was closed with an intraocular pressure of 61 mm Hg. A toxicology screen test result was positive for amphetamine. The aspirate showed gram-positive rods, and the culture revealed *B. cereus/Bacillus thuringiensis*. Intravenous vancomycin hydrochloride, ceftriaxone sodium, and clindamycin phosphate supplemented intraocular injections of vancomycin and ceftazidime. A normal echocardiogram ruled out a cardiac source of bacteria. On the fourth day, spontaneous scleral perforation occurred with extrusion of purulent uveal contents. The eye was eviscerated. Weeks later, 2 prison guards reported to a case worker that prior of the choroid and retina, fluid posterior to the sclera, inflamed extraocular muscles, and a nondisplaced lens.

Laser iridotomy, topical cycloplegics, and steroids failed to reduce the intraocular pressure or reverse the ocular inflammation (Figure 1). On the second day, an orbital computed tomography scan demonstrated marked scleral thickening, enlarged extraocular muscles, and subluxation of the lens (Figure 2). Suspicion of endophthalmitis led to vitreous aspiration. The aspirate showed gram-positive rods, and the culture revealed *B. cereus/Bacillus thuringiensis*. Intravenous vancomycin hydrochloride, ceftriaxone sodium, and clindamycin phosphate supplemented intraocular injections of vancomycin and ceftazidime. A normal echocardiogram ruled out a cardiac source of bacteria. On the fourth day, spontaneous scleral perforation occurred with extrusion of purulent uveal contents. The eye was eviscerated. Weeks later, 2 prison guards reported to a case worker that prior...
to the episode the patient had been observed injecting drugs into the periorcular tissues.

**Comment.** To our knowledge, this is the first reported case of endophthalmitis secondary to presumed illicit periorcular drug injection and the first reported case of lens dislocation associated with *B. cereus* endophthalmitis. Unreliable patient history delayed correct diagnosis and contributed to a poor outcome, but retrospective review of the initial signs and symptoms and subsequent eyewitness reports led us to suspect orbital injection. Inadvertent ocular penetration and intraocular injection may have caused acute angle closure at the patient’s initial visit. Periorcular injection or direct inoculation of the vitreous body with a dirty needle in turn progressed to *B. cereus* endophthalmitis that rapidly led to lens dislocation. Although other mechanisms may result in endophthalmitis, the lack of systemic sepsis, other infectious sources, or obvious trauma near the globe make these etiologies less probable. Our report emphasizes the virulent nature of *B. cereus* and includes severe endophthalmitis associated with a physical obstruction of the retinal vessels.

**Vitreous Surgery for Central Retinal Artery Occlusion**

Central retinal artery (CRA) occlusion is a devastating disease for which conventional therapies are often relatively ineffective. Since CRA occlusion is usually caused by a physical obstruction in the CRA, we propose a mechanical means of relieving the obstruction. We postulate that cannulation of the CRA with a stylet during vitreous surgery may disrupt the obstructive agent and restore blood flow.

**Report of a Case.** A 68-year-old man with diabetes developed CRA occlusion in his left eye. On initial examination, his best-corrected visual acuity was counting fingers at 0.5 m OS. Funduscopy revealed a cherry red spot and severely narrowed retinal arteries. At approximately 30 hours after the onset of vision loss, conventional treatments had still resulted in no improvement in vision. The patient declined the option of selective thrombolysis. The option of vitrectomy with vessel cannulation and thrombus disruption under local anesthesia was then offered. The patient understood the experimental nature of this treatment and gave his informed consent.

After vitrectomy, one of us (W.M.T.) used a microvitreoretinal blade to penetrate the arterial wall at the central bifurcation of the retinal artery (Figure 1). No notable hemorrhage was noted. A 50-gauge flexible stylet made of nickel titanium was extended from a 19-gauge support shaft and used to cannulate the CRA through the arteriotomy site. Cannulation was confirmed by the ease of passage of the stylet through the CRA. The stylet was moved using forward, backward, and circular motions, and then withdrawn. A small amount of semiclotted blood emerged from the arteriotomy site. The caliber of the superior retinal arteries increased partially after cannulation; however, the inferior retinal arteries remained severely narrowed. Because it was unclear how the procedure might affect the risk of ocular neovascularization, peripheral endophotocoagulation was performed prophylactically to minimize the risk.

Postoperatively, the patient was given oral aspirin, 325 mg daily. Examination findings on day 2 showed a visual acuity of counting fingers at 1.8 m. The appearance of the retinal vessels had not changed significantly since the procedure. By the next examination on day 10, the caliber of the retinal vessels had returned to normal (Figure 2). Angiographic images revealed a normal retinal circulation time. The latest follow-up examination findings at 4 months showed a corrected visual acuity of 20/25 OS. The cherry-red spot had disappeared. A laboratory evaluation revealed an atheromatous plaque in the thoracic aorta.

**Comment.** Cannulation of the CRA is a novel surgical approach that allows direct mechanical access to the site of obstruction. This method presents the advantage of avoiding the risk of neurological complications associated with selective thrombolysis. In this case, immediate improvement in blood flow was modest. It seemed that the thrombus was only partially disrupted. In the future, to relieve the obstruction more completely, one might consider using a longer stylet or an infusion cannula to deliver thrombolytic agents into the CRA. The excellent recov...
Leber’s Hereditary Optic Neuropathy Masquerading as Retinal Vasculitis

Leber’s hereditary optic neuropathy (LHON) is a maternally inherited mitochondrial disease caused by several specific point mutations in mitochondrial DNA. The mutation at nucleotide position 11778 was the first point mutation to be identified and is the most common point mutation associated with LHON. For decades the diagnosis of LHON was dependent on the presence of classic ophthalmoscopic findings of circumpapillary telangiectatic microangiopathy, swelling of the nerve fiber layer around the disc (pseudopapilledema), and the absence of leakage from the disc on fluorescein angiography. Genetic analysis allows characterization of the clinical spectrum of LHON through the identification of cases that in the past would have remained undiagnosed. We report a new finding of peripheral retinal phlebitis associated with the 11778 mutation in LHON.

Report of a Case. A 17-year-old African American woman with high-myopia was examined for a 5-month history of gradually declining vision in both eyes. There was no history of multiple sclerosis or LHON in family members. Visual acuity was 20/100 OD and 20/400 OS.

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was a left relative afferent pupillary defect and dyschromatopsia detected by Ishihara color plate testing in each eye. Slitlamp examination results from both eyes were unremarkable. There were trace vitreous cells in both eyes and vitreous inflammatory condensates located inferiorly in the left eye, but no pars plana snowbanks were observed in either eye. Goldmann visual field testing showed bilateral central scotomas. Funduscopic examination revealed a right-tilted, dysplastic optic nerve and left optic nerve temporal pallor (Figure 1). The clinical maculae of both eyes were unremarkable. The retinal periphery of both eyes revealed inactive vasculitis with vascular sheathing of the retinal veins (Figure 2), but no evidence of vaso-oclusion. There was no arteriovenous shunting, retinal neovascularization, or intraretinal hemorrhage in the retinal periphery of either eye. A fluorescein angiogram of both eyes appeared normal with no retinal vascular leakage or staining.

The results of diagnostic laboratory tests, including a chest x-ray, VDRL test, fluorescent treponemal antibody absorption test, sickle cell test, tests of purified protein derivative (tuberculin), angiotensin-converting enzyme, lysozyme, Lyme antibodies, and serum electrophoresis, were unremarkable. Also unremarkable were test results for antithrombin III, prothrombin time, activated partial thromboplastin time, protein C, protein S, protein C resistance, antinuclear antibody, antiphospholipid antibodies, antinuclear antibodies, antineutrophil cytoplasmic antibody, and hepatitis B surface antigen.

The patient was given prednisone (60 mg/d) but failed to return until 2 months later with progressive visual loss and severe frontal lobe headaches. Visual acuity was now 2/200 OD with light perception OS. Ocular examination and fluorescein angiography were unchanged. Magnetic resonance images of the brain and lumbar puncture were unremarkable with no evidence of multiple sclerosis. Molecular genetic analysis for known LHON mutations was positive for the 11778 mutation. The patient was treated with coenzyme Q supplementation (300 mg/d) for 6 months but her vision failed to improve in either eye. The patient has not developed clinically positive multiple sclerosis.

Comment. Leber’s hereditary optic neuropathy may masquerade as tobacco-alcohol amblyopia1 or Stargardt’s maculopathy.2 The 11778 mutation has been associated with a demyelinating neurological condition indistinguishable from multiple sclerosis.3 The findings from our examination of this patient, including vitritis, vasculitis, and optic neuritis are commonly seen in patients with multiple sclerosis4 or multiple sclerosis–like syndromes such as pars planitis,5 further supporting an association between multiple sclerosis and LHON mutation 11178. These patients may be at high risk of developing multiple sclerosis years after the ocular findings are identified.

This case extends the clinical spectrum of LHON and does not indicate a coincidental occurrence of some other retinal inflammatory process with the mitochondrial mutation 11778. A complete systemic evaluation for vasculitis, hyperco-

**Figure 1.** A, Tilted, dysplastic optic nerve. B, Optic nerve temporal pallor.

**Figure 2.** Old, inactive retinal phlebitis as seen in the periphery of both eyes.
agulative states, collagen vascular disease, and infection was unrevealing. This report lends support to the theory that LHON is a neuroretinopathy with a broad spectrum of genotype-specific phenotypes.

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Ophthalmological Numismatics

A look at the past...

Albrecht von Graefe, 1829-1870, the renowned ophthalmologist of Berlin and son of Carl F. von Graefe, is honored by the Ophthalmological Society of Heidelberg by being portrayed on its award medal, which is presented every 10 years. This, the first piece that was awarded in 1886, was presented to von Helmholtz. Von Graefe was among the first to use the newly invented ophthalmoscope of von Helmholtz. He announced a cure for acute glaucoma by surgical iridectomy and improved techniques for cataract and strabismus surgery. He is also the founder of the journal Archiv Fur Ophthalmologie. This medal was engraved by E. Weigang and F. Hartzer. The obverse (Figure 1) depicts von Graefe’s bust facing right surrounded by an inscription. The reverse (Figure 2) depicts an inscription surrounded by a wreath with an inscription above. The original prize medal is struck in gold. Bronze copies were also made.

Courtesy of: Jay M. Galst, MD, 30 E 60th St, New York, NY 10022.