rial of the embolus enters ocular circulation and the internal carotid artery through retrograde arterial flow after the inadvertent injection of hyaluronic acid into the dorsal nasal artery. Under the injection pressure of a syringe, the material is forced retrograde into the ophthalmic artery and internal carotid artery with subsequent distal movement into brain arteries. This peculiar case should be seen as a warning to all ophthalmologists and plastic surgeons that such widely performed simple procedures can cause devastating damage. To minimize this risk, intradermal injection for augmentation of the glabellar region should be given superficially and medially, and aspiration is also recommended. Patients should be informed of the possibility of this rare complication. Headache and ocular pain after injection could be warning signs of this complication, and physicians should be alert to stop further injections immediately if issues should arise while performing such procedures to minimize the damage.

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Choroidopathy Associated With AIDS
Ciliochoroidal effusions occur when fluid accumulates within the suprachoroidal space. Inflammatory effusions causing bilateral angle-closure glaucoma have been reported as an exceedingly rare manifestation of the human immunodeficiency virus (HIV).1-3 We describe a patient with AIDS-associated bilateral choroidal effusions with a clinical course marked instead by ocular hypotony.

Report of a Case | A 47-year-old man with AIDS (CD4 cell count, 33/μL; viral load, >1 × 10⁶/mL) visited the ocular emergency department with bilateral, painless peripheral vision loss. Findings on a previous workup at an outside hospital were unremarkable (negative rapid plasma reagin and fluorescent treponemal antibody absorption tests, negative hepatitis panel, negative urine and blood cultures, negative Epstein-Barr virus and cytomegalovirus serum antibody titers, normal brain magnetic resonance imaging findings). Visual acuity was 20/20 OU and intraocular pressure was 6 mm Hg OU. Anterior segment examination findings were significant for trace anterior chamber cell on the right and pseudophakia in both eyes. Posterior segment examination findings were notable for trace vitritis on the right and bilateral ciliochoroidal effusions nasally (Figure 1A and B). A purified protein derivative test, *Bartonella* species serum
titers, and aqueous humor polymerase chain reaction analysis for Epstein-Barr virus, herpes simplex virus 1, herpes simplex virus 2, cytomegalovirus, and varicella-zoster virus were all negative. During the subsequent week, the choroidal effusions worsened and eventually involved the posterior pole in both eyes. Visual acuity decreased to hand motions OU and intraocular pressure decreased to 1 mm Hg OU. Ultrasonography repeatedly revealed no scleral thickening or infiltration of the Tenon capsule.

The patient was consequently admitted to the hospital, where his vital signs and nonophthalmic physical examination results were unremarkable. Laboratory analysis showed a low-normal white blood cell count of 4200/μL (to convert to ×10⁹ per liter, multiply by 0.001) with negative blood cultures. Comprehensive metabolic panel, erythrocyte sedimentation rate, C-reactive protein, magnetic resonance imaging of his brain, and lumbar puncture (with cultures for virology, bacteria, and mycology) were all normal. The patient was then started on highly active antiretroviral therapy and intravenously administered methylprednisolone sodium succinate, 1 g/d for 3 days, which was tapered to oral prednisone, 30 mg/d. During the subsequent 2 months of follow-up, the patient was gradually tapered off prednisolone acetate eyedrops and oral prednisone as his choroidal effusions resolved.

At last follow-up, approximately 4 months after the initial visit, the patient’s central visual acuity was 20/30 OD and 20/50 OS. His intraocular pressure improved to 11 mm Hg OD and 10 mm Hg OS. The patient’s anterior chambers were quiet, although 1+ vitreous cell and fibrin were noted in the vitreous cavity of his left eye. His choroidal effusions resolved, although large, wedge-shaped chorioretinal pigmentary changes and a tessellated retinal appearance extended from the periphery into the macula of both eyes (Figure 1C and D). Fluorescein angiography (Figure 2A and B) and indocyanine green angiography (Figure 2C and D) of the right eye both revealed early hypofluorescence in regions of pigmentary lesions. Later frames revealed hyperfluorescence on fluorescein angiography but continued hypofluorescence on indocyanine green angiography. Similar findings were noted in the left eye. Optical coherence tomography (Figure 3A and B) and enhanced depth imaging optical coherence tomography (Figure 3C and D) of the right and left eyes demonstrated regions of inner neurosensory retinal loss, retinal pigment epithelial thinning, and diffusely attenuated choroid layers.

**Discussion** | We describe a patient with AIDS-associated bilateral ciliochoroidal effusions. Although such effusions have been described in the literature,1,2 the striking fundus findings in our patient have not been reported, likely from AIDS conferring an abbreviated life expectancy (with limited patient follow-up) in the decades during which such prior reports were published.

The triangular wedge-shaped pigmentary changes, tessellated retinal appearance, and their distribution indicate the
The early fluorescein hypofluorescence and late hyperfluorescence of these hypopigmented regions of the right eye are also consistent with impaired choroidal perfusion and late staining of necrotic choroidal interstitia. The left eye demonstrated similar findings but with a relatively mottled pattern, implying a more severe and/or chronic degree of hypoperfusion. The hypofluorescence in both early and late frames of the indocyanine green angiography (more extensively in the left eye) further corroborates choroidal ischemia with probable infarction involving the overlying retinal pigment epithelium and outer retina. Enhanced depth imaging optical coherence tomography demonstrates severe atrophy of the affected choroid (Figure 3), with this being the first report, to our knowledge, of choroidal infarctions imaged using this modality.

The patient’s choroidal effusions likely resulted from both choroidal ischemia and arterial endothelial inflammation. The early nasal localization of the effusion (Figure 1A) as well as the more severe nasal distribution of the later pigmentedary changes in the right eye (Figure 2A) indicate the nasal long posterior ciliary artery as its earliest and most severely affected artery. The eventual pigmentedary changes in the left eye suggest that both long posterior ciliary arteries were severely affected in that eye (Figure 2B). The patient’s ocular hypotony is also consistent with long posterior ciliary artery ischemia, putatively decreasing aqueous production through ciliary body hypoperfusion, which in turn abets suprachoroidal fluid accumulation when combined with choroidal endothelial dysfunction and ischemia.

While HIV generally causes ocular morbidity through opportunistic infection, our case implicates the virus itself. Specifically, HIV vasculopathy denotes arterial toxic effects through endothelial exposure to HIV-infected leukocytes, immune complexes, HIV proteins, and proinflammatory cytokines, all of which generate intimal hyperplasia and endothelial dysfunction. The net result is chronic inflammation of the arterial wall with increased vessel permeability and luminal stenosis. While described in the central nervous system and coronary arteries, our case is the first choroidal display of HIV vasculopathy, to our knowledge, and suggests it as the cause of the enigmatic AIDS-related ciliochoroidal effusions reported in the literature more than 20 years ago. While treatment involves first excluding opportunistic infection and initiating highly active antiretroviral therapy, antiretrovirals themselves have an attendant elevated risk of vascular occlusion. Careful monitoring of the patient’s posterior segment is therefore indicated, with consideration made toward...
reducing stroke risk factors and, as in this case, using a prophylactic course of systemic steroids.

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Ophthalmic Resident Education on Preventable Surgical Errors

An important objective of ophthalmic graduate medical education (GME) is to provide surgical training to residents so they are competent to enter comprehensive ophthalmic practice.¹ Training to reduce the risk of surgical errors is central to this objective. Errors involving the wrong patient, wrong site, or wrong procedure can have devastating consequences²,³ and are largely preventable by adherence to specific protocols.⁴,⁵

These errors can have catastrophic consequences for patients, such as vision loss, retinal detachments, and blindness. The primary method for preventing these errors is through the use of specific protocols that are implemented during training. These protocols include but are not limited to preoperative identification of the patient and site, proper marking, and the use of time-out procedures. These protocols are designed to reduce the risk of these errors and are an integral part of the training process in ophthalmic surgery.

Figure 3. Optical Coherence Tomographic Images and Enhanced Depth Imaging Optical Coherence Tomographic Images

A

B

C

D

Optical coherence tomography of the right (A) and left (B) eyes demonstrates relatively normal inner retinal architecture. Subfoveally, the external limiting membrane and photoreceptor inner segment/outer segment lines of both eyes appear preserved. Temporally, these layers appear sporadically disrupted in both eyes, with similar areas of loss nasally in the left eye. Both eyes also demonstrate temporal retinal pigment epithelium and Bruch membrane complex thinning with hyperreflective material overlying this layer and increased choroidal reflectivity underlying it. Enhanced depth imaging optical coherence tomography of the right (C) and left (D) eyes demonstrates diffuse thinning of the choroid in both eyes.