LASIK-Associated Visual Field Loss in a Glaucoma Suspect

We report a case of visual field loss first noted after laser in situ keratomileusis (LASIK). One similar case has been described.1 Since LASIK involves brief iatrogenic elevation of intraocular pressure (IOP), we are concerned about the possible rare instances of this occurrence in future patients.

Report of a Case. A 47-year-old high myope received a diagnosis of ocular hypertension in 1979 with an IOP of 30 OU. Her mother and sister had glaucoma. Treatment with 0.5% timolol maleate maintained her IOP in the high teens except for an occasional IOP in the low 20s. Findings on Humphrey 24-2 visual field testing were normal on June 4, 1997 (Figure 1).

On June 16, 1999, an experienced surgeon (R.S.R.) performed LASIK for correction of 10.5 diopters (D) of myopia in the right eye and 9.5 D of myopia in the left. Pachymetry measured a 473-µm thickness OU preoperatively and a 360-µm thickness OD and 390-µm thickness OU postoperatively. Dur-
cation with the LASIK procedure, rather than due to concurrent progression of the underlying disease.

The IOP has measured in the low teens OU when treated with 0.5% timolol and latanoprost, though her readings may be falsely low owing to her thin corneas. Visual field testing showed on December 1, 1999, showed no progression of the scotoma (Figure 3).

Comment. An experienced LASIK surgeon can limit the duration of the iatrogenic increase in IOP during LASIK to approximately 10 seconds. During this time a mechanical suction ring achieves a pressure of approximately 80 mm Hg. Although the suction ring achieves a pressure of approximately 10 seconds, the surgeon can limit the duration of the pressure elevation to approximately 10 seconds. The stress of pressure elevation is of shorter duration, but greater magnitude compared with gravity inversion or high-resistance wind instrument playing, activities that have been associated with elevated IOP and abnormal visual fields.

Inspection of the optic nerve prior to LASIK may allow identification of some of the patients who are at risk. Features of the optic nerve that may be of concern are the same variables that identify early glaucomatous optic nerve damage: vertical cup–disc diameter ratio corrected for optic disc size, total neuroretinal rim area, rim–disc area ratio, and cup–disc area ratio corrected for disc size. Patients with glaucoma, a family history of glaucoma, as well as glaucoma suspects should be cautioned of this risk prior to the performance of LASIK. For many of these patients, photorefractive keratectomy, intrastromal corneal ring segments, or continued use of contact lenses or eyeglasses may offer satisfactory vision without subjecting the optic nerve to the small but real risk of pressure-associated visual field loss.

Howard S. Weiss, MD, MPH
Roy S. Rubinfeld, MD
John F. Anderschat, MD
Washington, DC


Topiramate-Induced Acute Myopia and Retinal Striae

Drug-induced acute myopia is a rare phenomenon of debatable etiology. Its causative mechanism has never been fully illustrated. Topiramate is a new class of antiepileptic drug that has been deemed relatively safe by studies to date. Its major documented adverse effect is renal stones. We report an unusual case of acute myopia induced by topiramate in a 15-year-old patient with epilepsy, and include comments regarding the possible mechanism of action.

Report of a Case. A 15-year-old boy visited his ophthalmologist with a sudden decrease in distance vision (in both eyes) during the span of 24 hours. He stated that he awoke with decreased distance vision, but with preservation of near vision. His ophthalmic history was significant for “lazy eye” in the left eye, and he did not wear glasses at the time. His medical record was significant for a history of poorly controlled epilepsy. His neurologist discontinued valproic acid and gave him a regimen of topiramate 1 week previously to attempt better seizure control. His initial dose was doubled 2 days before onset of symptoms. His only medication included 25 mg of topiramate, 2 tablets by mouth twice daily.

On initial examination, his best-corrected visual acuity was 20/600 OD and 20/400 OS, improving with pinhole disc examination to 20/40 OD and 20/100 OS. The pupils were healthy with no sign of afferent pupillary defect in either eye. The anterior segment examination results were normal except for the presence of shallow anterior chambers. After administration of 2.5% phenylephrine hydrochloride and 1% cyclopentolate hydrochloride, a dilated fundus examination revealed bilateral retinal striae radiating from the fovea that were worse in the left eye (Figure 1). Cycloplegic refraction was −7.75/+1.75 × 80° OD and −6.75/+1.75 × 95° OS. The patient discontinued the topiramate that evening, and he refused to use it again.

On examination 1 day later, he stated that his vision had improved. Ophthalmic examination revealed a visual acuity of 20/80 OD and 20/400 OS. Cycloplegic refraction was −3.25/+0.75 × 94° OD and −2.75/+1.25 × 93° OS. Fundus examination results were unchanged.
At 1-week follow-up, the patient was still not taking topiramate. His visual acuity was 20/20 OD and 20/200 OS, which he stated was his normal visual acuity. Cycloplegic refraction was +0.50/+1.00 × 90° OD and −0.50/+1.75 × 70° OS. Fundus examination was normal (Figure 2).

At 1-month follow-up, the ophthalmic examination results were unchanged, and he had no new complaints.

Comment. Acute myopia has been previously described in association with several medications.1-9 Common medications that have been known to induce myopia include antibacterial sulfonamide preparations, acetazolamide, chlorthiazide, ethoxzolamide, and chlorothalidone.2-6 Two cases in the literature describe a transient myopia associated with topiramate.7-9 Various theories have been suggested for these acute changes, and the most logical of these is spasm of accommodation. In our patient, however, the pupils never became miotic, and significant myopia persisted after cycloplegia, which would eliminate accommodative spasm as a cause. The mechanism of action is most likely related to a disturbance of the osmotic state of the lens and concomitant alteration of the refractive index.3,10 Pallin and Ericsson10 did an elegant ultrasonography study of a patient who had acute myopia secondary to Hygroton (chlorthalidone; Rhone-Poulenc Rorer Pharmaceuticals Inc, Collegeville, Pa). They used a 5-mm crystal attached to a corneal contact lens to record the corneal echo, and the ultrasound image was found to be accurate to within 0.1 to 0.2 mm. In the acute myopic state (compared with emmetropia), it showed an increase in thickness of 0.3 mm in the lens of the right eye and 0.4 mm in the left lens.

Topiramate is a structurally novel antiepileptic drug. Its chemical structure is 2,3:4,5-bis-O-(1-methylethylidene)-β-D fructopyranose sulfamate, which is a sulfamated derivative of fructose, a naturally occurring monosaccharide. Topiramate is rapidly absorbed (serum levels peak after oral dose in 1-4 hours), has good bioavailability, a relatively long half-life (20-30 hours), and is predominantly renally excreted.11 Its main indication is in the treatment of refractory patients with partial seizures with or without secondary generalization.12 The main recognized adverse effects include dizziness,
ataxia, psychomotor slowing, weight loss, and nephrolithiasis. Placebo-controlled trials of topiramate as an adjunctive therapy for seizures in 1757 adults and 310 children reported ocular adverse effects, including diplopia, nystagmus, conjunctivitis, accommodation abnormalities, mydriasis, and myopia. Topiramate may also affect visual perception. It has several mechanisms of action but has documented effects on sodium and chloride movement that can interfere with ionic concentration in various tissues, including the crystalline lens.

In our patient, the acute myopia began to resolve 24 hours after discontinuation of the drug, which is compatible with its half-life. The acute retinal striae seen have been described in association with other drugs, but are not the cause of the myopia. Elevation of the retina secondary to fluid could be consistent with drug-induced altered membrane potential, but would cause hyperopia rather than myopia. The fact that we noted a shallow anterior chamber points to a swelling of the crystalline lens owing to altered ionic concentration. Unfortunately, at the time he was seen, we did not have high-resolution ultrasound biomicroscopy available to attain an accurate measurement.

This is the first documented case of acute myopia with fundus photographs of retinal striae secondary to topiramate we know of. We suggest that patients with acute refractive changes who are using topiramate have a cycloplegic refraction examination performed, and if possible, an ultrasound to measure the anterior-posterior length of the lens. We cannot say if this effect is transitory, as our patient stopped the treatment himself and refused to restart it, but progression or recovery could be documented with serial ultrasonography measurements that are safe and noninvasive.

Harsh A. Sen, MD
Henry S. O’Halloran, FRCSI
William B. Lee, MD
Lexington, Ky

Corresponding author: William B. Lee, MD, Department of Ophthalmology, University of Kentucky, 801 Rose St, Suite E321, Lexington, KY 40536 (e-mail: lee0003@aol.com).


Ocular and Cerebral Ischemia Following Facial Injection of Autologous Fat

Periocular and paranasal injections of various substances are becoming increasingly common since more procedures are being performed under local anesthesia. Their serious visual adverse effects are rare but potentially devastating when they occur. Because of the multiple anastomoses between the vascular supply of the face and orbit, the potential for retrograde embolization of substances exists. We believe we report the first case of paranasal autologous fat injection resulting in middle cerebral, ophthalmic artery, and central retinal artery occlusion.

Report of a Case. A 43-year-old right-handed man received an injection of autologous fat in the tissue on the left side of the bridge of his nose to repair a soft tissue defect, a result of a previous accident. The autologous fat was obtained from the abdominal area. After anesthetizing the sites with 2% lidocaine without epinephrine, a large-bore cannula was used to inject the autologous fat deep into the soft tissue on the left side of his nose (1/2 mL), each nasolabial fold (3 mL each), and the upper and lower lips (3 mL). Within 10 minutes postinjection, he complained of eye and head pain, became disoriented, and lost vision OS. He also became aphasic with right-sided hemiparesis. There was no light perception OS and the left pupil was amaurotic. The left fundus showed a pale optic disc and widespread retinal whitening with visible emboli in several retinal arterioles (Figure 1). Preretinal and intraretinal hemorrhages were present inferiorly. The right eye remained unaffected. Ocular adnexa, ocular motility, and intraocular pressure were normal. The site of the initial injection underwent patchy necrosis over the ensuing 5 days (Figure 2). His neurological condition improved to normal, the left eye remained blind. The clinical features were consistent with fat embolism to branches of the left middle cerebral artery and the ophthalmic artery. The skin lesions indicated that distal arterial branches supplying the nose were also occluded.

Comment. Visual loss secondary to fat embolization is a recognized, although uncommon, complication of autologous fat injection. The cases in the literature have involved injection of autologous fat around the glabellar area. This is the first report of ophthalmic artery occlusion after a paranasal injection of autologous fat. We believe the foreign material was injected into one of the peripheral branches of the ophthalmic artery, probably the dorsal nasal artery. Under the injection pressure of the syringe, the fat was likely forced retrograde into the ophthalmic and internal carotid arteries with subsequent distal movement of the emboli into the left middle cerebral, central retinal,
and posterior ciliary arteries. Because the peripheral arteries of the face are small and collapsible, blood may not appear in the delivering syringe during aspiration despite the presence of the needle within an arterial lumen.

Helen V. Danesh-Meyer, FRACO
Peter J. Savino, MD
Robert C. Sergott, MD
Philadelphia, Pa

Corresponding author and reprints:
Helen V. Danesh-Meyer, FRACO,
Neuro-ophthalmology Service, Wills Eye Hospital, 900 Walnut St, Philadelphia, PA 19107 (e-mail: hdm90@hotmail.com).


**Scleritis Occurring in Association With Takayasu Disease**

Takayasu disease (TD) (pulseless disease, aortic arch syndrome) is a rare but potentially life-threatening chronic giant cell vasculitis. We describe a patient with Takayasu disease who initially had scleritis. Associated with other systemic vasculitic conditions in approximately one half of cases, a relationship between TD and scleritis has not been well documented.

**Report of a Case.** A 36-year-old Filipino-Hawaiian woman had a 4-year history of fluctuating bilateral ocular redness and episodes of vision loss, along with intermittent numbness and stiffness in her left arm and neck, with exertion, palpitations, breathlessness, lightheadedness sometimes progressing to syncope, and fatigue. Her medical history included anemia.

Visual acuities were 20/25 OD and 20/20 OS. There was non-necrotizing sectorial scleral inflammation OS, as well as bilateral circumferential scleral thinning, but both corneas were healthy. Further ocular examination was unremarkable. Systemic examination revealed a systolic cardiac murmur, bilateral carotid and subclavian bruits, and diminished carotid and left arm pulses. Upper limb blood pressures were 160/90 (right arm) and 80/60 (left arm).

Laboratory results included an erythrocyte sedimentation rate of 102 mm/h and hypergammaglobulinemia. Syphilis serology was nonreactive. Antinuclear antibodies, rheumatoid factor, and anti-neutrophil cytoplasmic antibodies were not detected. Chest x-ray film showed an enlarged aortic knob. Aortography demonstrated dilatation of the arch and irregular stenoses of its branches, particularly the left common carotid and subclavian arteries (Figure).

**Figure 1.** A, Pallid disc edema and retinal whitening with interruption of several arteriole branches. B, Peripheral vascular occlusion with visible fat emboli in arterioles.

**Figure 2.** A, Site of initial injection. B, Necrosis 5 days later.
During this outpatient evaluation, the patient experienced a transient ischemic attack that caused expressive aphasia.

Treatment began with 3 intravenous methylprednisolone pulses (1 g/d), followed by high-dose oral prednisone (60 mg/d) that was slowly tapered, in combination with oral methotrexate (20 mg/wk) and folic acid supplementation and aspirin (80 mg/d). High-grade left common carotid stenosis prevented stenting, but stents have been deployed in the left subclavian and right innominate arteries. Two years after diagnosis, the patient was taking methotrexate, and remained systemically well with inactive scleritis.

Visual aberrations may also result from cerebral ischemia. To our knowledge scleritis has not previously been well recognized as a feature of this disease. Two reports describe scleritis in patients with TD, but both individuals also had Wegener granulomatosis, a well-known cause of scleritis. Causality cannot be proven, but the strong temporal relationship between active vasculitis involving sclera and great vessels strongly suggests a real association between TD and scleritis. Furthermore, temporal arteritis, the more common form of giant cell arteritis, may present with scleritis. Management of TD involves systemic immunosuppression for acute lesions and surgery for fibrotic pathology. Corticosteroids reduce inflammation but are not curative. Methotrexate may be useful in inducing a remission. Our patient’s scleritis and constitutional symptoms responded to immunosuppression, but surgery was required to relieve the symptoms of cicatrizing arterial stenoses. Aspirin was prescribed empirically, as there is no established role for anticoagulants in managing this disease. We document an association between TD and scleritis. Although rare, it is often possible to diagnose TD from the clinical history. A 5-year mortality rate as high as 35% requires that this condition not be overlooked. The diagnosis would have been expedited in our patient if there had been previous documentation of a clear association between TD and scleritis. Since acceptance of this paper, Jain et al have published a report that describes a case of TD occurring in association with scleritis.

Comment. Takayasu vasculitis generally involves large- and medium-sized arteries, in particular, the great vessels. A presumed T cell–mediated autoimmune inflammation of the vas vasorum progresses to fibrosis, and resultant arterial stenoses, occlusions, and aneurysms cause various ischemic symptoms. Estimated incidence is 2.6 cases per million persons per year, and most new patients are women of reproductive age, with an Asian or Hispanic ethnic background. Our patient’s case was typical, meeting all 6 American College of Rheumatology 1990 criteria. Three diagnostic criteria are required, including onset by age 40, claudication of the extremities, decreased brachial pulse(s), upper limb systolic blood pressure difference greater than 10 mm Hg, aortic or subclavian artery bruit(s), and angiographic narrowing of large arteries. Prominent constitutional symptoms are also common, as are the laboratory findings of anemia, raised erythrocyte sedimentation rate, and elevated serum γ globulins. Syphilis, an important differential diagnosis, was excluded by serological testing. There was no clinical or laboratory evidence for other autoimmune diseases clearly associated with scleritis, eg, rheumatoid arthritis and Wegener granulomatosis.

Ocular abnormalities are common in TD. Characteristic Takayasu retinal vasculopathy with potential neovascular complications occurs in a third of cases, and hypertensive retinopathy affects 20% of patients. Visual aberrations may also result from cerebral ischemia. To our knowledge scleritis has not previously been well recognized as a feature of this disease. Two reports describe scleritis in patients with TD, but both individuals also had Wegener granulomatosis, a well-known cause of scleritis. Causality cannot be proven, but the strong temporal relationship between active vasculitis involving sclera and great vessels strongly suggests a real association between TD and scleritis. Furthermore, temporal arteritis, the more common form of giant cell arteritis, may present with scleritis.

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