Corneal Ectasia After Laser-Assisted In Situ Keratomileusis

Laser-assisted in situ keratomileusis (LASIK) has become the most common surgical option to correct myopia. Certain preexisting conditions are contraindications for LASIK, and they include unstable vision, keratoconus, thin corneas, pregnancy, and autoimmune diseases. A careful evaluation can help select the good candidates and predict which patients will have a guarded outcome. We describe the clinical and histologic features of a patient who underwent penetrating keratoplasty due to progressively worsening vision from corneal ectasia after LASIK.

Report of a Case. A 32-year-old woman came to the Refractive Surgery Center, Wilmer Eye Institute, Baltimore, Md, seeking a second opinion after LASIK was done elsewhere. Her chief complaint was that her vision had worsened since surgery and she could not function anymore with any form of visual aids.

Her ocular history was significant for keratoconus suspected by 2 ophthalmologists but not by the last physician who recommended and performed bilateral simultaneous LASIK. Before LASIK, she wore rigid gas-permeable contact lenses until she became intolerant to those.

During the preoperative evaluation, an autorefractor revealed keratometry readings of 47.75 and 51.75 OD and 49.37 and 55.00 OS. Her manifest refraction revealed a visual acuity of 20/25 OD with −11.25+3.75/H11003 085 and 20/70 OS with −13.00+3.75/H11003 080. Her best-corrected visual acuity with rigid gas-permeable contact lenses was 20/20 OU. Preoperatively, her central corneal pachymetry readings were 511 µm OD and 482 µm OS, as obtained by a scanning slit measurement system (Orbscan Topography; Bausch & Lomb, Inc, Rochester, NY). She underwent full-correction LASHK in both eyes. One month postoperatively, the patient’s best-corrected visual acuity was 20/40 OD with −3.25+0.50/H11003 125 and 20/50 OS with −2.25+0.75/H11003 85.

When we first examined the patient, 5 months after the surgery, her best-corrected visual acuity was 20/200 OD with −10.50+0.50/H11003 90 and 20/100 OS with −9.75+0.75/H11003 90. Her central corneal pachymetry reading was 365 µm OU by ultrasonographic pachymetry. Corneal topography revealed central steepening in both eyes (Humphrey Visual Instruments, San Leandro, Calif) (Figure 1). Her keratometry readings, as indicated by the corneal maps, reached 55 and 61 OD and 54

Figure 1. Corneal topography of the right eye after laser-assisted in situ keratomileusis, showing central steepening. S-merid indicates the distance from the vertex (large crosshair) and its location on the semimeridian; CIM, corneal irregularity measure.
and 58 OS. No posterior corneal curvature measurements were obtained. On slitlamp examination, she had well-centered LASIK flaps with nasal hinges in both eyes. The corneal epithelium was dry centrally and stained with fluorescein. The stroma appeared thin and bulging forward (Figure 2). The lamellar interface appeared clear in both eyes. The rest of the anterior segment, including the anterior chambers, irides, and lenses, appeared normal. Intraocular pressures were 11 mm Hg OU by applanation tonometry. The results of her dilated retinal examination were unremarkable. Despite punctal plugs and the hourly use of preservative-free tears with lubricating ointment at night, her visual acuity continued to deteriorate. Corneal topography revealed that the surface of the right eye was more irregular. A contact lens fit was attempted, but the patient did not believe she could tolerate the lenses.

A diagnosis of corneal ectasia was made based on the progressive worsening of the cornea together with central thinning as measured by pachymetry, and penetrating keratoplasty was recommended because the cornea was too thin and dry to tolerate any other intervention. The patient underwent an uncomplicated corneal transplantation (8.0-mm donor size to 7.5-mm recipient size), and corneal tissue was sent for histologic studies (Figure 3).

The light microscopy reveals one margin of the corneal flap with a discontinuity of the Bowman layer and a slanted area of mild scarring (Figure 4). Looking at a central area, there is a faint delineation between the flap and the remainder of the stroma (Figure 5). The flap area has a slight increase in cellularity compared with the remainder of the stroma. Centrally, the cornea, including all layers, measured 317 µm in thickness, and the flap measured 81.5 µm.

Transmission electron microscopy showed the discontinuity of the Bowman layer (Figure 6) and a zone of irregular collagen with a slight increased number of fibrocytes in the plane between the flap and the remainder of the stroma (Figure 7). There was no endothelial damage.

Comment. This case represents an example of corneal ectasia after LASIK, for which the underlying risk factors for ectasia appear to be a combination of keratoconus, thin preoperative central corneas, and possibly a lamellar keratectomy.

Several cases of corneal ectasia after LASIK have been reported.1-3

![Figure 2](image2.png)

**Figure 2.** Slitlamp appearance of the cornea, showing stromal thinning and bulging after laser-assisted in situ keratomileusis, seen best just above the lower pupillary margin.

![Figure 3](image3.png)

**Figure 3.** Section of the corneal button showing the edge of the lamellar cut (lower right corner) and a paracentral thin area (periodic acid–Schiff, original magnification ×3).

![Figure 4](image4.png)

**Figure 4.** Discontinuity of the Bowman layer and a slanted area of mild scarring (arrow) at one margin of the corneal flap (periodic acid–Schiff, original magnification ×100).

![Figure 5](image5.png)

**Figure 5.** Central area with a faint delineation (arrow) between the flap and the remainder of the stroma (periodic acid–Schiff, original magnification ×160).
but only one report included pathologic findings. The histologic features of our case echo previous light microscopic features. However, to our knowledge, transmission electron microscopic features have not been previously described. Even though the histopathologic changes are consistent with the clinical findings, it is still the physical properties, strength, and stability of the thin cornea after LASIK that need to be studied to understand why some patients have a guarded outcome. Seitz et al studied the posterior corneal curvature changes after LASIK, and concluded that increased negative keratometric diopters and oblate asphericity of the posterior corneal curvature pointed toward early keratectasia after LASIK. Until a better understanding of the biomechanical properties of the corneal deformation is reached, certain general guidelines should be followed. These include performing central corneal pachymetry and corneal topographic imaging in the preoperative evaluation of every potential refractive surgery patient and avoiding LASIK in patients whose final corneal stromal bed thickness is less than 250 µm or who appear to have keratoconus. Finally, corneal deformation and ectasia with a possible need for more corneal surgery, including lamellar or penetrating keratoplasty, should always be included as part of the informed consent.

The pathologic features of corneal ectasia include an interruption of the Bowman membrane with mild anterior cellularity, a thinner stromal thickness, and no endothelial damage.

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Presumed Choroidal and Orbital Mastocytosis

Mastocytosis is a condition characterized by the presence of excessive numbers of tissue mast cells in distinctive distributions, which produce symptoms related to mast cell mediator release and the disruption of normal tissue function. To the best of our knowledge, mastocytosis with ocular and orbital involvement has not been previously documented. We describe a 42-year-old white man with biopsy-proven mastocytosis and multiple disseminated lesions who developed a reduction in visual acuity to counting fingers OS, an elevated choroidal mass in the macula, and bilateral orbital tumors.

Report of a Case. A 42-year-old non-Hispanic white man was diagnosed as having mastocytosis in 1996 after a bone marrow biopsy. He had a 1-year history of recurrent episodes of flushing, chest tightness, headache, and fever and on examination was noted to have lymphadenopathy and hepatosplenomegaly. No dermatologic lesions were present.

He subsequently developed an extramedullary mass lesion in thoracic vertebrae 2 through 8, which
was compressing the spinal cord and was treated with excision and radiation therapy. A pathologic examination confirmed the lesion to consist of confluent mast cells. Six months later the patient developed urinary retention, and magnetic resonance imaging demonstrated a new mass lesion at sacral vertebrae 1 and 2. The second lesion was non-resectable and responded minimally to radiation therapy.

In May 1999, the patient went to an outside ophthalmologist complaining of a 4-week history of vision loss in the left eye. According to the patient’s medical records, the examination results were remarkable for visual acuities of 20/20 OD, 20/40 OS, and a small serous retinal detachment involving the macula of the left eye. His visual acuity decreased further from 20/60 OS in August 1999 to 20/400 OS in January 2000, and the serous detachment had enlarged with extension beyond the vascular arcades.

The patient first came to the National Eye Institute (Bethesda, Md) in April 2000. At that time, his visual acuities were 20/16 OD and counting fingers OS, with intraocular pressure readings of 15 mm Hg OD and 13 mm Hg OS. There was no proptosis according to Hertel exophthalmometry. Extraocular movements were normal, and slitlamp examination results were normal in both eyes. A dilated retinal examination showed an approximately 200-µm pigment defect superior to the right fovea, an elevated macular lesion with pigmentary changes, and an inferior exudative retinal detachment in the left eye (Figure 1). The visual field was normal in the right eye but showed a dense central scotoma in the left (Humphrey Visual Field Analyzer; Zeiss Humphrey Systems, Dublin, Calif). An ultrasonographic B-scan showed an elevated subretinal mass in the macular area (Figure 2) and a shallow serous retinal detachment inferior to this lesion. The mass was well outlined, was located in the choroid or subretinal space, and had a diameter of 9.6 mm and an elevation of 3.5 mm. A standardized ultrasonographic A-scan indicated that the lesion had moderately low acoustic reflectivity, which suggested that the mass had a homogeneous internal structure and was not calcified.

An orbital T1-weighted postcontrast magnetic resonance imaging scan with fat saturation showed a thickened, enhanced posterior pole in the left eye corresponding to the sonographic lesion. An extraconal mass was centered on the greater wing of the sphenoid bone and projected into the orbit, medially displacing the lateral rectus. It also projected into the middle cranial fossa as an epidural mass. The marrow of the sphenoid bone was replaced throughout (Figure 3). A computed tomographic scan of the chest, abdomen, and pelvis revealed multiple pleural-based densities, a 1-cm low-density lesion in the right lobe of the liver, splenomegaly, and retroperitoneal adenopathy. A skeletal survey using plain film showed diffuse mottling of bony structures including the ribs, pelvis, and spine. Results of an upper gastrointestinal endoscopy and colonoscopy were negative.

Laboratory studies were notable for mild pancytopenia, but no
circulating mast cells were seen. A trephine biopsy at the iliac crest revealed diffuse involvement of the marrow with sheets of fusiform mast cells (Figure 4). Results of immunohistochemical staining for the mast cell markers tryptase and surface CD117 (kit) were positive. Findings from immunohistochemical staining for other hematologic neoplasms were negative. Results of a sternal marrow aspirate were abnormal, with 16% intact mast cells. Immunohistochemical staining of the resected paraspinous lesions was similarly consistent with mast cell tumor. Plasma tryptase concentrations greater than 20 ng/mL are associated with mastocytosis; the level in this patient was 293 ng/mL, suggesting an extremely high mast cell burden. The erythrocyte sedimentation rate was 35 mm/h. Results of serologies for human immunodeficiency virus, syphilis, and cytomegalovirus were negative.

The patient was treated with histamine1 (H1) and histamine2 (H2) blockers, aspirin, and oral corticosteroids. He underwent a series of radiation treatments that resulted in resolution of the choroidal lesion and marked reduction in the bulk of the orbital lesions.

Comment. We describe a patient with a choroidal lesion, neurosensory retinal detachment, and orbital masses emanating from the sphenoid bone. The differential diagnoses for the choroidal lesion would include a metastatic lesion, choroidal osteoma, amelanotic melanoma, hemangioma, neurilemoma, leiomyma, lymphoma, and retinal pigment epithelium hypertrophy or hyperplasia. The history of systemic mastocytosis with biopsy-proven mastocytomas from multiple sites and disseminated lesions involving bone and the reticuloendothelial system provided strong evidence that the intraocular and orbital masses were mastocytomas. Although fine-needle aspiration of the choroidal lesion or open biopsy of the orbital masses might have been of additional value in confirming the diagnosis,1 the patient instead opted for radiation therapy in light of the well-defined disseminated process and the increased risk of complications associated with submacular biopsy. A thorough systemic evaluation, including a computed tomographic scan and gastrointestinal endoscopy showed no evidence of a concurrent primary carcinoma or lymphoma.

Mastocytosis is generally an indolent disease2 characterized by the
presence of an excessive number of tissue mast cells. The current classification system divides the disease into 4 categories: category 1A, indolent cutaneous mastocytosis, is the most common type. Category 1B, indolent systemic mastocytosis, is defined by mast cell infiltration of at least 1 organ system other than the skin. Category 2 refers to mastocytosis associated with a hematologic disorder, namely, myelodysplastic and myeloproliferative disorders. Aggressive mastocytosis, category 3, is associated with lymphadenopathy, hepatosplenomegaly, and marked eosinophilia. Finally, category 4, mast cell leukemia, is marked by atypical mast cells in the marrow and peripheral blood. The course of disease in our patient may be best described as aggressive mastocytosis.

The pathogenesis of mastocytosis is believed to be related to kit, the mast cell receptor for stem cell factor. Dysregulated kit or an excess of its ligand could conceivably lead to abnormal mast cell proliferation and the episodic release of preformed mediators, accounting for the signs and symptoms of mastocytosis. Demonstration that most adult-onset mastocytosis is associated with activating c-kit mutations is consistent with this hypothesis. However, an initial screen of the resected mastocytoma tissue in this patient failed to demonstrate the presence of an activating c-kit mutation.

Therapy for mastocytosis is focused on blocking the effects of mast cell mediators. H1 and H2 antagonists and cromolyn sodium are effective. Topical and systemic corticosteroids may be efficacious in selected advanced cases. Aspirin may be used to ameliorate the adverse effects of prostaglandin release, although extreme caution must be exercised given the risk of precipitating secretory granule release. Triggers such as insect bites and alcohol should be avoided; portable injectable epinephrine is a prudent safeguard to treat anaphylaxis. Currently there are no curative therapies for disseminated mastocytosis. Aggressive mastocytomas may be treated with excision and/or radiation. Therapies under investigation at the National Institutes of Health (Bethesda, Md) include agents that target the kit pathway as well as bone marrow transplantation.

The ophthalmic literature includes only 1 report of a mastocytoma, in which a 1-cm firm yellow nodule on the lower right lid that had been present since birth was excised from a 4-month-old boy. A histopathologic diagnosis revealed this nodule to be a mastocytoma, and there was no reported recurrence within 8 months of follow-up.

Our case suggests that mastocytosis may involve the choroid and orbit. The patient had a particularly aggressive form of mastocytosis that diffusely involved both the bone marrow and reticuloendothelial system and formed solid extramural collections of mast cells. This may explain the involvement of the choroid, which normally contains resident mast cells and has characteristics similar to lymphoid tissue.

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The diagnosis of Bietti crystalline retinopathy was made. A 22-month-old boy (the son of the proband’s daughter) was seen because of esotropia in the left eye. The child was the result of an uncomplicated pregnancy and a full-term spontaneous vaginal delivery. Both parents were Chinese and had normal eye examination results. His only sibling was a reportedly normal half-brother (paternal).

Examination revealed a large retinoschisis cavity in the right inferior temporal quadrant, extending to 1 disc diameter from the fovea. The fovea displayed cystic schisis. In the left eye a large schisis cavity involved the entire inferior temporal quadrant, including the macula. The patient was reexamined at age 5 years. Visual acuity was 20/125 OD and 1/150 OS. The electroretinogram was markedly attenuated, with an electronegative B wave in the high-intensity white scotopic response. Examination revealed partial collapse of the large schisis cavity in the right eye and enlargement of inner layer holes in the left eye (Figure 2).

The RS1 gene was sequenced from the child, both parents, and both maternal grandparents. The grandfather and grandson were hemizygous for a T to C transition mutation at nucleotide 667 in exon 6 of the RS1 gene. The daughter was heterozygous for the same mutation, which substitutes arginine for cysteine at residue 223 (C223R) in the predicted protein.

Comment. The gene responsible for X-linked retinoschisis was recently identified and designated RS1.1 The gene consists of 6 exons and encodes a 224-amino acid protein. Exons 4 through 6 encode a highly conserved discoid domain believed to participate in cell-to-cell adhesion. The Retinoschisis Consortium recently reported 82 different RS1 mutations among 91% of 234 cases clinically diagnosed as having retinoschisis.2 An additional 12 novel mutations have recently been described, including that occurring in the grandson in this family.3 The vast majority of mutations occur in exons 4 through 6 and those that eliminate or create a cysteine are common.

It is possible that this is a coincidental polymorphism rather than a disease-causing mutation. However, the evidence supports that this mutation resulted in retinoschisis in our patient. The child has the classic phenotype of XLRS, with no other abnormalities in the XLRS1 gene. The mutation occurs in an exon associated with numerous disease-causing mutations, and involves an amino acid substitution likely to alter the protein conformational structure in or near the crucial discoid domain. This mutation was not found in 100 unrelated phenotypically normal patients whose X chromosomes were sequenced.2

Bietti crystalline retinopathy is a rare hereditary retinal degeneration. The defining feature is the presence of crystals throughout the posterior retina. Vision loss accompanies progressive atrophy of the retinal pigment epithelium and choriocapillaris. The gene responsible for Bietti crystalline retinopathy has not been previously identified. Although generally believed to be autosomal recessive, the predominance of male cases has led some authorities to suggest an X-linked inheritance.4,5 An autosomal dominant pedigree with phenotypic resemblance to Bietti crystalline retinopathy has also been reported.6 It seems likely that

Figure 1. A, Color fundus photograph of the left eye of the proband. Intraretinal crystals are seen in the posterior pole. B, Fluorescein angiogram of the left eye of the proband demonstrates attenuation of the pigment epithelium and patchy atrophy of the choriocapillaris.
Bietti crystalline retinopathy is a phenotypic description reflecting more than one genotype.

The pedigree in this report has 2 male members who carry a novel mutation in the RS1 gene. The grandson has a phenotypic expression typical for XLRS and the phenotype exhibited by the grandfather is a crystalline retinal degeneration typical of Bietti crystalline retinopathy. It is not clear why the 2 affected male members of the family, who share the same RS1 mutation, have such different phenotypic expressions. Perhaps some other, as yet unidentified, heritable or nonheritable factor influences the expression of the gene.

The findings in our pedigree suggest that the C233R mutation has variable phenotypic expression, including crystalline retinal deposition and typical retinoschisis. This or other mutations in the RS1 gene may be responsible for some cases of Bietti crystalline retinopathy.

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Bilateral Angle-closure Glaucoma and Ciliary Body Swelling From Topiramate

Topiramate (Topomax; Ortho-McNeil Pharmaceutical Inc, Raritan, NJ), an oral sulfamate, is used for the treatment of partial-onset seizures. We report the case of a patient with topiramate-induced acute bilateral myopia and angle-closure glaucoma with an echographic description of the pathogenic mechanism.

Report of a Case. A 43-year-old woman had blurred distance vision in both eyes, accompanied by a mild frontal headache. Her symptoms began 1 day after starting topiramate, progressed for 24 hours, and remained stable for the last 4 days before visiting the ophthalmologist. Prior to the onset of symptoms, she stated that she had excellent uncorrected distance vision and only used corrective lenses to read. She stopped her medication after 3 doses, at the onset of symptoms. Initial examination determined a visual acuity of 20/20 OU with a 5-diopter (D) myopic correction, narrow angles, and an intraocular pressure (IOP) of 29 mm Hg OD, and 30 mm Hg OS. Her medical history and review of systems were unremarkable. She had no known previous drug allergies. Her only other medication was paroxetine (Paxil; SmithKline Beecham Pharmaceuticals, Philadelphia, Pa). She was treated with 0.5% timolol maleate in both eyes and referred for further evaluation.

The fifth day after discontinuing topiramate, visual acuity was 20/20 OU with a −4.50-D correction. The pupils responded normally. The peripheral anterior chamber (AC) depth was estimated to be less than 0.5 corneal thicknesses, and the central AC depth was 2 corneal thicknesses at the pupillary margin (Figure 1 A). Iris bombe was not present, and IOP was 12 mm Hg OD and 16 mm Hg OS. Gonioscopic examination of both eyes showed a 360º appositional closure (Figure 2 A) with opening on indentation. The optic nerve appeared normal, with a cup-disc ratio of 0.3 in both eyes.
Ascan echography determined a central AC depth of 1.4 mm in both eyes and a distance of 15.6 mm from the posterior aspect of the lens to the macula. High-frequency B-scan (25 MHz) revealed a narrow AC (Figure 2A), forward displacement of the lens, and swollen ciliary processes in both eyes (Figure 2C).

Topical timolol maleate, twice daily in both eyes was continued for 5 days. Twelve days after stopping topiramate, visual acuity was 20/15 OU without correction, IOP was 10 mm Hg OU, the AC depth was normal (Figure 1B), and the AC angles were open on gonioscopy (Figure 1D). A-scan measurements showed a central AC depth of 2.5 mm and a distance of 14.5 mm from the lens to the macula. Follow-up high-frequency B-scan demonstrated deepening of the anterior chamber (Figure 2B, normal-sized ciliary processes (Figure 2D), and a more posteriorly positioned lens in both eyes.

Comment. Acute myopia, a rare idiosyncratic reaction to sulfonamides, was first described in 1938.1 Two previous occurrences of acute myopia following use of topiramate have been reported.2,3 The authors of both articles speculated that the mechanism was related to partial inhibition of carbonic anhydrase. Although controversy exists regarding the exact mechanism of acute myopia and angle-closure glaucoma after sulfonamide use, most authors have attributed this to ciliary body swelling.4 The 5D myopic refractive shift, sequential ultrasound examinations, and slit-lamp photographs in this patient document the ciliary body swelling and the associated forward migration of the crystalline lens.

The pathophysiology of the ciliary body swelling is unknown. Krieg and Schipper5 questioned an acute hypersensitivity reaction based on the observation that rechallenging with the same medication failed to produce a second event. They speculate that drug-induced elevated prostaglandins contribute to the formation of edema within the ciliary body without evidence of a systemic allergic response. As the mechanism of angle closure does not involve pupillary block, peripheral iridectomy and topical miotics are not useful in the treatment of this type of secondary angle-closure glaucoma. Our patient improved after discontinuing the topiramate.

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Figure 1. Representative slitlamp photograph of the right eye demonstrating the following: A, a shallow anterior chamber; B, a deepened anterior chamber depth and gonioscopic view of the right eye; C, appositional closure of the anterior chamber angle; and D, open anterior chamber angle.
Late-Onset Bleb-Related Panophthalmitis With Orbital Abscess Caused by Pseudomonas stutzeri

Late-onset bleb-related endophthalmitis is a potentially disastrous complication of trabeculectomy that may occur months to years after surgery. The route of infection is believed to involve migration of bacteria across the conjunctiva, in contrast to early postoperative endophthalmitis, which results from intraocular inoculation of microorganisms at the time of surgery. Late-onset bleb-related infection occurs more frequently following full-thickness procedures, adjunctive antifibrosis chemotherapy, or when a late-onset bleb leak is present.

The disease spectrum ranges from infection limited to the bleb to frank endophthalmitis. We present a case of panophthalmitis and orbital abscess, which occurred as a late-onset complication of trabeculectomy with adjunctive 5-fluorouracil (5-FU).

Report of a Case. A 69-year-old man had bilateral posterior chamber pseudophakia and a failed trabeculectomy in his right eye, and a thin-walled, avascular conjunctival filtering bleb in his left eye. He developed pain, redness, and discharge in his left eye while vacationing in another country. Examination of the right eye was unremarkable and revealed a visual acuity of 20/50, a flat superior


Figure 2. Representative high-frequency B-mode ultrasound image of the right eye demonstrating: A, a shallow central anterior chamber (AC); and B, a deepened central AC. C, Swollen ciliary body process (arrow) and a narrow AC angle (asterisk). D, Normal-appearing ciliary body process (arrow) and a deep AC angle (asterisk). By comparing the corneal thickness, it is apparent that the images are taken at the same magnification.
bleb, and a deep and quiet anterior chamber. The left eye was noted to have severe proptosis and absence of light perception. Extensive lid ecchymosis and edema were present. Eye movements were severely restricted in all directions. The conjunctiva demonstrated extensive chemosis and a superior filtering bleb with pronounced purulence. The cornea was edematous and there was extensive fibrin and hypopyon in the anterior chamber. The posterior chamber and the posterior segment could not be visualized.

The patient was hospitalized and treated with fortified cefazolin sodium and tobramycin sulfate drops, as well as intravenous vancomycin hydrochloride and ceftazidime. Computed tomography (CT) scans of the orbits (Figure 1) revealed proptosis of the left eye, with significant periorbital inflammatory tissue as well as a 1-cm retrobulbar abscess. There was also diffuse infiltration of the retrobulbar fat, and linear enhancement along the optic nerve sheath. A diagnosis of infectious panophthalmitis with orbital abscess was made.

The left eye was enucleated on the basis of the CT scan findings and the clinical scenario. This was combined with drainage of the orbital abscess. Histopathological examination (Figure 2) revealed acute bacterial endophthalmitis with vitreous abscess, acute choroiditis, and end-stage glaucoma, as well as optic nerve, retinal, and choroidal atrophy with choroidal effusion. Cultures obtained at the time of enucleation revealed *Pseudomonas stutzeri*. Blood cultures obtained at the time of admission were negative for organisms. The patient was treated with intravenous vancomycin and ceftazidime for 4 days, and was discharged home receiving oral amoxicillin/clavulanate potassium, topical tobramycin/dexamethasone, topical neomycin sulfate/polymyxin B sulfate, and dexamethasone ointment.

**Comment.** Late-onset bleb-related endophthalmitis is a serious complication of filtering surgery. Previously reported risk factors for the development of this condition include inferior bleb location, blepharconjunctivitis, contact lens use, chronic bleb leak, nasolacrimal duct obstruction, young age, male sex, use of adjuvant antifibrosis agents, and the existence of a cystic, thin-walled bleb. Panophthalmitis occurs when the intraocular infection extends into and involves the sclera. Our patient developed late-onset bleb-related panophthalmitis with orbital abscess—a complication rare enough that we could not find any similar case reports in the literature. Presumably, the risk factors for development of late-onset panophthalmitis are similar to those for endophthalmitis, as the former represents an untreated extraocular extension of the latter.

*Pseudomonas stutzeri*, the causative organism in our case, is a nonfermentative, Gram-negative bacteria. The species is in manure, straw, pond water, sewage, and similar environments. Most patients in whom the organism has been isolated have been elderly and in poor health. Isolation of the organism from eyes is extremely rare. We found only one case report in the ophthalmic literature, a case of delayed-onset endophthalmitis after cataract surgery, that mentioned *P stutzeri* as the infecting organism. Our patient was elderly, but his general health status was not poor. He had recently been abroad (in urban areas only) and it is unclear whether this contributed to the development of his infection.

The severe course of this case reemphasizes the need for early detection and rapid, appropriate intervention based on the disease severity and offending organism. All patients with thin-walled filtering blebs should be urged to seek immediate attention should symptoms of late-onset bleb-related infection appear.

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Age-related macular degeneration (AMD) is the leading cause of vision loss in people older than 65 years. Most cases of severe vision loss are related to the development of exudative AMD, which is characterized by the growth of abnormal vessels from the choroidal circulation. These abnormal vessels leak fluid and blood into the macula. Eventually, a fibrotic macular scar can form.

Symptoms of exudative AMD include blurred vision, metamorphopsia, relative central scotoma, dyschromatopsia, photopsias, and formed hallucinations. Individuals often complain of difficulty reading and recognizing facial features. Symptoms related to unilateral exudative AMD also might include reduced binocular visual function and abnormal depth perception. Although individuals might not report reduced binocular visual perception, many admit to closing the “bad eye” to improve their ability to see fine detail.

Report of a Case. A series of paintings by a Pennsylvania artist illustrates the effect of unilateral exudative AMD on binocular visual function. The artist is a 68-year-old woman with a 3-year history of AMD characterized by drusen with mild retinal pigment epithelial alterations in the right eye and a fibrovascular macular scar in the left eye (approximately 6 disc areas). Her vision is 20/20 OD and 6/200 OS. Her symptoms included reduced vision with her left eye and the need to occlude her left eye to perform tasks requiring fine visual acuity. Her symptoms were noticeable from the time of her initial vision loss and did not change significantly during the following 2 years.

She performed a series of oil paintings using monocular and binocular vision. Figure 1 was painted with her left eye occluded. This painting is remarkable for vibrant colors and fine detail. The peaches are well defined using bright colors; the leaves and branches are distinct using appropriate shadowing. Figure 2 was painted with her right eye occluded. There is marked rearrangement of the image quality and loss of detail, with an inability to distinguish the peaches, leaves, or branches. The vibrant colors are replaced by a brown-green amorphous smudge.

Figure 3 demonstrates the adverse effect of unilateral exudative AMD on binocular visual function. When painting this figure, neither eye was occluded. There clearly is a detrimental effect of her “bad” left eye on her ability to perceive fine detail. She is unable to suppress the abnormal image from her left eye. As a result, the painting is notable for loss of detail. Two central objects are identifiable, although it is difficult to distinguish these objects as peaches. The details of the leaves, branches, and shadowing are lost.

Comment. Faubert and Overbury examined monocular and binocular spatial contrast sensitivity in older people with and without AMD. They measured contrast sensitivity thresholds using spatial sine wave gratings. Contrast thresholds were calculated using Michelson contrast $[1_{\text{max}} - 1_{\text{min}}]/[1_{\text{max}} + 1_{\text{min}}]$, where $1_{\text{max}}$ and $1_{\text{min}}$ corresponded to the maximum and minimum luminances in the image, respectively. In almost half of the population with AMD, sensitivity to spatial information, as measured using spatial contrast sensitivity, was worse when both eyes were used together compared with when the stimuli were viewed with only one eye. They use the term “binocular inhibition” to describe this phenomenon and suggest that for some patients with visual impairment related to AMD, one eye might be better than two.

The effect of AMD on the visual perception of an artist has been examined previously. Terrence Billings, MD, a physician and trained artist, demonstrated the impact of...
AMD in a series of paired oil paintings using first the vision of both eyes and then the vision of his more severely affected right eye alone. In contrast to the artist described herein, the distortion and relative central scotoma of Dr Billings’ more affected right eye had little impact on his ability to paint with both eyes together.

Exudative AMD can have a profound effect on monocular and binocular visual function. In some individuals with unilateral vision loss related to exudative AMD, a relative degree of visual impairment can occur when visualizing objects with both eyes simultaneously. The possibility of binocular inhibition should be considered when providing visual rehabilitative services for individuals with unilateral vision loss related to AMD.

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Announcement

Ophthalmic Pathology Fellowship

The American Ophthalmological Society–Knapp Fund and Research to Prevent Blindness is offering a new 2-year postgraduate fellowship for training in ophthalmic pathology with an annual stipend of $52,500. The first year of the proposed fellowship program will be spent in the study of diagnostic pathology in the initiation of experimental eye pathology laboratory research. The second year of fellowship training will include experimental pathology research combined with exclusive time in diagnostic pathology or time in a relevant clinical subspecialty. Applicants must be graduates of a medical school accredited by the American Medical Association, citizens of the United States, and have plans for an academic career. Deadline for submission of applications: January 15, 2002, for fellowship starting in July 2002. Please direct all inquiries and requests for application materials to Froncie A. Gutman, MD, AOS–Knapp Fund, Cleveland Clinic Foundation, 9500 Euclid Ave, Desk I-32, Cleveland, OH 44195; (216) 445-8145.