Medical Treatment of Operative Corneal Perforation Caused by Laser In Situ Keratomileusis

Laser in situ keratomileusis (LASIK) is an effective procedure to treat a wide range of myopia. The advantages of LASIK over photorefractive keratectomy (PRK) are rapid visual recovery, lower risk of corneal haze, greater regression of myopia, and less postoperative pain. However, LASIK requires more skillful surgical technique and more instrumentation than PRK. Thus, inaccurate and inadequate procedures during LASIK have a higher potential of complications.

The complications of LASIK include severing of the corneal flap, epithelial ingrowth, flap wrinkling, corneal astigmatism, and corneal infection. To our knowledge, only one case report by Pallikaris and Siganos has been published of corneal perforation following LASIK; it was a survey of their early 43 patients. Unfortunately, the treatment and the clinical course of the corneal perforation was not reported. We describe the treatment and clinical course of a patient with a LASIK-induced corneal perforation that affected the final visual acuity. We believe that this report on the treatment and recovery of the corneal perforation will be valuable information for refractive surgeons.

Report of a Case. A 33-year-old man was referred to us with a complaint of decreased visual acuity in the left eye after bilateral simultaneous LASIK performed 3 days previously at a different facility. A corneal perforation was noticed in the left eye during LASIK laser ablation in this eye. The surgeon stated that, before surgery, the patient had myopia in both eyes (−9.5 diopters [D] −6.0 D × 65° OD and −9.0 D −5.5 D × 110° OS) and his best-corrected visual acuity was 10/20 OU. The corneal flap was intended to be 160 µm thick, otherwise, precise information on the intended ablation depth could not be obtained from the surgeon. However, the surgeon found that the remaining stromal bed was thin due to excessive thickness of the corneal flap.

Our initial examination showed best-corrected visual acuity of 20/60 OD and 20/500 OS. Slitlamp examination of the right eye revealed that the corneal flap was missing and that the stromal bed was thin; in the left eye, the corneal flap was very edematous and a space was visible between the corneal flap and the stromal bed (Figure 1, left). The anterior chamber was very shallow, and aqueous humor was observed to leak onto the ocular surface with blinking (Figure 1, right). A round, 0.25-mm diameter perforation site was observed in the center of the stromal bed by slitlamp examination.

We treated the corneal perforation by applying a therapeutic soft contact lens with topical antibiotics, oral carbonic anhydrase inhibitors, and eye patching. After 1 week of treatment (10th day postoperatively), the aqueous humor leakage had ceased, the anterior chamber depth was normal, and the corneal flap was closer to the stromal bed. However, the flap remained edematous. Topical medications were discontinued but the therapeutic contact lens and eye patching were continued for 3 more weeks. On the 32nd postoperative day, the edema and diffuse opacification of the corneal flap suddenly resolved with no space observed between the flap and the stromal bed although one had been present on the previous day. The patient’s best spectacle-corrected visual acuity was still de-

Figure 1. Left eye. Three days after bilateral laser in situ keratomileusis. Left, The corneal flap is edematous and a space is evident between the flap and the stromal bed (arrow). The anterior chamber is very shallow. Right, Following a blink, aqueous humor is visible on the ocular surface (arrows).
creased at 30/50 OS due to residual areas of corneal opacification and irregular astigmatism. In addition, neovascularization was noted between the corneal flap and the stromal bed and epithelial ingrowth was present focally at the temporal edge of the flap (Figure 2).

The surgeon did not mention whether the patient had keratoconus; however, preoperatively the patient had high myopia and astigmatism, with a best-corrected visual acuity of 10/20 OS. In addition, the eye appeared very thin. These data suggest that the patient may have had keratoconus. In cases of keratoconus, the cornea is thin and excessive thickness of the flap may be created by the microkeratome because of distortion of the cornea, resulting in a thin stromal bed. We presume that this may have led to perforation with photoablation in this case.

The most interesting observation was that abrupt clearing of the corneal edema and the recovery of transparency occurred approximately 1 month after the injury by LASIK. This phenomenon suggests that wound closure and functional recovery of the endothelium leads to a prompt normalization of the corneal thickness. The site of perforation in the LASIK surgery was 0.25 mm in diameter and, therefore, may require approximately 1 month for recovery of the endothelial function.

Despite recovery of the cornea, high corneal irregular astigmatism and residual opacification remained, and this patient will eventually require lamellar or penetrating keratoplasty for better vision. One may question whether surgery should have been immediately performed. However, suturing of the flap to promote wound closure carries a high risk of inducing additional high irregular astigmatism. In our patient who had no suturing, the corneal edema disappeared completely in 1 month. We can then choose the most appropriate treatment after residual astigmatism and opacification have resolved. In our patient, it was more effective to use a therapeutic contact lens than to suture the corneal flap, because the suturing method has a high possibility of inducing new irregular astigmatism.

This case suggests that eye surgeons should treat corneal perforations by conservative medical therapy, waiting at least 1 month before determining the best course of possible subsequent surgical therapy.

Comment. No standard treatment has been established for corneal perforation with leakage of aqueous humor onto the ocular surface after LASIK. Because this is the first case of corneal perforation due to LASIK presenting to our institution, medical treatment was intuitive. Nevertheless, it was effective; the perforation closed, the edema cleared, and visual acuity improved to 30/50 OS.

Infectious Ulcerative Keratitis After Laser In Situ Keratomileusis

With any laser refractive surgical procedure in which the epithelial barrier is broken, there is an inherent risk of infectious complication. As laser in situ keratomileusis (LASIK) becomes more widely available, cases of LASIK-associated infectious keratitis have begun to be

Figure 2. Left eye. Thirty-two days after laser in situ keratomileusis and following treatment as described in the “Report of a Case” section. The cornea is generally clear and anterior chamber depth is normal, but areas of corneal opacification and irregular corneal astigmatism remained. Neovascularization is evident between the corneal flap and the stromal bed (arrows) and epithelial ingrowth was localized at the temporal edge of the flap (arrowheads).
We report 6 eyes of 5 patients that developed ulcerative keratitis after LASIK.

Report of Cases. Case 1. A 33-year-old woman noted a foreign body sensation in the right eye 4 days after bilateral LASIK. She was found to have a corneal infiltrate. She was given subconjunctival injections of vancomycin hydrochloride, cefazolin sodium, and betamethasone sodium phosphate and started on therapy with ciprofloxacin hydrochloride eye drops every 20 minutes. After 3 weeks of minimal improvement with various combinations of topical ciprofloxacin and prednisolone acetate, the patient was referred to the Doheny Eye Institute, Los Angeles, Calif. On her initial visit to us, visual acuity was 20/200 OD and 20/20 OS. A 1.5 × 2-mm epithelial defect with surrounding infiltrate was present at the 7-o’clock position within the flap edge, extending to approximately 30% of the stromal thickness in the right eye. Both corneas showed moderate punctate staining. Corneal scrapings for culture and sensitivity were obtained. All eye drops were stopped, and the patient was prescribed topical fortified cefazolin sodium (50 mg/mL) and tobramycin sulfate (14 mg/mL), alternating every hour while awake. Within 2 days reepithelialization had begun, and medications were tapered to every 2 hours. One week later the patient had no epithelial defect, but there was still an organizing infiltrate. The next week the infiltrate was nearly resolved, and the eye drops were tapered off over the ensuing 3 weeks. After 3 weeks the infiltrate had resolved. The patient was left with a paraxial, 2-mm-round, anterior stromal scar within the flap edge (Figure 1). No organism was recovered. Final uncorrected visual acuity was 20/40 OD.

Case 2. One year after bilateral LASIK, a 31-year-old male physician complained of acute tearing and photophobia of the left eye, in which he had been using a soft contact lens. He was found to have an infiltrate at the flap edge. He was started on a regimen of topical ofloxacin every hour, diclofenac 4 times daily, and 1% atropine daily, and was referred to us the next day. On his initial visit, visual acuity was 20/20 OD and 20/40 OS. A curvilinear epithelial defect with surrounding stromal infiltrate was noted, extending from the 5- to 7-o’clock position at the LASIK flap edge. Cultures were obtained, and the patient was started on a regimen of topical fortified vancomycin hydrochloride (25 mg/mL), alternating hourly with ofloxacin. A cephalexin was not used because of a history of penicillin allergy. Two days later the epithelial defect had widened to just beyond the flap margin and the stromal infiltrate had begun to organize (Figure 2). Cultures revealed the infectious organism to be Staphylococcus aureus. One week later the epithelial defect had healed and the infiltrate was organizing. The drops were tapered to every 3 hours. Over the next week the patient showed continued improvement of the infiltrate, with consolidation into a small stromal scar at the inferior flap margin. Antibiotics were slowly tapered over the next week. Uncorrected visual acuity returned to 20/30 OS.

Case 3. A 46-year-old woman complained of a foreign body sensation in her right eye 6 days after bilateral LASIK. She was found to have a small infiltrate at the flap edge. The patient had been wearing a soft contact lens in her right eye and was using topical prednisolone acetate in both eyes twice daily. She was prescribed ciprofloxacin eye drops every 30 minutes and referred to us the next day. On her initial visit, best-corrected visual acuity was 20/80 OD and 20/50 OS, with an 80% epithelial defect of the flap and a small infiltrate at the 7-o’clock position on the flap edge in the right eye, as well as marked punctate keratopathy in the left eye. The time at onset of the epithelial defect is unclear. Cultures were obtained, and the patient was prescribed topical ciprofloxacin every hour in the right eye and lubrication with artificial tears in both eyes. Drops were tapered over the next 2 weeks. At 2 weeks the epithelial defect had resolved in the right eye, and punctate keratopathy had improved in both eyes. A pinpoint scar remained at the 7-o’clock position at the LASIK flap edge. Final uncorrected visual acuity was 20/40 OD. No organism was recovered.

Case 4. A 50-year-old man developed bilateral eye pain and photophobia 1 day after bilateral LASIK. The patient had also undergone radial keratotomy in the left eye 16 years previously. He had been seropositive for the human immunodeficiency virus (HIV) for 10 years but had no major sequelae of the viral infection. He was taking various anti-retroviral medications, including protease inhibitors; his CD4 cell count was 0.3 × 10^9/L, and his viral load was undetectable. Because bilateral bacterial keratitis was suspected, both flaps were lifted, cultures were obtained, and the stromal beds were irrigated. The patient was hospitalized and given topical fortified cefazolin sodium (25 mg/mL), alternating with gentamicin sulfate (14 mg/mL), every 30 minutes. Two days after hospitalization, cultures returned positive for ciprofloxacin-resistant S aureus. Gentamicin was discontinued at this time. Four days later the patient’s condition was improved and he was discharged with a regimen of hourly topical fortified cefazolin in both eyes. His con-
ition slowly improved over the next 6 weeks as the drops were tapered. At 6 weeks, the right corneal flap was lifted to remove epithelial ingrowth. The patient’s visual acuity stabilized to 20/40 OD and 20/200 OS. One month after discontinuation of all medications, he noted a sudden decrease in visual acuity and the onset of photophobia and was referred to us.

On his initial visit, visual acuity was 20/60 OD and 20/400 OS. Examination was remarkable for severe blepharitis. The right cornea had a hazy central anterior stromal scar, with corneal neovascularization extending across the flap from the 7- to 9-o’clock position. The left cornea had 8 radial keratotomy incisions with a dense, central, anterior stromal scar, extending to 30% depth, with neovascularization extending across the flap edge from the 8- to 9-o’clock position. The patient was treated for blepharitis with lid hygiene, topical erythromycin ointment, and oral tetracycline hydrochloride. Penetrating keratoplasty was eventually performed in the left eye. Final visual acuity was 20/40 OD and 20/100 OS.

Case 5. Two days after hyperopic LASIK in the right eye, a 54-year-old man developed pain and acutely decreased vision in his right eye. The patient had undergone radial keratotomy, followed by 2 enhancement procedures in the right eye, 9 years previously. Eight years later he elected to undergo myopic photorefractive keratectomy, which left him overcorrected. One year later, the patient underwent hyperopic LASIK. On a visit to the referring ophthalmologist 2 days later, the flap appeared nonadherent and infected. With the presumptive diagnosis of “flap infection,” the patient underwent flap revision and suturing. He also received various topical and subconjunctival antibiotics. After 2 weeks without improvement, the patient was referred to us.

On his initial visit to us, visual acuity was counting fingers OD and 20/40 OS. Diffuse corneal edema was noted in the right eye, with scattered focal infiltrates under the flap. The flap was thinned and poorly adherent in a wedge-shaped distribution at the 5- to 9-o’clock position. Three sutures at the 5-, 10-, and 11-o’clock positions held the flap in place (Figure 3). Cultures were obtained from the area under the peripheral flap thinning, and the patient was started on a regimen of topical fortified cefazolin sodium (50 mg/mL) and tobramycin sulfate (14 mg/mL), alternating every hour, along with ciprofloxacin ointment at bedtime. Cultures were positive the next day for *Streptococcus viridans*. Over the next several days, the thinned region of the flap became necrotic, but visual acuity improved to 20/400 OD. A topical antibiotic taper was then started. The inferotemporal peripheral region of the flap was noted to have been lost 10 days later. The decision was made to remove the remaining sutures, lift the flap, irrigate the stromal bed, and repair a radial keratotomy incision that had opened in the temporal bed. However, 2 days later, just prior to the planned surgery, the patient returned with markedly decreased symptoms and visual acuity improved to 20/150 OD. The amount of corneal edema had decreased, and the area of flap melt had reepithelialized. The patient refused further surgery. Final uncorrected visual acuity stabilized at 20/60 OD.

Results. All patients were treated with topical antibiotics for their presumed infectious ulcerative keratitis. Four of 5 eyes (3 patients) were culture positive for bacteria. In all cases, patients began to show improvement once a stable antibiotic regimen had been started and corticosteroids had been discontinued. Secondary interventions were only necessary in 3 eyes (2 patients): flap revision in 2 eyes and penetrating keratoplasty in 1 eye. All 5 patients reported foreign body sensation as their chief complaint, with 4 of 5 patients reporting severe photophobia as well.

All patients had residual stromal scarring after developing ulceration, but 4 of 6 eyes (4 patients) had uncorrected final visual acuities of 20/40 or better. The remaining 2 eyes had visual acuities of 20/100 or better but had undergone secondary interventions and had had radial keratotomy performed previously.

Comment. We are beginning to see more cases of LASIK-associated ulcerative keratitis in our referral practice as LASIK becomes more widely available. Ulcerative keratitis may present at any time after LASIK; in fact, in 1 of our 5 patients it was noted to occur as late as 1 year after surgery. A review of our patients suggests a few possible risk factors for the development of ulcerative keratitis. Two patients were using soft contact lenses postoperatively, 1 in association with corticosteroid use. Contact lens wear and steroid use have long been associated with an increased risk of corneal ulceration. In both of our cases, the patients were not satisfied with the visual outcomes of their LASIK procedures and were given corrective contact lenses to augment their surgical correction. Neither patient was willing to undergo enhancement procedures.

Two patients had previously undiagnosed dry eye. Corneal ulceration can be one of the sequelae of keratoconjunctivitis sicca. In the setting of LASIK, dry eyes may significantly prolong epithelial healing time and therefore place the patient at a greater risk for ulcerative keratitis.

Our last patient had severe blepharitis and was HIV positive. In this particular case, the patient experienced no adverse sequelae of HIV, had a low viral load, and a normal CD4 cell count. It is unclear whether HIV is a risk factor for ulceration after LASIK. However, HIV-positive patients often experience severe ocular surface disease. The increased bacterial load associated
with severe blepharitis, as well as the impaired ability to clear this load, may greatly increase the patient's risk for infection.3

A summary review of the literature reveals several reports of infectious complications associated with LASIK (Table). Aras et al4 reported a case of corneal interface abscess that occurred 6 days after LASIK and improved with topical antibiotics. Final best-corrected visual acuity was 20/20 with an inferotemporal, 1-mm-round, granular stromal opacity. Reviglio et al5 reported a case of Mycobacterium chelonae infection centrally on and under the flap 1 month after LASIK. This patient's condition did not improve with antibiotics, but had a good result with penetrating keratoplasty. Watanabe et al6 described a case of bilateral infectious keratitis, occurring 1 day after bilateral LASIK, from which Staphylococcus aureus was isolated as the causative agent. The patient's condition improved after a protracted course of topical and intravenous antibiotics, with a final best-corrected visual acuity of 20/40 OU. Scattered stromal opacities remained in both eyes. Mulhern et al9 described a case of corneal abscess

<table>
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<th>Subjective Complaints</th>
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<th>Secondary Interventions</th>
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<tr>
<td>Case 1</td>
<td>4 d</td>
<td>SPK</td>
<td>None</td>
<td>Cefazolin, tobramycin/3</td>
<td>20/40</td>
<td>FBS, photophobia</td>
<td>Central corneal scar and peripheral NV</td>
<td>Flap revision</td>
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<td>Case 2</td>
<td>1 y</td>
<td>SCL (worn for correction)</td>
<td>Staphylococcus aureus</td>
<td>Vancomycin, ofloxacin/3</td>
<td>20/30</td>
<td>FBS, tearing, photophobia</td>
<td>(2) second flap lift and removal of epithelial ingrowth</td>
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<tr>
<td>Case 3</td>
<td>6 d</td>
<td>SPK, SCL wear and topical corticosteroid use (SCL worn for correction)</td>
<td>None</td>
<td>Ciprofloxacin/2</td>
<td>20/40</td>
<td>FBS</td>
<td>Small stromal scar at inferior flap margin</td>
<td>None</td>
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<tr>
<td>Case 4</td>
<td>1 d</td>
<td>s/p RK, HIV+ (10 y), severe blepharitis</td>
<td>Ciprofloxacin-resistant S aureus</td>
<td>Cefazolin, gentamicin/7</td>
<td>20/40</td>
<td>FBS, pain, photophobia</td>
<td>Central corneal scar and peripheral NV</td>
<td>(1) Flap lift and irrigation, (2) second flap lift and removal of epithelial ingrowth, (3) penetrating keratoplasty</td>
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<tr>
<td>Case 5</td>
<td>2 d</td>
<td>s/p RK, s/p PRK</td>
<td>Streptococcus vividans</td>
<td>Cefazolin, ciprofloxacin, tobramycin/3</td>
<td>20/60</td>
<td>FBS, photophobia</td>
<td>Flap thinning and poor adhesion, followed by focal necrosis</td>
<td>Central scar</td>
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<tr>
<td>Perez-Santonja et al†</td>
<td>6 d</td>
<td>s/p LASIK</td>
<td>Nocardia asteroides</td>
<td>Tobramycin, vancomycin/6</td>
<td>20/45</td>
<td>Photophobia, blurring, ghost images</td>
<td>Central scar</td>
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</tr>
<tr>
<td>Nascimento et al‡</td>
<td>120 d</td>
<td>s/p RK</td>
<td>N aeroides</td>
<td>Sulfacetamide, prednisolone/6</td>
<td>20/60</td>
<td>Photophobia, blurring, ghost images</td>
<td>Paracentral scar</td>
<td>Cap exchange</td>
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<tr>
<td>Watanabe et al§</td>
<td>3 d</td>
<td>None specified</td>
<td>S aureus</td>
<td>Topical ofloxacin, IV imipenem and cilastatin/6</td>
<td>20/40</td>
<td>Photophobia, pain, discharge</td>
<td>Multiple scattered stromal scars</td>
<td>None</td>
</tr>
<tr>
<td>Aras et al^</td>
<td>6 d</td>
<td>None specified</td>
<td>None</td>
<td>Vancomycin, ciprofloxacin/3</td>
<td>20/20</td>
<td>Photophobia, redness</td>
<td>No scarring</td>
<td>Likelihood of diffuse lamellar keratitis</td>
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<tr>
<td>Reviglio et al§</td>
<td>5 d</td>
<td>None specified</td>
<td>Mycobacterium chelonae</td>
<td>Tobramycin, erythromycin/2</td>
<td>20/20</td>
<td>FBS, pain, photophobia</td>
<td>Central abcess</td>
<td>Penetrating keratoplasty</td>
</tr>
</tbody>
</table>

* LASIK indicates laser in situ keratomileusis; SPK, superficial punctate keratitis; SCL, soft contact lens; s/p, status post; RK, radial keratotomy; HIV+, human immunodeficiency virus; PRK, photorefractive keractomy; IV, intravenous; FBS, foreign body sensation; and NV, neovascularization.
† Cefazolin was given as cefazolin sodium; tobramycin, tobramycin sulfate; vancomycin, vancomycin hydrochloride; ciprofloxacin, ciprofloxacin hydrochloride; gentamicin, gentamicin sulfate; prednisolone, prednisolone acetate; and cilastatin, cilastatin sodium.
with hypopyon and intense vitreous cellular reaction (ie, endophthalmitis) 3 days after LASIK. The infecting organism was Streptococcus pneumoniae. The patient’s condition improved with intravenous and topical antibiotics. Final best-corrected visual acuity was 20/25 with a hazy stromal scar. Perez-Santana et al1 reported a case of infectious keratitis involving the central cornea, secondary to Nocardia asteroides infection, 6 days after LASIK retreatment. The patient was treated with antibiotics, and final visual acuity returned to 20/40 with a small, round central scar. Nascimento et al2 also reported N asteroides infection centrally after a cap exchange was performed because of severe flap edema. The patient’s condition improved with repeated cap exchange and topical antibiotics. Final visual acuity was 20/200 with a paracentral leukoma.

It should be emphasized that all of these described cases of infectious ulcerative keratitis differ from the entity of “diffuse lamellar keratitis” described by Smith and Maloney.3-8 They described a syndrome with infiltrates that were diffuse, multifocal, and confined to the flap interface, with no posterior or anterior extension, and with an intact overlying epithelium in each case. None of the infiltrates we observed were confined to the interface, and an epithelial defect was always present.

While not all of our cases had positive cultures for organisms, the clinical appearance and response to treatment were typical of infectious keratitis. Most also had antibiotic therapy prior to culture.

The increasing number of reported cases of infectious keratitis after LASIK, while still very small, provides additional support for a conservative approach when considering bilateral surgery and when discussing informed consent.

**Use of a Polyurethane Patch for Temporary Closure of a Sterile Corneal Perforation**

Treatments for corneal perforation after thinning processes include acute penetrating keratoplasty, lamellar keratoplasty, grafting of conjunctival flaps, suturing of a scleral lamella into the perforation, tarsorrhaphy, and sealing of the perforation site with tissue adhesives.4 However, the use of homologous tissue is often followed by immunological allograft rejection and may be technically demanding in a soft eye. Tissue adhesives are especially suitable for perforations smaller than 1.5 mm, but the application is not always easy.1 For larger perforations, a patching material may be used. In this study we demonstrate that Neuro-Patch (B Braun Melsungen AG, Melsungen, Germany), a microporous, purified polyurethane material with excellent biocompatibility properties, can be effective in achieving temporary closure of corneal perforations. Neuro-Patch is primarily used as a dural substitute in neurosurgery. According to the product information, the material allows rapid immigration of connective tissue cells and is biostable and biocompatible. The material has a thickness of 0.45 mm, bends easily, and has a variable pore size ranging from 1 to 30 µm. The polyurethane fibers have a thickness of approximately 5 to 10 µm.

**Report of Cases. Case 1.** An 84-year-old woman with rheumatoid arthritis developed a 2 × 3-mm central corneal perforation 2 weeks after phacoemulsification of the left eye. Since donor tissue was not available at that time for emergency grafting and the perforation was too large to apply tissue adhesive, it was decided to use Neuro-Patch for closure of the perforation.

After all necrotic corneal tissue had been removed, the edges of the piece of Neuro-Patch were matched to the shape of the wound bed. A 2 × 3-mm patch was cut and sutured into the defect with six 10-0 nylon sutures, and the cornea was covered with a bandage contact lens. Six months later, the patch was still perfectly positioned with neither aqueous leakage nor any signs of anterior chamber inflammation (Figure 1). Meanwhile the patient had been treated with prednisone, 30 mg daily, and methotrexate, 10 mg weekly, and a penetrating keratoplasty was performed. Findings from histologic examination of the corneal button revealed epithelialization of the patch and infiltration of fibrolamellike cells into the deeper layers of the patch (Figure 2). No inflammatory cells were seen in the stroma or patch. Findings from transmission electron microscopy

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revealed invagination of epithelial cells into the patch and the formation of a new basal membrane by the epithelial cells. In addition, there was collagen formation of the keratocytes around the fibrillar architecture of the patch. Despite the systemic immunosuppression and topical treatment with serum drops, the patient developed marginal thinning of the graft. A conjunctival flap ended in necrosis, and ultimately tarsorrhaphy was performed. Nineteen months postkeratoplasty, the graft was relatively clear with mild punctate epitheliopathy, and the patient's visual acuity was counting fingers OS.

Case 2. A 5-year-old boy with Smith-Lemli-Opitz syndrome, an autosomal recessive disorder with severe psychomotor retardation and microcephaly, was seen for a 2 × 1.5-mm corneal perforation in the right cornea owing to neurotrophic keratopathy caused by infrequent blinking. A 2 × 1.5-mm piece of Neuro-Patch was sutured into the perforation with six 10-0 nylon sutures (Figure 3). Prior to treatment, informed consent was obtained from the patient's parents. One month later, the sutures were removed, and the patch was tightly fastened to the corneal wound margins. Postoperatively, neither anterior chamber leakage nor signs of anterior chamber inflammation were noted. Nevertheless, after tapering the topical preservative-free 0.5% prednisolone, the patch was lost. Findings from an examination with the patient under general anesthesia revealed that the stromal thickness had increased to 30% of the original thickness (Figure 4). Twelve months after the placement of the patch, the eye was quiet, and the epithelial surface was well controlled with artificial tears and ointments.

Comment. The closure of corneal perforations with human tissue in noninfectious corneal thinning is hampered by rejection of the corneal graft and by the recurrence of corneal thinning in the donor. Factors include the timing of the perforation, which often necessitates immediate closure in a patient at risk of immune reactions with high levels of corneal polymorphonuclear cells, collagenases, and proteases that destroy corneal collagen and proteoglycans. Nobe et al recently reported that all grafts failed in 4 patients with rheumatoid arthritis and corneal perforation. Palay et al demonstrated a survival probability of only 32% 2 years after the first keratoplasty in patients with rheumatoid arthritis. In a study by Bernauer et al, penetrating keratoplasty in corneal thinning resulted in an 80% failure of the grafts 6 months postoperatively. Notable improvement in graft survival could
be achieved with immunosuppression, suggesting that a delay of penetrating keratoplasty until the inflammation has subsided would be beneficial.

The use of tissue adhesives has been advocated for perforations that are less than 1.5 to 2 mm and have a small amount of surrounding stromal ulceration. The application of the smallest amount of glue to create a smooth surface may be technically demanding, and multiple applications may be needed. In a large study of 80 patients with corneal perforation or impending perforation, N-butyl-cyanoacrylate remained in place for an average of 50 days, and 44% of patients healed without further treatment. With respect to impending or actual perforation associated with rheumatoid arthritis, use of tissue adhesive was successful in 6 of 12 cases.

We surrned Neuro-Patch into 2 corneal perforations and observed good adhesion of the patch to the host tissue.

Epithelialization of the patch occurred gradually without any signs of inflammation of the anterior eye chamber. Findings from histopathologic examination demonstrated the development of an epithelial cell layer above the patch, and findings from electron microscopy showed the beginning of the deposition of the basal membrane. Legeas et al reported a study of 6 patients treated with a temporary polytetrafluoroethylene graft 0.7 to 1.0 mm in thickness (Gore-Tex). Although this material was well tolerated, no epithelialization of the implant or cellular ingrowth into the porous polymer was seen on histologic examination. Portnoy et al described the successful use of lophilized donor tissue (Kerato-Patch; Allergan Medical Optics, Irvine, Calif) as aplanomellar button to manage central stromal ulceration. Recently, Neuro-Patch also has been used for the closure of a scleral defect after the result of an explosion with multiple shrapnel. We believe that Neuro-Patch offers an additional method for the temporary closure of corneal perforations. Its advantages include the ease in technical handling and the excellent biocompatibility as proven by these 2 cases. In addition, the use of Neuro-Patch allows the surgeon to schedule graft surgery until the time corneal disease is adequately controlled. Finally, in one case, graft surgery was avoided since stromal regeneration occurred underneath the patch.

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**Sterile Mucopurulent Conjunctivitis Associated With the Use of Dorzolamide Eyedrops**

Dorzolamide hydrochloride, a nonbacteriostatic sulfonylamine derivative, is the first topical carbonic anhydrase inhibitor clinically available. It reduces intraocular pressure by inhibiting aqueous humor production in the ocular ciliary processes. The most common adverse effects associated with dorzolamide use are bitter taste and local ocular complaints such as burning, blurred vision, itching, tearing, foreign body sensation, stinging, eyelid discomfort, and nonspecific conjunctival hyperemia. As dorzolamide represents a completely new class of topical ocular drugs, its spectrum of side effects is not yet as well defined as for older drugs. With the increased use of dorzolamide, it is important to be aware of possible unreported side effects that will invariably occur. We report the unusual finding of a severe sterile purulent conjunctivitis in 7 patients using dorzolamide drops that resolved immediately after discontinuation of treatment. We believe that this condition is most probably attributed to the use of this drug.

**Report of Cases.** Case 1. A 73-year-old male patient had been treated bilaterally with 0.5% betaxolol hydrochloride twice a day and 2% pilocarpine 3 times a day during several years for chronic angle-closure glaucoma after an acute attack of angle closure in the right eye. Because of bradycardia of unknown origin, treatment with betaxolol was discontinued and replaced by 2% dorzolamide hydrochloride (Trusopt) drops. Other topical medications included occasional fucidic acid gel for chronic blepharitis and, once to twice a year, additional 0.1% fluorometholone drops for episodes of marginal keratitis. One month after the change of therapy, the patient returned, complaining of red eyes with a bilateral purulent discharge that was first ascribed to recrudescence of blepharitis. There was a diffuse papillary reaction in the inferior palpebral conjunctiva and cul-de-sac that was less pronounced in the superior palpebral conjunctiva as well as hyperemia of the bulbar conjunctiva that was preponderant inferiorly. Treatment with fucidic acid gel was temporarily discontinued to perform conjunctival bacterial cultures, according to standard ophthalmologic microbiologic practice, which did not show any growth of organisms. A 10-day course of a fixed combination of 0.1% dexamethasone phosphate and 0.5% chloramphenicol drops was given in addition to the long-term fucidic acid gel.
The patient consulted us again and complained of the same, persisting symptoms. The purulent discharge was massive and the cul-de-sac was thickened with rugae (Figure). A second bacterial culture was again negative. Pilocarpine drops were discontinued and replaced by systemic acetazolamide sodium, 500 mg twice a day, because a cataract operation was planned. Several courses of antibiotic drops, including a neomycin sulfate– polymyxin B sulfate–gentamicin combination, the fluoroquinolone 0.3% lomefloxacin hydrochloride, and again 0.5% chloramphenicol, had absolutely no effect on the purulent conjunctivitis. The planned cataract operation had to be postponed 3 times. It was only after the discontinuation of the dorzolamide drops that rapid resolution of all symptoms within 2 days occurred, with progressive regression of signs. The cataract of the right eye was operated on uneventfully 1 week later. The patient remained without recurrence during a follow-up of 2 years.

Case 2. A 77-year-old pseudophakic patient was successfully treated for glaucoma with 0.5% timolol maleate (Timoptic) for more than 2 years. Six months before his referral, 0.5% timolol maleate drops had been replaced by dorzolamide drops because of “cardiovascular problems.” The patient had also been treated for blepharitis and meibomitis in the past. The reason for his referral was a bilateral refractory purulent conjunctivitis present for the last 4 months that had been treated with 4 different antibiotics, including fusidic acid, lomefloxacin, chloramphenicol, and a combination of neomycin and bacitracin. Bacterial cultures had been performed twice after a 48-hour antibiotic washout period. The initial swab revealed no bacterial growth and the second swab showed only saprophytic organisms (Staphylococcus epidermidis and Corynebacterium). On examination, a thickened inferior palpebral conjunctiva with a prominent papillary reaction was noted. Some papillae were very large, having the size of follicles, and there were rugae in the inferior cul-de-sac with prominent purulent secretions. Dorzolamide drops were discontinued and replaced by systemic acetazolamide. Chloramphenicol drops that had been prescribed by his ophthalmologist were continued. The purulent discharge and symptoms resolved almost completely within 36 hours with progressive regression of the other signs and no recurrence during an 11-month follow-up.

Case 3. An 81-year-old female patient was treated for 2 years with 2% carteolol hydrochloride twice a day in both eyes for ocular hypertension; dorzolamide was added in her left eye because of uncontrolled intraocular pressure. Three months after initiation of dorzolamide treatment, she complained of occasional redness and tearing in the left eye that was attributed to a moderate ectrion of the inner third of the inferior lid; a plasty of the lacrimal punctum was performed with resolution of symptoms. Several months later, she complained of stinging and purulent discharge in her left eye. On examination, there was an inferior bulbar and palpebral hyperemia and a thickened conjunctiva without a frank papillary reaction but the presence of purulent secretions. The cornea was clear and no dermatitis was noted. Conjunctival bacterial cultures were negative. Dorzolamide treatment was interrupted and all symptoms resolved within 24 hours and signs resolved within 3 days.

Other Cases. Four additional bilateral cases that received bilateral dorzolamide therapy, for which no bacterial examinations were performed, were diagnosed soon after we had been aware of the described side effect. The durations of dorzolamide treatment until occurrence of the adverse clinical signs were 12, 21, 21, and 22 months. The duration until dorzolamide discontinuation was 2 months in two patients, 3 months in one patient, and 4 months in another patient. All patients had been treated unsuccessfully by several courses of topical antibiotics and at least 1 course of corticosteroid eye preparation. Resolution of symptoms and disappearance of the mucopurulent discharge occurred in all 4 patients 24 to 72 hours after discontinuation of topical dorzolamide treatment, with progressive regression of signs.

Comment. Dorzolamide is a topically active carbonic anhydrase inhibitor used in a 2% ophthalmic solution for the treatment of elevated intraocular pressure in patients with ocular hypertension or glaucoma. It may be used as first-line therapy for patients who are unable to tolerate β-blockers. It can also be used as an add-on therapy when more than one drug is needed. Dorzolamide is generally well tolerated and has few adverse effects. Mild stinging and burning on instillation, resolving within minutes, and a bitter taste following instillation have been reported in up to 27% of patients. 1,2 Allergic reactions, mainly nonspecific conjunctival irritation and lid reactions, have
been described in 10% to 15% of patients. White granular deposits on the bottle tip have also been reported.3

We describe herein an unreported adverse effect due to dorzolamide treatment occurring in 7 patients. The well-defined clinical picture of a sterile purulent conjunctivitis developed gradually in all patients after several weeks or months of dorzolamide treatment and all patients were mistakenly treated for a bacterial conjunctivitis for 2 to 4 months. The following elements represent strong arguments for a relation between the clinical picture reported and dorzolamide use: (1) the prompt and spectacular improvement in all 7 patients after the discontinuation of dorzolamide, (2) the unilateral involvement in the patient who was treated unilaterally with dorzolamide, and (3) the repeatedly negative bacterial cultures in the 3 first patients. The presence of the preservative benzalkonium chloride in other drops that were well tolerated by all patients eliminates this substance as a possible factor for the reported side effect. The absence of itching and dermatitis as well as the gradual development of symptoms over months speaks more for a toxic type of conjunctival involvement rather than a predominantly allergic reaction. Although allergic and toxic conjunctivitis are theoretically different clinical entities,4 it is not always easy to sort out the contribution of toxic or immunologic mechanisms in clinical practice. Instead of discussing the mechanism involved, we stress the well-characterized findings that all patients had in common. Dorzolamide is used predominantly in an elderly glaucomatous population in which blepharitis is a common finding. The clinical picture described herein might well be ascribed to an infectious conjunctivitis related to blepharitis, and thus may lead to prolonged, unnecessary antibiotic use as was the case for all our patients. Because of the delayed onset of adverse signs, after prolonged dorzolamide use, the link to dorzolamide is not always obvious. It is therefore important that the clinician be aware of this adverse effect, which should be suspected in cases of recalcitrant and unexplained purulent conjunctivitis in patients taking dorzolamide drops.

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Figure 1. Fundus photograph of the left eye showing the large nematode in the nasal retina. Narrowing of the retinal vessels and diffuse degeneration of the retinal pigment epithelium are apparent.

The First South American Case of Diffuse Unilateral Subacute Neuroretinitis Caused by a Large Nematode

Diffuse unilateral subacute neuroretinitis (DUSN) is characterized by early visual complaints, vitritis, papillitis, and recurrence of evanescent gray-white outer retinal lesions and later by progressive visual loss, optic atrophy, retinal vessel narrowing, and diffuse retinal pigment epithelium (RPE) degeneration occurring unilaterally in healthy patients.1 Classically, a motile, white, glistening nematode may be found during any disease stage, and should be suspected even in patients with advanced optic atrophy and degenerative RPE changes. At least 2 unidentified nematodes are associated with the syndrome. In endemic areas of the southeastern United States and South America, the nematode is 400 to 700 µm; in the northern midwestern United States and Germany the nematode is 1500 to 2000 µm.2-6 We report the first South American case of DUSN caused by the larger nematode.

Report of a Case. A healthy 15-year-old boy with a history of amblyopia in the right eye had acute visual loss in his left eye in June 1995. Visual acuity was 20/40 OS and 20/200 OD. Biomicroscopy revealed a normal anterior segment and fundus in the right eye and trace cells in the anterior chamber and anterior vitreous of the left eye. Signs of mild vitritis and papillitis associated with diffuse RPE alterations were present in the left eye. Early-stage DUSN was diagnosed, and argon laser treatment was applied to the superior temporal retina where a small worm was presumed to be present. After 4 weeks of laser treatment, visual acuity was 20/20 OS and the ocular inflammation had lessened. The patient returned 2 years later with severe visual loss in the left eye. Visual acuity was counting fingers and an afferent pupillary defect was observed. Examination through a slightly cloudy vitreous showed a 1500- to 2000-µm, white, motile nematode in the nasal retina (Figure 1). The optic nerve was pale and narrowing of the retinal vessels associated with evidence of more RPE involvement was observed. Despite no history of exposure to raccoons, a similar second-stage larva of Baylisascaris procyonis was suggested as a probable cause of DUSN. The patient had no other systemic complaints, and the unidentified large worm was destroyed by strong photocoagulation (Figure 2). Visual acuity improved to 20/200 OS and had not changed at the final examination (August 1998) (Figure 3).

Comment. In Brazil, DUSN is increasingly considered an important cause of posterior uveitis in chil-
Figure 2. Fundus photograph of the left eye immediately after treatment of the worm with strong application of argon laser.

Figure 3. Recent fundus photograph of the left eye showing the fundus 1 year after laser treatment. More pronounced fundus degeneration and a pale optic nerve are apparent.

dren and young healthy adults. Most patients are unaware of the disease until ocular examination performed in school or an ocular examination later in life.1,2 The typical late signs of DUSN predominate in these cases, and a solitary 400- to 700-µm nematode is frequently present at this stage, even years after disease onset. According to Gass,1 laser treatment of the nematode at any disease stage may improve visual acuity and inflammatory ocular signs. However, in our experience this improvement may be temporary, even if laser is applied to retinal areas simulating the presence of a worm. It is possible that some laser response in the RPE may interfere temporarily with the activity of the subretinal worm.

In 1984, Kazacos et al3 showed that at least some DUSN cases are caused by B. procyonis (Nematoda, family Ascarididae larvae), which are common intestinal roundworms of lower carnivores, including raccoons and skunks. Those authors experimentally produced DUSN in primates that were fed B. procyonis eggs.5 Additionally, the size of the intraretinal larvae and previous patients’ contact with raccoons made the hypothesis that B. procyonis was the probable cause of the disease even stronger. In their opinion, DUSN is caused by 2 species of nematodes or 2 sizes of a single species, reflecting different ages of larvae.4 The latter seems to make this possibility likely.4,5 Dogs and rats should also be considered potential sources of infection. It is important to emphasize that other species of nematodes should be considered as potential candidates for the cause of our patient’s symptoms. As more local clinicians and veterinarians become aware of these larger ocular nematode infections, other important epidemiologic findings will be reported.

Bilateral Massive Retinal Hemorrhages in a 6-Month-Old Infant: A Diagnostic Dilemma

Retinal hemorrhages in infants sometimes pose a diagnostic dilemma for ophthalmologists.

Report of a Case. A 6-month-old infant was treated in the hospital for a rotavirus gastroenteritis. Two days after discharge from the hospital, he was readmitted, profoundly dehydrated and in hypovolemic shock. He had collapsed and was unresponsive. His Glasgow Coma Scale score was 4, and his pupils were fixed and dilated. He was intubated and ventilated. Serum and plasma levels were measured and revealed hypernatremic dehydration consistent with severe water loss via the gastrointestinal tract: sodium, 169 mmol/L; potassium, 7.3 mmol/L; chloride, 136 mmol/L; urea nitrogen, 25 mmol/L (70 mg/dL); and creatinine, 222 µmol/L (2.51 mg/dL). The infant was acidotic with a pH of 6.8; Po2, 10.6; Pco2, 5.2; standard bicarbonate, 6.8 mmol/L; and base excess, 23 mmol/L.

Observations from funduscopic examination revealed massive bilateral retinal hemorrhages radiating from the posterior pole of the eyeball (Figure 1). Findings from coagulation studies, complete blood cell and differential cell counts, and thrombophilic screens were normal. His profiles for amino acid, fatty acid, and organic acid were normal. His profiles for amino acid, fatty acid, and organic acid were normal.


noid hemorrhage (Figure 2). The infant's clinical condition deteriorated and he died.

Findings from histopathologic examination of the eyes revealed massive retinal hemorrhages with subhyaloid and subretinal hemorrhages in both eyes (Figure 3). The brain scan revealed venous and capillary congestion with subarachnoid hemorrhage, a subdural collection, and focal intracerebral hemorrhages. There was also diffuse microvessel thrombosis in many organs, including the lungs, kidneys, and myocardium, consistent with disseminated intravascular coagulation. Nonaccidental injury (NAI) was suspected because of the findings from clinical examination; in particular, massive retinal hemorrhages in association with intracerebral hemorrhage. However, there was no evidence of trauma. A skeletal survey revealed no abnormalities. The findings from clinical examination were consistent with severe hypernatremic dehydration causing diffuse intracerebral hemorrhage, subarachnoid hemorrhage, retinal hemorrhages, and ultimately brain death.

Comment. Retinal hemorrhages in infancy are believed to be a cardinal sign of NAI. They may occur in up to 89% of infants with NAI. They may result from direct head trauma or the acceleration and deceleration forces generated by the shaking of the head. Shaken baby syndrome is a unique form of child abuse in which the only consistent external physical signs are ocular manifestations. Differential diagnoses of retinal hemorrhages include thrombocytopenias, leukemias, and infections such as infective endocarditis.

Finberg2,3 reported 12 cases of subarachnoid or subdural hemorrhage from hypernatremic dehydration in infants, 2 of whom died and 10 who had severe residual neurological damage. Pathologic effects of hypernatremia were also reported following a nursery disaster in which an improper infant food mixture—containing an excess of sodium chlo-
ride was administered to infants. Findings from autopsy showed subarachnoid hemorrhage, intracerebral hemorrhage, cortical venous thrombosis, and venous infarctions. Similar cases in adults have shown widespread cerebral hemorrhage.4 Infants are more susceptible to hypernatremia because of their large surface area and poor renal concentrating ability. The typical radiological findings from computed tomographic scans of infants with hypernatremia include brain parenchymal abnormalities, multifocal areas of hemorrhage, and infarction.5 Retinal hemorrhages were not documented in these cases.

In conclusion, we report a case of massive bilateral retinal hemorrhages and intracranial hemorrhages attributable to profound hypernatremic dehydration in an infant. The findings from clinical examination are similar to those seen in NAI. It is important to highlight this to avoid potential mistaken diagnoses. Unexplained retinal hemorrhages in infancy mandate a full clinical workup. The NAI remains high on our list of differential diagnoses. Other pathologic conditions can mimic NAI and have an identical clinical presentation.

Retinal Periphlebitis in a Patient With Pineal Germinoma

Patients with pineal germinomas commonly show signs and symptoms related to increased intracranial pressure and direct compression of the upper brainstem or cerebellum.1 Ocular manifestations typically include papilledema and extraocular movement disturbances. We describe a patient with a pineal germinoma who had posterior segment inflammatory changes.

Report of a Case. A 14-year-old boy of East Indian descent sought care because of a 2-month history of floaters and difficulty with visual tasks. The patient complained of headaches, nausea, vomiting, and an intermittent auditory bruit in the right ear. Systemic examination disclosed an unsteady gait and tremor. Ophthalmologic examination revealed a visual acuity of 6/12 OU. Convergence-retraction nystagmus, pupillary light-near dissociation, and slight limitation of upward gaze were present bilaterally. Diplopia was noted on right gaze with evidence of slight underaction of the left superior oblique. Findings from slit-lamp examination were normal. Dilated fundus examination revealed bilateral 2+ vitreous cells and marked optic nerve head edema (Figure 1). Tortuosity of the retinal vessels with areas of focal sheathing and exudates in a “candle wax dripping” configuration were also present. Large clumps of vitreous cells were noted in the inferior vitreous base bilaterally.

Fluorescein angiography revealed numerous areas of segmental hyperfluorescence of the retinal venules along the temporal arcades and leakage from both discs bilaterally. Neither capillary nonperfusion nor retinal neovascularization was noted. Laboratory investigations indicated a minimally elevated erythrocyte sedimentation rate and a normal serum angiotensin-converting enzyme level. Findings from tuberculin skin test, rapid plasma reagin test, and chest x-ray film were normal. A preliminary diagnosis of sarcoidosis was suspected. The patient was referred to the neurology service for evaluation of possible central nervous system involvement.

Computed tomographic scans and magnetic resonance images revealed a partially calcified homogeneous 3-cm pineal mass and obstructive hydrocephalus. The neurology service felt the clinical and radiographic findings were consistent with a germinoma and recommended radiotherapy. Based on the clinical examination and the presence of the sarcoid-like appearance in the fundus, the ophthalmology service recommended biopsy to obtain a tissue diagnosis.

A ventriculostomy of the third ventricle with endoscopic biopsy was performed. Findings from histologic examination confirmed the diagnosis of a germinoma. A cerebrospinal fluid specimen was analyzed, and the results were negative for tumor markers and inflammatory cells. Fractionated focal radiotherapy (45...
Gy) was administered over a 5-week period. Follow-up serial fundus examinations revealed a marked decrease in optic disc edema and periphlebitis bilaterally. Complete resolution of retinal findings was noted by 3 weeks after termination of radiation therapy (Figure 2). Throughout the patient’s medical course, no local or systemic corticosteroid treatment was used.

Comment. Germinoma is the most common intracranial germ cell tumor and typically arises in the pineal gland or suprasellar region. It occurs frequently in boys aged 10 to 12 years and often is seen with ophthalmic manifestations. In our patient the most striking ophthalmic clinical finding was the presence of intraocular inflammation and retinal phlebitis. The fundus appearance was strikingly similar to that seen in patients with retinal involvement of sarcoidosis. It has been estimated that up to 35% of individuals with posterior segment involvement of sarcoidosis will have concomitant central nervous system disease. Despite the normal findings from chest x-ray film and normal levels for angiotensin-converting enzyme, our clinical suspicion for sarcoidosis remained strong enough to warrant our insistence that tissue biopsy be performed prior to radiation treatment.

Both the histologic confirmation and the prompt resolution of the retinal lesions following radiotherapy to a distal site supported our new hypothesis that the fundus findings were related to the tumor. Though unproven, this suggests the possibility of a remote influence of the pineal tumor on the retinal vasculature in a manner comparable to a paraneoplastic syndrome.

To our knowledge there are no reported cases of paraneoplastic syndromes associated with pineal germinoma. There are, however, numerous examples of paraneoplastic syndromes associated with seminomas of the testis and dysgerminomas of the ovary, both of which are histologically similar to intracranial germinomas. These latter tumors have been associated with hypercalcemia and demyelination disorders.

Paraneoplastic syndromes typically manifest with widespread outer retinal findings resulting in visual loss and have been associated with a number of different carcinomas. The pineal gland contains differentiated photoreceptor tissue and pos-
sibly may be a source of antigenic presentation in the context of germ-cell tumors. The pineal gland may also be involved in patients with sympathetic ophthalmia and trilateral retinoblastoma.\(^5\)

We report this case to bring attention to the possible association of retinal vasculitis with germ cell tumors. Knowledge of this association may prevent unnecessary biopsies in future cases.

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Massive Orbital Myiasis Infestation

Infestation by dipterous fly larvae in ocular and orbital tissues (ophthalmomyiasis) occurs throughout the world, mostly in children and older people and, depending on the genus of the fly, the infestation can be by single or multiple larvae.\(^3\) We report a case of orbital myiasis in a man who had more than 100 larvae removed, followed by orbital exenteration.

Report of a Case. A 54-year-old man had severe pain and swelling in his right orbital region. The sensation was noted 4 hours prior to examination, just after waking up from an estimated 24-hour sleep in a countryside house after ingesting a bottle of a sugarcane alcoholic beverage. The patient reported undergoing exeresis of a suspected skin cancer in his right inferior eyelid a few years previously that then recurred with an exudative inferior eyelid lesion. In addition, alcoholism of 30 years' duration was mentioned.

Findings from examination revealed periorbital edema, erosion of the skin and conjunctiva of the inner canthus of his right orbit, and a wound full of larvae that displaced the eye globe to the outer canthus. Findings from gross examination revealed a hypotonic eye, an inflamed conjunctiva, and a severely swollen cornea. Visual acuity OD was no light perception (Figure 1 and Figure 2).

The patient underwent orbital exenteration under general anestheisia (Figure 3) and more than 100 larvae of Cochliomyia hominivorax (Coquerelli) (Diptera: Calliphoridae) were removed. Findings from histological analysis of the specimen showed the presence of basal cell carcinoma in the eyelid with infiltration into the muscle layer; the margins were free of neoplasm. This patient's oculoplastic aspect has been followed up periodically to provide a satisfactory cosmetic result. In addition, a psychiatrist was enrolled to provide needed assistance.

Comment. Ophthalmomyiasis is considered a rare, life-threatening condition. Mechanical removal with
or without chemical immobilization of the larvae are the options of treatment, depending on the time and degree of infestation. The preservation of larvae in ethanol for genus identification may be useful for epidemiological purposes and the subsequent application of large-scale measures to reduce the fly population. Outbreaks of C. hominivorax have been reported, and although considered to be confined to the New World, reports have identified the parasite in Africa and Asia.2

The massive infestation seen here may have been precipitated by an open wound caused by basal cell carcinoma in the nasal canthus. This hypothesis is based on previous observations that adult flies of C. hominivorax locate their hosts via visual and olfactory stimuli and are strongly stimulated by fresh blood, usually present in this type of skin neoplasia.3 Alcoholism and the prolonged state of unconsciousness in a rural location contributed to the fast destruction of the orbit of our patient. Exenteration was conducted to prevent intracranial progression.

Although there is no method for completely protecting against myiasis, prevention may be conducted on a local scale by practicing adequate personal hygiene and proper care of wounds. To our knowledge, this is the first reported case of ophthalmomyiasis caused by C. hominivorax in which exenteration was necessary to contain the progression of the infestation.

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Ocular Injuries Caused by Airsoft Guns

In the past 2 years, we observed in our outpatient clinic an escalating number of ocular injuries caused by "airsoft" or "softgun" toy weapons. In contrast to the conspicuous hazard of classical air guns (BB guns), this new kind of toy weapon implies a dangerously misleading harmlessness, both because of the airsoft name and because the gun is made of plastic. These toy guns have gained popularity among youngsters since they are relatively cheap, can be purchased without age restrictions, and look real.

To our knowledge, no reports have previously been published on eye injuries caused by these guns. The purpose of this study is to illustrate their potential ocular hazards and to propose preventive measures.

Airsoft Guns. Airsoft guns consist of a plastic pistol that shoots hard plastic bullets with a diameter of 6.0 mm (Figure 1). These bullets are available in different weights (0.12, 0.2, and 0.25 g), all of which are much lighter than the lead bullets used in BB guns (0.52-1.6 g). The guns use the direct force of a spring coil or compressed air to fire bullets. In an independent ballistic investigation, the calculated energy for bullets weighing 0.2 and 0.12 g was 0.363 and 0.347 J, respectively. This energy results in velocities of 61.5 and 74.9 m/s, respectively, and in-flight ranges of 30 to 50 m. These bullets are made of a very hard and noncompressible plastic material and thus, do not absorb energy. They can cause injuries, even when they ricochet from rigid surfaces. In comparison, BB gun bullets reach speeds of 100 to 200 m/s and a flight range of 100 to 150 m.

Patients. Between October 1996 and July 1998, 9 male patients were treated, 8 at the eye clinic of the University of Bern and 1 at the eye clinic of the University of Basel, Switzerland, for ocular injuries caused by airsoft bullets. After reviewing the
medical charts, patients were scheduled for an additional eye examination. Mean follow-up was 8.8 months (range, 0.5-24 months). Mean ± SD age was 13.9 ± 2.3 years (range, 11-17 years). All patients were male and only 1 eye was affected. In 2 cases, bullets ricocheted from a wall into the eye; in the other 7 cases, the eye was hit directly.

Results. A summary of the results is given in the Table. Initial visual acuity was counting fingers or light perception in 3 patients, between 20/100 and 20/25 in 4, and 20/20 in 2. Final visual acuity was 20/20 in 8 patients and 20/25 in 1.

In 6 patients, corneal erosion and stromal edema matching the bullet size (Figure 2) were seen. Cataract development was observed in 3 patients; 2 showed a transient increase of intraocular pressure due to hemorrhagic glaucoma. Chamber angle recession was present in 2 patients, iridodialysis in another.

Injuries of the posterior segment were observed in 5 eyes. In 1 of these eyes, there was total hyphema with subsequent hematocornea and dense vitreous hemorrhage. A pars plana vitrectomy, combined with phacoemulsification and a posterior chamber lens implantation, was performed. Initial visual acuity was light perception; final visual acuity of 20/25 was attained 3 months after injury.

Another case of posterior segment injury showed a peripheral choroidal rupture but only a small vitreous hemorrhage. Initial and final visual acuity was 20/20. The area of choroidal rupture remained unchanged during the 14-month follow-up.

Comment. Previous to our series, ocular injuries due to air guns have been reported only in the context of BB guns, including penetrating globe injuries or retrobulbar optic nerve trauma leading to severe visual impairment or loss of the eye. Other severe nonocular injuries have been observed, some of which have led to death. This severity is due to the much higher energy of BB bullets (0.072 J/mm²)—which exceeds that necessary for scleral penetration (0.06 J/mm²)—than the considerably lower energy density (0.01 J/mm²) of airsoft bullets. In our series, airsoft bullet injuries ranged from light contusions to severe sight-threatening closed-globe injuries. These are less severe than with BB guns, possibly because airsoft bullets are larger and have lower energy. The worst injury in our series was a total hyphema combined with traumatic cataract, and a dense vitreous hemorrhage.

However, in contrast to BB guns, airsoft guns are easily available and their sale is not restricted by age. Airsoft guns were designed as a toy and are still marketed as a toy. The innocent name and misleading marketing contribute to the danger of airsoft guns. Safety goggles are not supplied with the guns, but are at least recommended in product manuals. In all cases, goggles could have prevented injuries.

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*† indicates increase in; IOP, intraocular pressure; CF, counting fingers; LP, light perception; x, finding present on initial clinical examination; conserv, conservative treatment; IOL, implantation of a posterior chamber intraocular lens; and ppV, pars plana vitrectomy.
Airsoft guns should not be considered to be toys but rather, as weapons with the potential of causing severe eye injuries. Globe ruptures or penetrating traumas were not observed in our series, but 1 injury was severe enough to require intraocular surgery. In the United States, some states recently passed legislations limiting the import and sale of these weapons.

All patients with eye injuries due to airsoft guns should be referred to an ophthalmologist for further evaluation. Safety goggles should be included with the guns and wearing them should be mandatory while playing. Age or sale restrictions should also be considered.

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