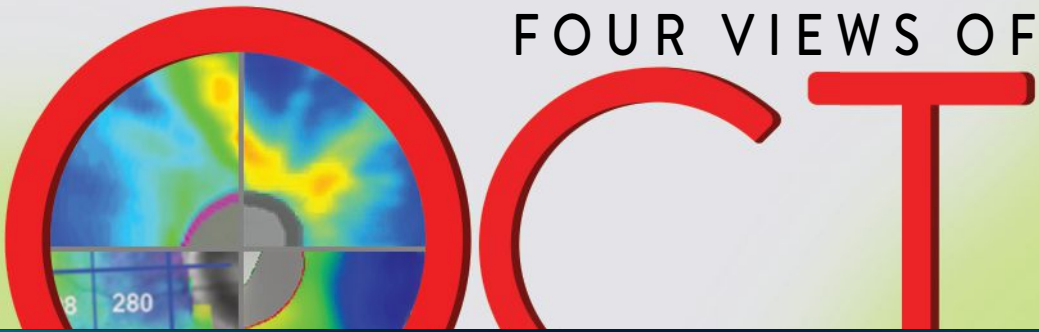
Sinking the Floaters: When and How to Perform YAG Vitreolysis, ***P. 80***

REVIEW® OF OPTOMETRY

February 15, 2020 **www.reviewofoptometry.com**



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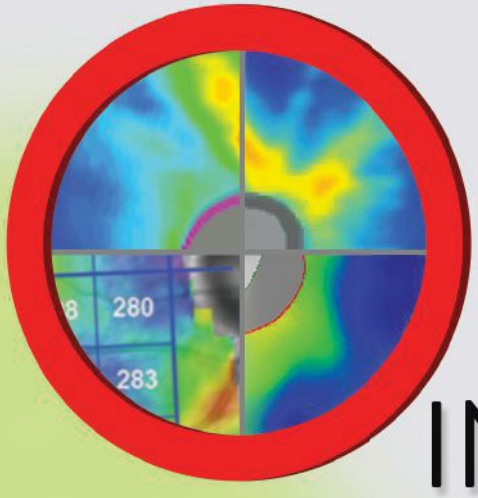

FOUR VIEWS OF OCT IN GLAUCOMA

Seeing the same patient tested on several devices provides a unique window into their similarities and differences, **P. 32**

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*Oxygen levels for single vision spherical (SVS) lenses only.
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VOL. 157 NO. 2 ■ FEBRUARY 15, 2020

IN THE NEWS

A blood clot in the dural venous sinuses, known as **cerebral venous sinus thrombosis (CVST), can result in papilledema that may not set in for weeks**, researchers warn. They initially found papilledema present in 54% of 65 patients. However, during the course of the disease, another 46% developed the complication. On average, **papilledema was first documented 29 days after CVST diagnosis**. In 21.5% of cases, it took about 55 days for papilledema to progress and six months to clear up.

Liu K, Bhatti M, Chen J, et al. Presentation and progression of papilledema in cerebral venous sinus thrombosis. Am J Ophthalmol. January 9, 2019. [Epub ahead of print].

In a study in *The Ocular Surface*, researchers found that **some dry eye patients with severe ocular pain were more inclined to report a history of fibromyalgia, depression, anxiety and migraine**. While treating the underlying symptoms can help reduce ocular discomfort, the study's participants still had low satisfaction rates, highlighting the need for more effective therapies.

Siedlecki AN, Smith SD, Siedlecki AR, et al. Ocular pain response to treatment in dry eye patients. Ocul Surf. January 10, 2020. [Epub ahead of print].

Researchers have discovered that **the prevalence of visual impairment and major eye diseases was approximately two- to seven-fold higher in participants with chronic kidney disease**. The disease's presence was closely related to visual impairment and retinopathy among diabetic participants. The study authors noted "the importance of ocular screening among these patients and potential common pathogenesis underlying these conditions."

Zhu Z, Liao H, Wang W, et al. Visual impairment and major eye diseases in chronic kidney disease: the National Health and Nutrition Examination Survey 2005 to 2008. Am J Ophthalmol. January 13, 2020. [Epub ahead of print].

Macular, GCC Changes Common for All

POAG patients still have faster thinning, which is associated with more severe disease.

By Catherine Manthorp, Associate Editor

Researchers recently observed ganglion cell complex (GCC) thinning and macula vessel density decrease over time, regardless of the presence of glaucoma. However, in primary open angle glaucoma (POAG) eyes specifically, they did find that the macula vessel density decreased faster than the GCC thinned and was associated with disease severity.

This prospective, longitudinal study evaluated 139 eyes (23 healthy, 36 pre-perimetric glaucoma and 80 POAG) of 94 patients who had at least three visits. The study followed the healthy eyes for an average of two years and the pre-perimetric glaucoma and POAG eyes for 2.6 years.

The team analyzed optical coherence tomography angiography (OCT-A) vessel density and OCT structural thickness of the same 3×3mm^2 GCC scan slab at each visit. They calculated and compared the rates of vessel density and thickness change within each diagnostic group. They also looked at the association between the rates of thickness and vessel density change and potential factors, including severity of disease and intraocular pressure (IOP).

The investigators discovered significant rates of GCC thinning and macula vessel density decrease in all diagnostic groups. In healthy eyes and pre-perimetric glaucoma eyes, they noted that the rates of GCC thinning and macula vessel density decrease were comparable.

In contrast, they detected a significantly faster rate of macula vessel density decrease in the POAG eyes than the rate of GCC thinning. In this group, they added that more than two-thirds of the eyes showed faster macula vessel density decrease than GCC thinning, with a significant association between faster macula vessel density decrease rate and worse glaucoma severity.

They concluded that the association between GCC thinning rate and glaucoma severity was not significant, while IOP observed during follow-up significantly affected the rate of GCC thinning in all groups but had no association with the rate of macula vessel density decrease.

"Macula vessel density is useful for evaluating glaucoma progression, particularly in more advanced disease," the study authors wrote in their paper.

Hou H, Moghimi S, Proudfoot JA, et al. Ganglion cell complex thickness and macula vessel density loss in primary open angle glaucoma. Ophthalmology. January 13, 2020. [Epub ahead of print].

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AI Now an ADA Standard

Technology touted as a viable alternative to traditional DR screening methods.

The American Diabetes Association (ADA) recently released its latest *Standards of Medical Care in Diabetes* publication, which normally wouldn't raise any eyebrows in the optometry world—except for this year. The ADA's 2020 standards finally make a formal leap of faith with artificial intelligence (AI), stating that "systems that detect more than mild diabetic retinopathy (DR) and diabetic macular edema (DME) authorized for use by the FDA represent an alternative to traditional screening approaches."[1]

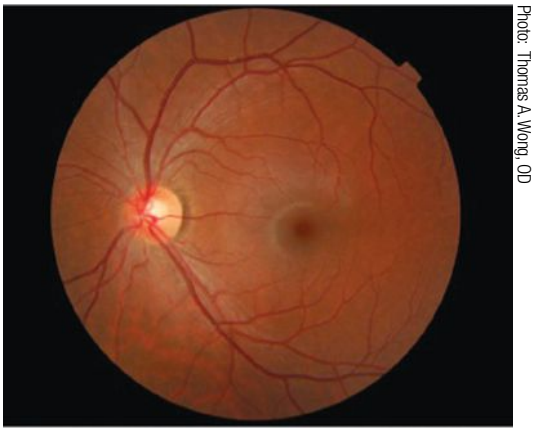

Photo: Thomas A. Wong, OD

Systems that detect more than mild DR represent an alternative to traditional screening approaches.

New Game in Town

The FDA cleared the first AI system for DR and DME screening in April 2018, the IDx-DR.[2] The device was cleared as a stand-alone tool that "provides a screening decision without the need for a clinician to also interpret the image or results," according to the FDA release. Such autonomy "makes it usable by health care providers who may not normally be involved in eye care."[1]

But the system isn't perfect, according to A. Paul Chous, OD, whose practice focuses on diabetes eye care and education. "It had a 13% false-negative rate for detecting 'more than mild DR,' does not evaluate the peripheral retina whatsoever, and has no sensitivity data reported for the detection of DME, the leading cause of vision loss due to diabetes and the detection of which multiple raster SD-OCT is the gold standard," he points out.

Some health clinics—including grocery store chain Albertsons' CarePortMD—have already embraced the technology, but with some caveats.[3] In these retail settings, the IDx-DR system requires a prescription (only those at risk are screened) and is performed by a registered nurse, according to a press release.[3]

Play at Your Own Risk

While the inclusion of AI in the standards is a significant change, the ADA included a word or two of caution when considering implementing the device: "the benefits and optimal utilization of this type of screening have yet to be fully determined. Artificial intelligence systems should not be used for patients with known retinopathy, prior retinopathy treatment, or symptoms of vision impairment."[2] The standards further emphasize that remote image reading, including use of AI, is not a substitute for a dilated eye examination by an optometrist or ophthalmologist, and that patients with previously diagnosed DR of any severity should see an eye care provider, explains Dr. Chous.

"This makes sense, given that the algorithm is not designed to assess change in individual patients' DR status, as well as the fact that patients with diabetes are more likely to experience a number of ocular conditions apart from DR, including cataract, ocular surface disease and glaucoma," he adds.

For most clinicians, implementation of the technology still raises a host of questions, including concern that patients with early signs of DR, such as peripheral lesions, given the green light by the AI system will forego a clinical exam. "What about the 30% of patients who have subclinical DME only detected by OCT and who are significantly more likely to develop significant DME over time?" Dr. Chous asks.

Nonetheless, Dr. Chous is convinced AI is here to stay, and it will improve. "It may very well help more patients be screened for DR and save vision, especially in underserved and some rural communities. It is not designed, however, to detect DR at its earliest stages when prevention and patient education are key. Nor will it replace knowledgeable providers when DR progresses and clinical acumen, experience and more sophisticated/comprehensive examination and technology is requisite."

1. US Food and Drug Administration. FDA permits marketing of artificial intelligence-based device to detect certain diabetes-related eye problems. www.fda.gov/NewsEvents/Newsroom/PressAnnouncements/ucm604357.htm. April 11, 2018. Accessed December 30, 2019.
2. American Diabetes Association. 11. Microvascular complications and foot care: standards of medical care in diabetes–2020. Diabetes Care. 2020;43(Supplement 1):S135-51.
3. IDx. Autonomous AI diagnostic launch in retail health clinics. Ciston PR Newswire. www.prnewswire.com/news-releases/autonomous-ai-diagnostics-launch-in-retail-health-clinics-300961103.html. November 19, 2019. Accessed December 30, 2019.

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Sleep Apnea Damages Meibomian Glands

Obstructive sleep apnea-hypopnea syndrome (OSAHS) is sometimes accompanied by posterior and anterior segment diseases, the latter of which can cause ocular surface damage. Knowing this, practitioners need to take a closer look at the meibomian glands, which protect the ocular surface and prevent ocular morbidity.

Researchers recently found that OSAHS yielded a loss in meibomian glands and a shortened first noninvasive tear break-up time (f-NTBUT), indicating that there could be a predisposition for evaporative dry eye originating from meibomian gland damage.

The study evaluated the right eye of 59 participants (32 OSAHS patients and 27 controls). The team measured f-NTBUT and average noninvasive TBUT and collected Schirmer scores, meibography and conjunctival impression cytology.

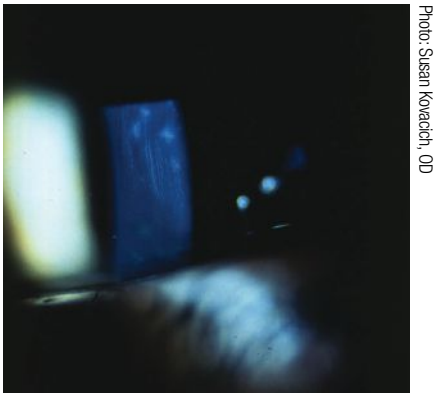

Photo: Susan Kovacich, OD

ODs may know sleep apnea can cause keratoconus, but a new study now also documents meibomian gland damage.

In the study and control groups, the investigators observed median f-NTBUTs of 2.1 seconds and 5.7 seconds and median average noninvasive TBUTs of 5.6 seconds and 7.2 seconds, respectively. They noted that the mean Schirmer values were 16.3±5.9mm and 17.3±6.6mm in the study and control groups, respectively.

In upper eyelid meibography, they discovered that the median losses in the meibomian glands were 20.10% in the study group and 14.70% in the control group; whereas, they were 19.00% in the study group and 12.40% in the control group in lower eyelid meibography. They added that the median Nelson grades in conjunctival impression cytology were one in both groups.

"Damage occurs in the meibomian glands of patients with OSAHS, and the meibomian gland loss causing this damage can be objectively determined with meibography," the study authors concluded in their paper. "Therefore, it can be considered that in patients presenting with meibomian gland damage of unknown cause, OSAHS should be kept in mind."

Muhafiz E, Ölçen M, Erten R, et al. Evaluation of meibomian glands in obstructive sleep apnea-hypopnea syndrome. Cornea. January 14, 2020. [Epub ahead of print].

Private Equity Acquisitions on the Rise

Firms are putting more money than ever into optometry offices.

Researchers from several universities have found a sharp increase in private equity-backed acquisitions of ophthalmology and optometry practices in the United States since 2012, according to their cross-sectional study of acquisition and investment data from January 2012 to October 2019.

Between 2017 and 2019, 186 practices in the United States were acquired. By comparison, only 42 were acquired between 2012 and 2016. The researchers found 228 practices (with 1,466 clinical locations) were acquired by 29 private equity-backed firms. Of those acquired, 127 were comprehensive/multispecialty, 92 were optometry practices and nine were retina clinics.

The companies acquiring these eye care facilities have developed both regionally focused and multistate models of acquisitions. Acquisitions were made across 40 states, but mostly in New York, which saw 22, and California, with 19.

The investigators arrived at their findings after compiling acquisition and financial investment data from six financial databases, four industry news outlets and publicly available press releases from private equity firms or platform companies.

The study also looked into subsequent sales, median holding period, geographic footprint and financing status of each platform company. In the future, the researchers intend to review the impact of private equity investments on patients, providers and practice metrics, including health outcomes, expenditures and staff employment.

Chen E, Cox J, Begaj T, et al. Private equity in ophthalmology and optometry: analysis of acquisitions from 2012 to 2019 in the United States. Ophthalmology. January 10, 2020. [Epub ahead of print].

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Reference: 1. Results from an in vitro laboratory study. TheraTears® SteriLid® Antimicrobial Eyelid Cleanser and Facial Wash showed efficacy in reduction of colony forming units for eight common eyelid organisms. Data was captured at 30 and 60 seconds.

M18-069-00

Glaucoma During Pregnancy a Challenge

Treating glaucoma during pregnancy may involve a balancing act between managing the mother's condition and weighing the potential risks to the developing child. Researchers recently suggested a multidisciplinary team that includes obstetrics and neonatology should counsel pregnant patients being treated for glaucoma and help monitor fetal health, especially since limited data exists regarding potential adverse events.

If medical management is necessary, a recent literature review published in *Current Opinion in Opthalmology* recommends clinicians take steps to reduce systemic absorption. They added that only two medications carry a historic FDA category B recommendation with inferred safety in humans from animal studies. They advise discontinuing medical therapy during the first eight weeks of gestation whenever possible, as this period is critical for major fetal organ development.

The team said clinicians should consider laser and surgical management, even before medical management in some cases, to prevent fetal exposure and maternal harm. They noted that traditional procedures, such as trabeculectomy and tube-shunts, are always options, as are newer minimally invasive glaucoma surgeries. When indicated, orphan trabeculectomy and collagen matrix implantation may be viable solutions for severe or progressive glaucoma during pregnancy, and the gelatin stent may also provide a minimally invasive option for pregnant patients.

"The management of the gravid patient with glaucoma entails consideration of both maternal disease severity and progression, and the potential adverse effects of therapeutic intervention on fetal growth and development," the authors wrote in their paper. "Although no systematic studies are likely to ever exist in this population, observational case-control studies and case reports help to place the array of therapeutic options into some context of safety for use during pregnancy."

Strelow B, Fleischman D. Glaucoma in pregnancy: an update. Curr Opin Ophthalmol. January 9, 2020. [Epub ahead of print].

GDD Suited for Iatrogenic Glaucoma

Glaucoma drainage devices (GDD) are safe and effective in the management of silicone oil–induced glaucoma and may pose less of a risk than diode laser cyclophotocoagulation (CPC). Although CPC has the advantage of shorter operative times and repeatability, this option carries a higher risk of failure, blindness and phthisis, the researchers noted.

Glaucoma surgery in silicone oil–filled eyes can be challenging, and no consensus exists on the preferred surgical option, the team from Saudi Arabia noted. They compared the effectiveness of GDD and CPC in 56 eyes of patients with refractory glaucoma following vitrectomy with silicone oil injection.

Seventeen eyes had GDD implants, and 39 eyes underwent CPC. Only six patients had pre-existing glaucoma before silicone oil injection.

The preoperative intraocular pressures (IOP) were 35.7±7.9mm Hg and 27.8±8.4mm Hg in the CPC and GDD groups, respectively, with a worse baseline visual acuity and higher proportion of silicone oil–filled eyes in the CPC group.

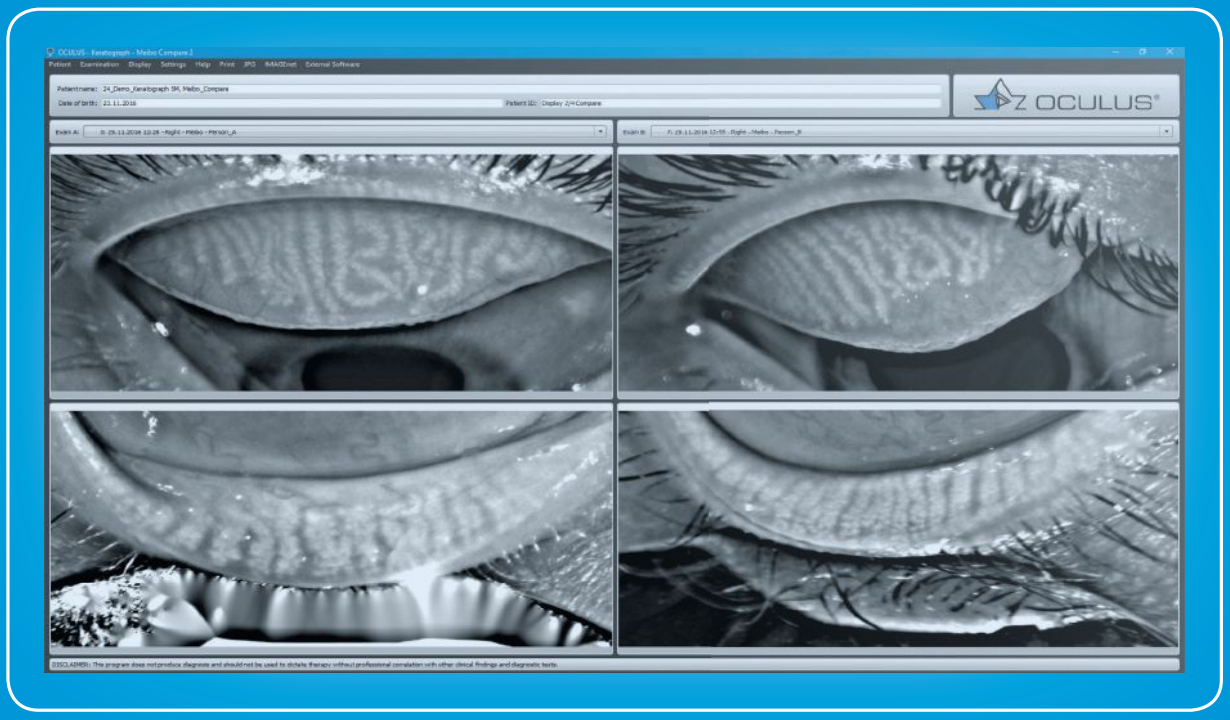
At one year, both groups achieved a reduction in IOP (23.5±11.5mm Hg in the CPC group compared with 15.3±5.9mm Hg in the GDD group) and number of medications taken. The GDD group had a much higher success rate of 94.1% (16/17) vs. 53.8% (21/39) in the CPC group. Additionally, five eyes in the CPC group lost light perception compared with one eye in the GDD group.

The re-operation rate and larger proportion of eyes going blind in the CPC group also contributed to the higher surgical failure: in the CPC group, five eyes required additional procedures. In the GDD group, none of the eyes required additional surgery. Six eyes in the GDD group had received prior CPC, whereas only one in the CPC group had received a prior GDD.

The investigators suggested CPC could be reserved for eyes with poorer visual potential and those with greater risk of retinal re-detachment after silicone oil removal. ■

Albahlal A, Alshamrani A, Khandekar R, Malik R. Outcome of surgical management of glaucoma following complex retinal detachment repair with silicone oil tamponade-drainage implant versus cyclophotocoagulation. J Glaucoma. January 6, 2020. [Epub ahead of print].

Let's Focus on Dry Eye!



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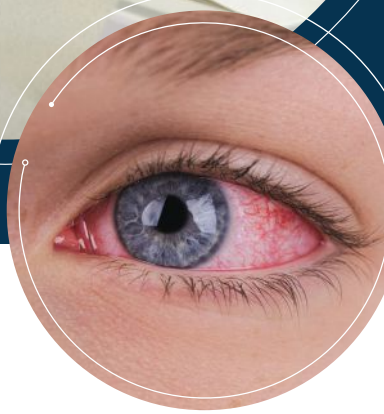

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Douglas K. Devries, OD

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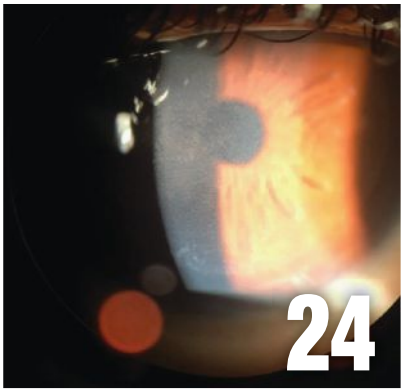

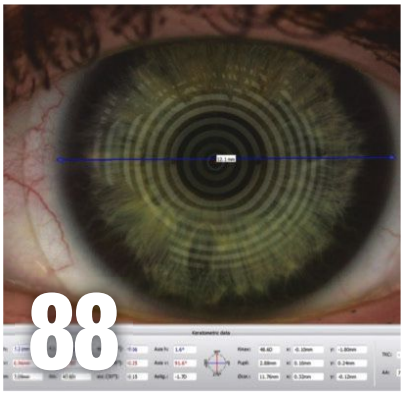

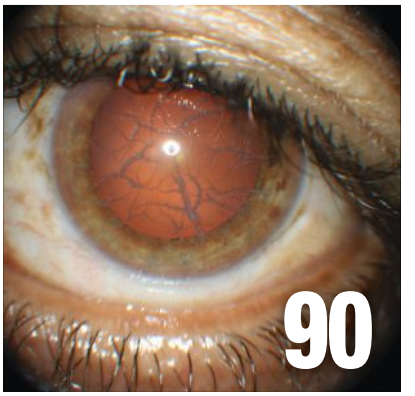

REVIEW OF OPTOMETRY

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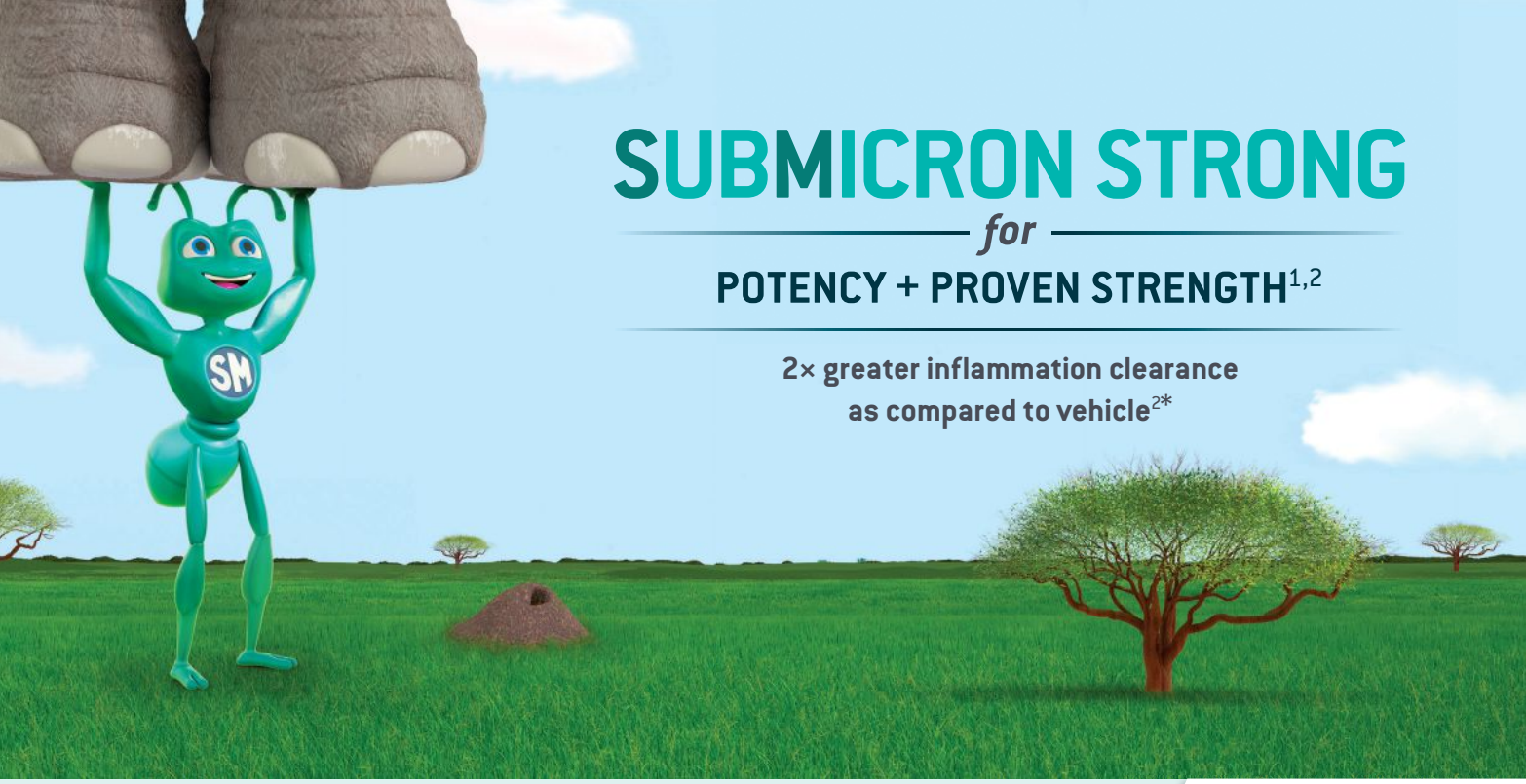

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†Pooled analysis of Phase 3 clinical studies. **Study 1:** 29% LOTEMAX® SM (N=171) vs 9% vehicle (N=172). **Study 2:** 31% LOTEMAX® SM (N=200) vs 20% vehicle (N=199); *P*<0.05 for all.

‡Pooled analysis of Phase 3 clinical studies. **Study 1:** 73% LOTEMAX® SM (N=171) vs 48% vehicle (N=172). **Study 2:** 76% LOTEMAX® SM (N=200) vs 50% vehicle (N=199); *P*<0.05 for all.

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- Use of corticosteroids may result in posterior subcapsular cataract formation.

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- The use of steroids after cataract surgery may delay healing and increase the incidence of bleb formation. In those with diseases causing thinning of the cornea or sclera, perforations have been known to occur with the use of topical steroids. The initial prescription and renewal of the medication order should be made by a physician only after examination of the patient with the aid of magnification such as slit lamp biomicroscopy and, where appropriate, fluorescein staining.
- Prolonged use of corticosteroids may suppress the host response and thus increase the hazard of secondary ocular infections. In acute purulent conditions, steroids may mask infection or enhance existing infections.
- Employment of a corticosteroid medication in the treatment of patients with a history of herpes simplex requires great caution. Use of ocular steroids may prolong the course and may exacerbate the severity of many viral infections of the eye (including herpes simplex).
- Fungal infections of the cornea are particularly prone to develop coincidentally with long-term local steroid application. Fungus invasion must be considered in any persistent corneal ulceration where a steroid has been used or is in use. Fungal cultures should be taken when appropriate.
- Contact lenses should not be worn when the eyes are inflamed.
- There were no treatment-emergent adverse drug reactions that occurred in more than 1% of subjects in the three times daily group compared to vehicle.

You are encouraged to report negative side effects of prescription drugs to the FDA. Visit **www.fda.gov/medwatch** or call **1-800-FDA-1088**.

Please see brief summary of Prescribing Information on adjacent page.

References: 1. LOTEMAX SM Prescribing Information. Bausch & Lomb Incorporated. **2.** Data on file. Bausch & Lomb Incorporated. **3.** Cavet ME, Glogowski S, Lowe ER, Phillips E. Rheological properties, dissolution kinetics, and ocular pharmacokinetics of loteprednol etabonate (submicron) ophthalmic gel 0.38%. *J Ocul Pharmacol Ther.* 2019. doi: 10.1089/jop.2019;35(5):291-300.

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LOTEMAX® SM
(loteprednol etabonate ophthalmic gel) 0.38%

BRIEF SUMMARY OF PRESCRIBING INFORMATION

This Brief Summary does not include all the information needed to use LOTEMAX® SM safely and effectively. See full prescribing information for LOTEMAX® SM.

LOTEMAX® SM (loteprednol etabonate ophthalmic gel) 0.38%

For topical ophthalmic use
Initial U.S. Approval: 1998

INDICATIONS AND USAGE

LOTEMAX® SM is a corticosteroid indicated for the treatment of post-operative inflammation and pain following ocular surgery.

DOSAGE AND ADMINISTRATION

Invert closed bottle and shake once to fill tip before instilling drops. Apply one drop of LOTEMAX® SM into the conjunctival sac of the affected eye three times daily beginning the day after surgery and continuing throughout the first 2 weeks of the post-operative period.

CONTRAINDICATIONS

LOTEMAX® SM, as with other ophthalmic corticosteroids, is contraindicated in most viral diseases of the cornea and conjunctiva including epithelial herpes simplex keratitis (dendritic keratitis), vaccinia, and varicella, in mycobacterial infection of the eye and fungal diseases of ocular structures.

WARNINGS AND PRECAUTIONS

Intraocular Pressure (IOP) Increase: Prolonged use of corticosteroids may result in glaucoma with damage to the optic nerve, defects in visual acuity and fields of vision. Steroids should be used with caution in the presence of glaucoma. If this product is used for 10 days or longer, intraocular pressure should be monitored.

Cataracts: Use of corticosteroids may result in posterior subcapsular cataract formation.

Delayed Healing: The use of steroids after cataract surgery may delay healing and increase the incidence of bleb formation. In those diseases causing thinning of the cornea or sclera, perforations have been known to occur with the use of topical steroids. The initial prescription and renewal of the medication order should be made by a physician only after examination of the patient with the aid of magnification such as slit lamp biomicroscopy and, where appropriate, fluorescein staining.

Bacterial Infections: Prolonged use of corticosteroids may suppress the host response and thus increase the hazard of secondary ocular infections. In acute purulent conditions of the eye, steroids may mask infection or enhance existing infection.

Viral infections: Employment of a corticosteroid medication in the treatment of patients with a history of herpes simplex requires great caution. Use of ocular steroids may prolong the course and may exacerbate the severity of many viral infections of the eye (including herpes simplex).

Fungal Infections: Fungal infections of the cornea are particularly prone to develop coincidentally with long-term local steroid application. Fungus invasion must be considered in any persistent corneal ulceration where a steroid has been used or is in use. Fungal cultures should be taken when appropriate.

Contact Lens Wear: Contact lenses should not be worn when the eyes are inflamed.

ADVERSE REACTIONS

Because clinical trials are conducted under widely varying conditions, adverse reaction rates observed in the clinical trials of a drug cannot be directly compared to rates in the clinical trials of another drug and may not reflect the rates observed in practice. Adverse reactions associated with ophthalmic steroids include elevated intraocular pressure, which may be associated with infrequent optic nerve damage, visual acuity and field defects, posterior subcapsular cataract formation, delayed wound healing and secondary ocular infection from pathogens including herpes simplex, and perforation of the globe where there is thinning of the cornea or sclera. There were no treatment-emergent adverse drug reactions that occurred in more than 1% of subjects in the three times daily group compared to vehicle.

USE IN SPECIAL POPULATIONS

Pregnancy: Risk Summary: There are no adequate and well controlled studies with loteprednol etabonate in pregnant women. Loteprednol etabonate produced teratogenicity at clinically relevant doses in the rabbit and rat when administered orally during pregnancy. Loteprednol etabonate produced malformations when administered orally to pregnant rabbits at doses 4.2 times the recommended human ophthalmic dose (RHOD) and to pregnant rats at doses 106 times the RHOD. In pregnant rats receiving oral doses of loteprednol etabonate during the period equivalent to the last trimester of pregnancy through lactation in humans, survival of offspring was reduced at doses 10.6 times the RHOD. Maternal toxicity was observed in rats at doses 1066 times the RHOD, and a maternal no observed adverse effect level (NOAEL) was established at 106 times the RHOD. The background risk of major birth defects and miscarriage for the indicated population is unknown. However, the background risk in the U.S. general population of major birth defects is 2 to 4%, and of miscarriage is 15 to 20%, of clinically recognized pregnancies. Data: *Animal Data.* Embryofetal studies were conducted in pregnant rabbits administered loteprednol etabonate by oral gavage on gestation days 6 to 18, to target the period of organogenesis. Loteprednol etabonate produced fetal malformations at 0.1 mg/kg (4.2 times the recommended human ophthalmic dose (RHOD) based on body surface area, assuming 100% absorption). Spina bifida (including meningocele) was observed at 0.1 mg/kg, and exencephaly and craniofacial malformations were observed at 0.4 mg/kg (17 times the RHOD). At 3 mg/kg (128 times the RHOD), loteprednol etabonate was associated with increased incidences of abnormal left common carotid artery, limb flexures, umbilical hernia, scoliosis, and delayed ossification. Abortion and embryofetal lethality (resorption) occurred at 6 mg/kg (256 times the RHOD). A NOAEL for developmental toxicity was not established in this study. The NOAEL for maternal toxicity in rabbits was 3 mg/kg/day. Embryofetal studies were conducted in pregnant rats administered loteprednol etabonate by oral gavage on gestation days 6 to 15, to target the period of organogenesis. Loteprednol etabonate produced fetal malformations, including absent innominate artery at 5 mg/kg (106 times the RHOD); and cleft palate, agnathia, cardiovascular defects, umbilical hernia, decreased fetal body weight and decreased skeletal ossification at 50 mg/kg (1066 times the RHOD). Embryofetal lethality (resorption) was observed at 100 mg/kg (2133 times the RHOD). The NOAEL for developmental toxicity in rats was 0.5 mg/kg (10.6 times the RHOD). Loteprednol etabonate was maternally toxic (reduced body weight gain) at 50 mg/kg/day. The NOAEL for maternal toxicity was 5 mg/kg. A peri-/postnatal study was conducted in rats administered loteprednol etabonate by oral gavage from gestation day 15 (start of fetal period) to postnatal day 21 (the end of lactation period). At 0.5 mg/kg (10.6 times the clinical dose), reduced survival was observed in live-born offspring. Doses ≥ 5 mg/kg (106 times the RHOD) caused umbilical hernia/incomplete gastrointestinal tract. Doses ≥ 50 mg/kg (1066 times the RHOD) produced maternal toxicity (reduced body weight gain, death), decreased number of live-born offspring, decreased birth weight, and delays in postnatal development. A developmental NOAEL was not established in this study. The NOAEL for maternal toxicity was 5 mg/kg.

Lactation: There are no data on the presence of loteprednol etabonate in human milk, the effects on the breastfed infant, or the effects on milk production. The developmental and health benefits of breastfeeding should be considered, along with the mother's clinical need for LOTEMAX® SM and any potential adverse effects on the breastfed infant from LOTEMAX® SM.

Pediatric Use: Safety and effectiveness of LOTEMAX® SM in pediatric patients have not been established.

Geriatric Use: No overall differences in safety and effectiveness have been observed between elderly and younger patients.

NONCLINICAL TOXICOLOGY

Carcinogenesis, Mutagenesis, Impairment of Fertility: Long-term animal studies have not been conducted to evaluate the carcinogenic potential of loteprednol etabonate. Loteprednol etabonate was not genotoxic *in vitro* in the Ames test, the mouse lymphoma tk assay, or in the chromosomal aberration test in human lymphocytes, or *in vivo* in the mouse micronucleus assay. Treatment of male and female rats with 25 mg/kg/day of loteprednol etabonate (533 times the RHOD based on body surface area, assuming 100% absorption) prior to and during mating caused preimplantation loss and decreased the number of live fetuses/live births. The NOAEL for fertility in rats was 5 mg/kg/day (106 times the RHOD).

Bausch + Lomb, a division of Valeant Pharmaceuticals North America LLC
Bridgewater, NJ 08807 USA

LSM.0091.USA.19
Based on 9669600-9669700 Revised: 02/2019

REVIEW OF OPTOMETRY

PRINTED IN USA

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1891-1913

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Outlook

By Jack Persico, Editor-in-Chief

Something or Nothing?

Tech may be aiming to take over disease screenings, but artificial intelligence is no match for the human kind.

Al Pacino's famous line from *The Godfather Part III*—"Just when I thought I was out, they pull me back in"—turned out to be more memorable than the movie itself. For optometrists, it seems the opposite statement is true. Every time optometry looks to be in the mainstream network of medical care, someone or something tries to pull you back out.

The American Diabetes Association just formally endorsed artificial intelligence (AI) use in disease screening efforts. As described in the news story on page 6 of this issue, the organization's latest standards document says AI systems that detect diabetic eye disease "represent an alternative to traditional screening approaches."

That'd be you, of course.

Optometrists are now pretty well accepted as the ideal entry point into the healthcare system for new eye disease cases, given the sheer number of practicing ODs and the preponderance of primary eye care performed by the profession. When someone's eye health starts to go awry, you're the nation's first responders. But even vital services are vulnerable to the threat of disruptive technology.

A good deal of your diagnostic responsibility is simply looking at an unusual presentation and asking, "Is this something? Or nothing?" It's the first big fork in the road for the patient, and you're the person who steers them one way or the other.

But if there's one thing computers excel at, it's binary decisions. Tech firms are busy feeding enormous datasets into ever more sophisticated pattern recognition algorithms to answer that something/nothing question. Without you.

The problem isn't so much the technology—AI-powered diagnostics have great potential to help—but rather the ways it might be used to do an end run around optometry. The worry is that automated screening tools set up in retail locations will just bypass optometry and steer disease suspects right to ophthalmology practices. If AMD or diabetic retinopathy patients have to end up in an ophthalmology practice to receive treatment anyway, the argument goes, why not just send them there in the first place?

Whenever that argument surfaces, it's important to remember optometry's competitive advantages over ophthalmology: affordability and access. Especially when the work is triaging brand new patients and educating them about their condition, an AI-to-OD 'referral' makes a lot more sense.

All this makes it incumbent upon you to keep sharpening your diagnostic skills, which is the theme of this issue. We present five feature articles on the art and science of making that something-or-nothing call with far greater sophistication than a chunk of silicon ever could.

Consider, for one, the article on medical history taking on page 44. It distills years of training and clinical experience by a VA optometrist into a cogent narrative simplifying the vast, interconnected world of systemic disease and the eye. And I think that's really something. ■

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Dry Eye: Tools of the Trade

Here's what you need to get your dry eye practice off the ground. Some of the equipment may surprise you. **By Paul M. Karpecki, OD, Chief Clinical Editor**

To effectively manage ocular surface disease in your office, you may need to invest in a few new tools—all of which can help you better assess, diagnose and manage these patients.

One incredibly useful piece of equipment is a slit lamp that captures images and video, such as the Haag Streit imaging system, TelScreen or Eyefficient. This pays for itself multiple times over because you can recall previous findings, educate patients about their condition (which increases efficiency and compliance) and bill for anterior segment photography.

Another helpful tool in patient education is Rendia (previously known as Eyemaginations). Patients want to understand their condition and its management, and the dry eye modules provide crucial information before, during and after the exam.

Diagnostics for All

Other necessary tools depend on how much you are dedicating to dry eye disease (DED). If you are managing mild to moderate forms and referring advanced DED or non-responders, you may only need a good history or dry eye questionnaire, sodium fluorescein dye and a device for meibomian gland expression. The dye is important for measuring tear break-up time, ocular surface and corneal staining and tear meniscus height. Meniscus height in particular can be a good determinant of aqueous deficiency. Expression will indicate meibomian gland dysfunction, pointing to an evaporative form.

A Dedicated Dry Eye Center

If you plan to dedicate your practice (or a segment of it) to DED, you will need other items. For symptoms assessment, consider SPEED, DEQ-5 or OSDI as potential options.

If you are receiving referrals you need diagnostics beyond those in general practice, including osmolarity testing. I could not run my dry eye clinic without it. I see patients who complain of symptoms of dry, gritty, irritated eyes who have normal osmolarity (between 280mOsmol/L to 300mOsmol/L and both eyes within 6mOsmol/L). Typically, these patients have been treated for dry eye for months or even years with no improvement in their symptoms—because the diagnosis is not DED.

Dry eye has a laundry list of possible differential diagnoses, including allergic conjunctivitis, epithelial basement membrane dystrophy or map-dot fingerprint dystrophy, recurrent corneal erosion, limbal stem cell deficiency, *Demodex*, trigeminal dysphoria, giant papillary conjunctivitis, neurotrophic keratitis, computer vision syndrome and Salzmann's nodular degeneration, to name a few.

Another valuable test is that of MMP-9 levels. Patients with high osmolarity and a positive MMP-9 test may need greater inflammation treatment and avoidance of punctal plugs.

Meibography is extremely valuable in determining the severity of evaporative DED or differentiating the type of DED. I've seen many cases where I could not express meibum, and meibography reveals either ample glands or few glands remaining. The treatment approach varies greatly between these two patient types.

Other vital dyes such as lissamine green are beneficial for assessing the conjunctiva for early DED or more advanced mucin deficient dry eye. And certainly noninvasive break-up time is more accurate than subjective dye testing tear film break-up time.[1]

Finally, you need to assess corneal sensitivity, even with something as simple as dental floss. Far too many patients with DED have neurotrophic keratitis components.

Final Steps

Ultimately, you need a good DED protocol, as dry eye is one of the most difficult conditions to manage. Clinicians initially saw DED as something of a nuisance condition similar to, say, chapped lips, but as doctors started working with these patients they quickly realized the complexity, variability, numerous masquerading conditions and comorbidities make it a unique condition to manage. You must understand this from the start; otherwise, you'll get frustrated. You don't want to miss out on incorporating one of the most prevalent and rewarding fields in all of medicine. Just ask a DED patient who has been well diagnosed and treated. ■

Note: Dr. Karpecki consults for companies with products and services relevant to this topic.

1. Versura P, Campos EC. TearLab Osmolarity System for diagnosing dry eye. Expert Rev Mol Diagn. 2013 Mar;13(2):119-29.

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Happy Optometrist Day

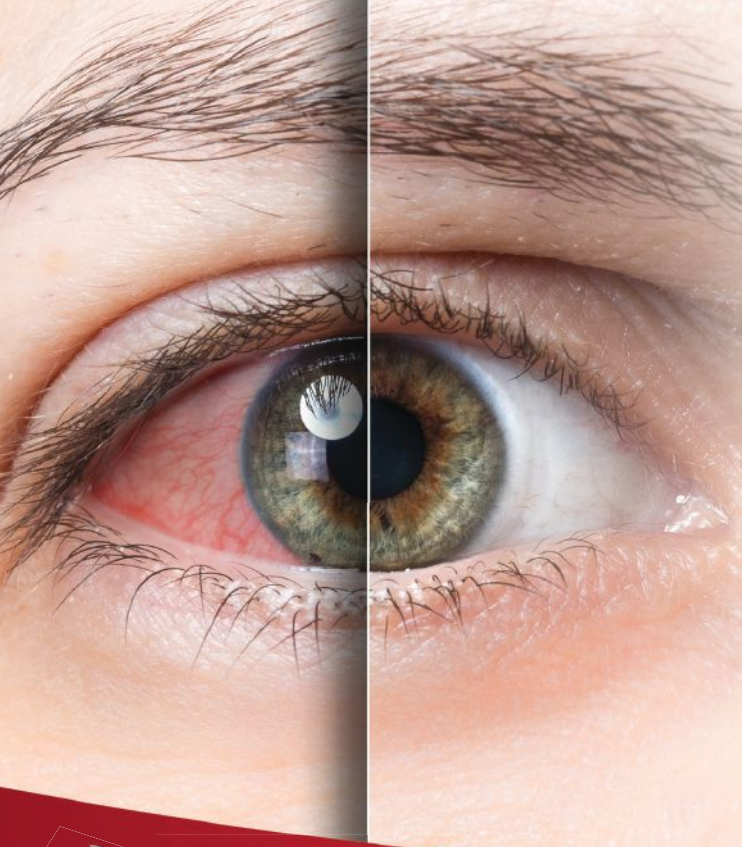
Every day is worth celebrating, but some are more special than others.

By Montgomery Vickers, OD

Happy birthday? Happy anniversary? Happy boss's Day? Happy New Year? Yes, these are all important milestones for us, and each one is a great opportunity to reload or, at least feel old. But sometimes, it does become a kind of slog because there are so many so-called "Happy _____ Day" celebrations. Since the list is already long, I decided, what's a few more? To give us something to celebrate at work, here's a few "Happy _____ Days" especially for optometrists: Happy…

- Correct seg height Day
- Teen boy can insert his own contact lens in one try Day
- The new phone system worked immediately Day
- The doctor remembered to zip his fly Day
- No staffer called in sick on a beautiful, sunny Friday Day
- The patient who saw sudden onset flashes came in before Friday at 4pm Day
- No, I won't call you in something for your red eye on Saturday Day
- No breakage at the lab Day
- The day's last patient showed up early Day
- The patient says "I still want it" when insurance doesn't cover it Day
- The post LASIK patient adores his new progressives Day
- I lost one of my GP contact lenses and I'm leaving for Italy this evening Day
- Two guys named Jeff came in at the same time and we totally mixed up their charts Day
- My sister gives me free contacts from the supermarket she works at Day
- I'm an airline pilot, and don't tell my boss but I can't see crap out of my left eye Day
- You just found out all of your employees meet at Happy Hour after work every day Day
- I would like to schedule all six members of my family on the same morning, but they may not all be able to be there Day
- You've remade my glasses three times, and I still can't see and I totally blame myself Day
- I sleep in my lenses because my cousin does and has never had a problem Day
- I think wearing glasses will make my kid's eyes worse Day
- Your office is a 10-minute drive, which not convenient, so I'm switching to a big box store that's 20 minutes away Day
- My legislators cannot pronounce "glaucoma" Day
- I want LASIK so I can wear sunglasses Day
- Doctor, do you think I should buy my contacts from you or on the internet Day
- I could never wear my glasses and I'm telling you that eight months later at the gym Day
- The IRS called, so I gave them your home phone number Day
- I accidently stuck a spoon in my eye so can you write me a prescription for fentanyl Day
- The state board can't find your CE hour submission Day
- Another forgotten password Day
- An OSHA agent is here, and he wants you to tell him the boiling point of G-15 dye Day
- A 90-year-old patient wants you to call her to discuss her $8.00 copay Day

Enjoy your special days, doctors. If you can survive just one day at a time, you'll make it all the way to "Happy I'm outta here Day." ■



Dry Eye Disease

Many symptoms

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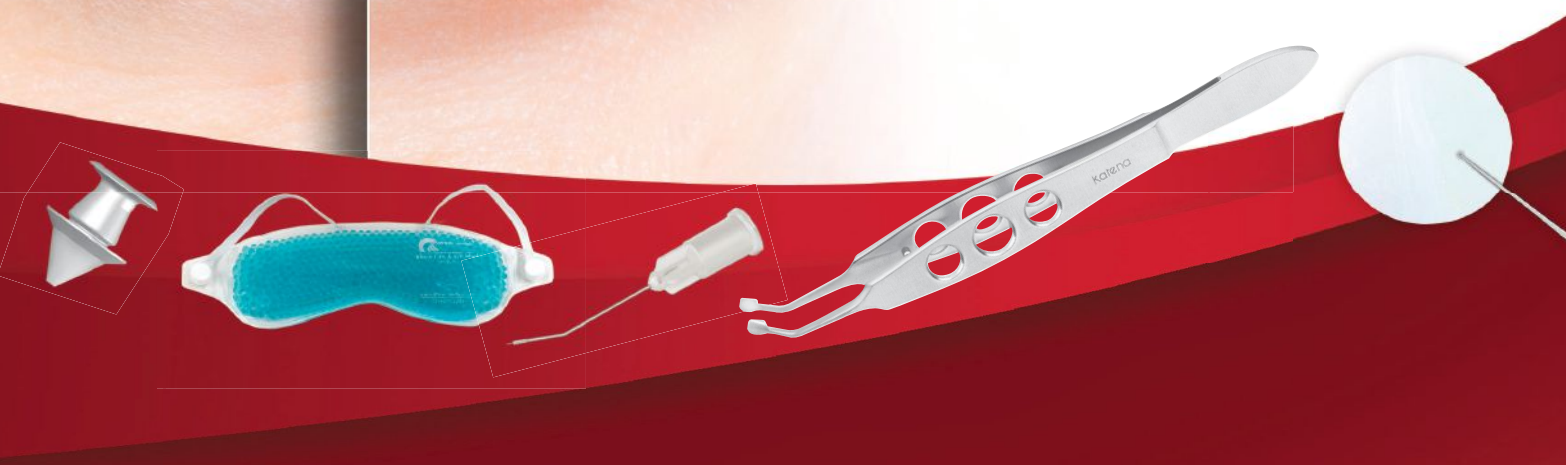

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Late to the Party

Prompt diagnosis and aggressive management of late-onset DLK guarantee optimal outcomes. **Edited by Paul C. Ajamian, OD**

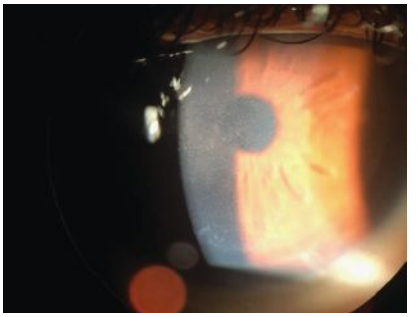
Q I have a patient who had LASIK two years ago and presented with a traumatic corneal abrasion. After the defect healed, I was surprised to see what looked like DLK. What gives?

A We most commonly think of diffuse lamellar keratitis (DLK) as a complication in the immediate postoperative days following LASIK. "Although rare, DLK can be a potentially devastating complication after LASIK, and we are all taught to keep a keen eye out for this flap-based inflammatory response at the early stage post-operative visits," says Chris Kruthoff, OD, of Northwest Eye Clinic in Golden Valley, MN. "However, this sterile inflammatory response can occur at any point, even years after this procedure, with any traumatic manipulation of the epithelium or flap."

Sterile or Infectious?

The presence of diffuse anterior stromal inflammation after an abrasion should lead to one major question—is this a sterile response, or is this infectious? "Infectious keratitis should always remain the top concern after any corneal abrasion," notes Dr. Kruthoff. Key findings include anterior chamber reaction, worsening pain, a non-healing epithelial defect and infiltrate formation. "While the corneal inflammation was troubling, we were confident it was non-infectious, with no anterior chamber reaction and the patient noting only mild discomfort."

Based on these considerations and the clinical picture at hand, a diagnosis of Stage 2 DLK secondary to the corneal abrasion was made.



Keep DLK in mind with any potential insult to the cornea down the road.

Hit It Hard

DLK presents as a collection of small, fine corneal infiltrates and forms at the interface of the corneal flap following LASIK. The condition can be graded on a four-stage scale as follows:

- Stage 1—Peripheral white blood cell accumulation with a clear central cornea.
- Stage 2—Diffuse spreading of these inflammatory cells across the entire cornea including the central cornea.
- Stage 3—Accumulation and clumping of white blood cells centrally. This stage is critical, as prompt flap lifting and irrigation is warranted to prevent corneal scarring and permanent vision decrease.
- Stage 4—Further corneal scarring and stromal melt. This stage is very rare and is associated with hyperopic shift and vision loss.

With any occurrence of DLK, either in the immediate post-op period or more long term such as this case, institute aggressive steroid therapy to prevent progression to the later stages. In this case, the patient was instructed to increase her prednisolone to one drop every hour while awake, with daily follow-up visits to ensure that she did not progress to the critical Stage 3 threshold.

"In my experience, DLK may take up to two days to show true improvement and decrease in staging as the steroid builds to counter the inflammatory response," says Dr. Kruthoff. After increasing the steroids, the patient returned the next day to ensure that the condition was improving. By the following day, the DLK was showing clear signs of regression. "We initiated a very slow steroid taper, and, by two weeks out, her cornea had completely cleared with uncorrected visual acuity of 20/20," Dr. Kruthoff said.

The presence of DLK can be a very concerning finding for doctor and patient alike. Consulting with a cornea specialist or the original LASIK surgeon is always a good idea if the diagnosis is uncertain. Occasionally, prompt flap irrigation may be needed. While we most often associate DLK with the immediate postoperative period following LASIK, keep this condition in mind with any potential insult to the cornea down the road.

"DLK can be a potential sequelae to ocular surface trauma at any point after LASIK," says Dr. Kruthoff. "Prompt diagnosis and aggressive management may quite well save your patient's vision." ■

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Look at the Bigger Picture

It could benefit both you and your patients if you approach each condition you diagnose as if it were a spectrum. **By Marc B. Taub, OD, MS, and Paul Harris, OD**

Too often, we focus on the details and, in doing so, miss the bigger picture. We tend to categorize things as black or white, leaving little opportunity for grey.

Just as there is a spectrum of colors, there too is a spectrum for many of the conditions we diagnose every day. In the world of binocular vision, it is rare that a diagnosis is as textbook or straightforward as we are led to believe. This column will focus on approaching conditions as if they are a spectrum and not a single set of findings that must occur to earn a specific label.

The spectrum of signs and symptoms of any condition, including CI, is similar to the color spectrum.

Convergence Insufficiency

One of the most common binocular vision conditions is convergence insufficiency (CI). From double-blind, placebo-controlled studies, we believe that in-office vision therapy is superior to all other treatments.[1,2] In those studies, the diagnosis of CI was based on reduced near point of convergence (6cm or greater), reduced base-out vergence ranges at near (having a positive fusional vergence value of less than twice that of the near phoria and failing Sheard's criterion or having a minimum base-out break of 15 on the positive fusional vergence) and a greater exophoria at near (at least 4).[1,2] The patients needed to be symptomatic as well.[1,2] Other signs include a low AC/A ratio, reduced vergence facility, low MEM, low negative relative accommodation and trouble clearing plus on accommodative facility. These three cases all seen by the same student in the same morning session on the same day will help highlight our treatment philosophy.

Case #1

An 11-year-old male presented with a history of frontal headaches for two years. His primary care physician referred him to determine if his vision was the issue, as sinus-related causes were also under investigation. The headaches were not task-specific or associated with visual blur, nausea or vomiting. He was an average student, read on grade-level and took Vyvanse (lisdexamfetamine dimesylate, Shire) for attention deficit hyperactivity disorder.

The patient's quality of life score was a 25, which was a red flag. His visual acuities were 20/15 at distance and at near OD, OS and OU. Stereopsis showed

20 seconds of arc, and the cover test showed orthophoria at distance and 12 exophoria at near. Retinoscopy and binocular balance were +1.50 OD, +1.75 OS and +0.75 OD, +1.00 OS, respectively. His accommodative amplitudes were 16D OD and OS. The near point of convergence was "to the nose" X 3, which binocular balance confirmed. The vergence ranges at near were X/24/11 base-in and 10/24/-1 base-out.

He was diagnosed with CI and sent to vision therapy, with instructions to continue to use his near vision glasses.

Case #2

A 15-year-old male presented complaining that he had been experiencing constant near vision blur during the previous year and trouble while reading. He had been wearing near vision glasses (+0.75 OU) for the past five years and said they help. He received good grades and read on grade level.

The patient's quality of life score was a 10. His visual acuities were 20/15 at distance and at near OD, OS and OU. Stereopsis showed 20 seconds of arc, and the cover test showed orthophoria at distance and six exophoria at near. Retinoscopy and binocular balance were +1.00-0.50 X 180 OD and OS and +0.75 OD, +1.00 OS, respectively. His negative and positive relative accommodations were +2.00 and -2.75, respectively. His accommodative amplitudes were 8D OD and OS. The near point of convergence was 15/17cm X 3, which did not improve with low plus. The vergence ranges at near were X/20/13 base-in and X/12/5 base-out.

He too was diagnosed with CI and sent to vision therapy.

Case #3

A 10-year-old female presented with complaints of blurry vision at distance for the past year despite wearing glasses (-0.75 OD, -0.25 OS). She suffers from headaches when she does not wear them. She was a good student and was on grade-reading level.

Her quality of life score was a 15. Her unaided visual acuities were 20/125 OD and 20/25 OS and OU at distance and 20/25 OD, OS and OU at near. Retinoscopy and binocular balance were both -2.00 OD and -0.25 OS, providing 20/20 acuity at distance and at near OD, OS and OU. Her uncorrected cover test showed orthophoria at distance and six exophoria at near. The near point of convergence and stereopsis were 14/17cm X 3, and stereo acuity was 70 seconds of arc. The vergence ranges at near were X/18/12 base-in and X/16/4 base-out. The near point of convergence and stereopsis were repeated with binocular balance in a trial frame. While stereopsis improved to 30 seconds of arc, the near point of convergence remained the same.

She was also diagnosed with CI but, unlike the last two cases, was referred to a research study investigating treatment options.

Discussion

While studies must be stringent in their inclusion criteria for a variety of reasons, clinicians have flexibility in practice. Even though many, if not most, cases tend to fit the textbook definition of CI, none of these three cases did. However, that should not—and did not—stop clinicians from making the diagnosis as they see fit.

In the first case, the patient's near point of convergence was not reduced, and he barely squeaked by when considering Sheard's criteria, but the cover test showed a much greater exophoria at near. In the second, the near point of convergence was reduced, and the cover test showed greater exophoria at near, but the patient barely passed Sheard's. In the third, the cover test showed greater exophoria at near, and the near point of convergence was reduced, but the patient passed Sheard's. Only the first reported a level of symptoms that should cause concern.

As when considering autism, multiple sclerosis and glaucoma, we must pay close attention to a spectrum of signs and symptoms as we clinically evaluate potential binocular vision issues. Withholding necessary treatment just because our findings do not fit a certain criteria is not an acceptable way to practice. Neither is trying to make something fit into a box it doesn't belong in. Our patients deserve the best treatment we have to offer, regardless of what box they do or do not fit into. Luckily, we're trained to find the answers our results don't always give us. ■

1. Scheiman M, Mitchell GL, Cotter S, et al. A randomized clinical trial of treatments for convergence insufficiency in children. Arch Ophthalmol. 2005;123(1):14-24.
2. Convergence Insufficiency Treatment Trial Study Group. Randomized clinical trial of treatments for symptomatic convergence insufficiency in children. Arch Ophthalmol. 2008;126(10):1336-49.

Cequa™
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Formulated with NCELL™ TECHNOLOGY

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- NCELL helps improve the delivery of cyclosporine to where it is needed[2,3]
- Significant improvement in tear production at 3 months[1]
- Significant improvement in corneal staining as early as 1 month[2,4]
- In a comfort assessment at 3 minutes post instillation, 90% (Day 0) and 85% (Day 84) of patients had no or mild ocular discomfort[4]

INDICATIONS AND USAGE

CEQUA™ (cyclosporine ophthalmic solution) 0.09% is a calcineurin inhibitor immunosuppressant indicated to increase tear production in patients with keratoconjunctivitis sicca (dry eye).

IMPORTANT SAFETY INFORMATION

WARNINGS AND PRECAUTIONS

Potential for Eye Injury and Contamination: To avoid the potential for eye injury and contamination, advise patients not to touch the vial tip to the eye or other surfaces.

Use with Contact Lenses: CEQUA should not be administered while wearing contact lenses. If contact lenses are worn, they should be removed prior to administration of the solution. Lenses may be reinserted 15 minutes following administration of CEQUA ophthalmic solution.

ADVERSE REACTIONS

The most common adverse reactions reported in greater than 5% of patients were pain on instillation of drops (22%) and conjunctival hyperemia (6%). Other adverse reactions reported in 1% to 5% of patients were blepharitis, eye irritation, headache, and urinary tract infection.

Please see brief summary of Full Prescribing Information on the adjacent page.

References: 1. CEQUA [package insert]. Cranbury, NJ: Sun Pharmaceutical Industries, Inc.; 2018. **2.** Data on file. Cranbury, NJ: Sun Pharmaceutical Industries, Inc. **3.** US Patent 9,937,225 B2. **4.** Tauber J, Schechter BA, Bacharach J, et al. A Phase II/III, randomized, double-masked, vehicle-controlled, dose-ranging study of the safety and efficacy of OTX-101 in the treatment of dry eye disease. *Clin Ophthalmol.* 2018;12:1921-1929.

PM-US-CQA-0328 10/2019

SUN OPHTHALMICS

Brief Summary of Prescribing Information for CEQUA™ (cyclosporine ophthalmic solution) 0.09%, for topical ophthalmic use

CEQUA™ (cyclosporine ophthalmic solution) 0.09%
See package insert for Full Prescribing Information.

INDICATIONS AND USAGE

CEQUA ophthalmic solution is a calcineurin inhibitor immunosuppressant indicated to increase tear production in patients with keratoconjunctivitis sicca (dry eye).

CONTRAINDICATIONS

None.

WARNINGS AND PRECAUTIONS

Potential for Eye Injury and Contamination

To avoid the potential for eye injury and contamination, advise patients not to touch the vial tip to the eye or other surfaces.

Use with Contact Lenses

CEQUA should not be administered while wearing contact lenses. If contact lenses are worn, they should be removed prior to administration of the solution. Lenses may be reinserted 15 minutes following administration of CEQUA ophthalmic solution.

ADVERSE REACTIONS

Clinical Trials Experience

Because clinical trials are conducted under widely varying conditions, adverse reaction rates observed in the clinical trials of a drug cannot be directly compared to rates in the clinical trials of another drug and may not reflect the rates observed in practice.

In clinical trials, 769 patients received at least 1 dose of cyclosporine ophthalmic solution. The majority of the treated patients were female (83%).

The most common adverse reactions reported in greater than 5% of patients were pain on instillation of drops (22%) and conjunctival hyperemia (6%). Other adverse reactions reported in 1% to 5% of patients were blepharitis, eye irritation, headache, and urinary tract infection.

USE IN SPECIFIC POPULATIONS

Pregnancy

Risk Summary

There are no adequate and well-controlled studies of CEQUA administration in pregnant women to inform a drug-associated risk. Oral administration of cyclosporine to pregnant rats or rabbits did not produce teratogenicity at clinically relevant doses.

Data

Animal Data

Oral administration of cyclosporine oral solution (USP) to pregnant rats or rabbits was teratogenic at maternally toxic doses of 30 mg/kg/day in rats and 100 mg/kg/day in rabbits, as indicated by increased pre- and postnatal mortality, reduced fetal weight, and skeletal retardations. These doses (normalized to body weight) were approximately 3200 and 21,000 times higher than the maximum recommended human ophthalmic dose (MRHOD) of 1.5 mcg/kg/day, respectively. No adverse embryofetal effects were observed in rats or rabbits receiving cyclosporine during organogenesis at oral doses up to 17 mg/kg/day or 30 mg/kg/day, respectively (approximately 1800 and 6400 times higher than the MRHOD, respectively).

An oral dose of 45 mg/kg/day cyclosporine (approximately 4800 times higher than MRHOD) administered to rats from Day 15 of pregnancy until Day 21 postpartum produced maternal toxicity and an increase in postnatal mortality in offspring. No adverse effects in dams or offspring were observed at oral doses up to 15 mg/kg/day (approximately 1600 times greater than the MRHOD).

Lactation

Risk Summary

Cyclosporine blood concentrations are low following topical ocular administration of CEQUA. There is no information regarding the presence of cyclosporine in human milk following topical administration or on the effects of CEQUA on breastfed infants and milk production. Administration of oral cyclosporine to rats during lactation did not produce adverse effects in offspring at clinically relevant doses. The developmental and health benefits of breastfeeding should be considered along with the mother's clinical need for CEQUA and any potential adverse effects on the breastfed child from cyclosporine.

Pediatric Use

The safety and efficacy of CEQUA ophthalmic solution have not been established in pediatric patients below the age of 18.

Geriatric Use

No overall differences in safety or effectiveness have been observed between elderly and younger adult patients.

PATIENT COUNSELING INFORMATION

Handling the Vial

Advise patients to not allow the tip of the vial to touch the eye or any surface, as this may contaminate the solution. Advise patients also not to touch the vial tip to their eye to avoid the potential for injury to the eye.

Use with Contact Lenses

CEQUA should not be administered while wearing contact lenses. Patients with decreased tear production typically should not wear contact lenses. Advise patients that if contact lenses are worn, they should be removed prior to the administration of the solution. Lenses may be reinserted 15 minutes following administration of CEQUA ophthalmic solution.

Administration

Advise patients that the solution from one individual single-use vial is to be used immediately after opening for administration to one or both eyes, and the remaining contents should be discarded immediately after administration.

Rx Only

Distributed by: Sun Pharmaceutical Industries, Inc.
Cranbury, NJ 08512

PLR-00020 2018

SUN OPHTHALMICS

The Future of Case Histories

Recent and upcoming changes in how you take these can make you more efficient.

By John Rumpakis, OD, MBA, Clinical Coding Editor

The foundation for each and every professional encounter begins with the case history; from a coding perspective, it is the most integral portion of your encounter. Since the adoption of the Resource Based Relative Value System (RBRVS), for coding compliance, the scorable elements of the case history were:

1. Chief complaint—primary reason for visit
2. History of present illness
3. Review of systems
4. Past family and social history

The chief complaint always drives the encounter, both from a perspective of what type of service you are providing—ophthalmic office visits or E/M visits—and the level of service provided. A properly recorded chief complaint also determines who is the responsible party for charges and payment for the respective services, whether it be a managed vision care plan, a medical carrier or the patient.

Four different levels of case history scoring exist: problem-focused, expanded problem-focused, detailed and comprehensive.

The case history is often overscored because individuals count all elements in the review of systems category that are asked and answered, rather than only those pertinent and germane to the chief complaint and patient presentation.

To see the current case history scoring, visit www.reviewofoptometry.com, or scan the QR code.

From a rules perspective, traditionally, the physician had to retake and reenter all of the information about the patient's chief complaint and history that a staff member had already taken; but as of 2019, physicians only have to document in the medical record that they "reviewed and verified" the chief complaint and history information already recorded by ancillary staff or the patient, thus saving time and eliminating redundancy for all.

In our current system, the level case history you perform must be commensurate with the patient's presenting problem. You cannot perform the same level of case history on everyone because it is "your standard of care." It must be an individualized case history based upon the patient's presenting problem.

Beginning January 1, 2021, the case history will no longer be individually scored as it contributes to the level of E/M service performed. The history and physical will not be elements for code selection and scoring. While capturing the patient's pertinent history and performing a relevant physical exam contributes to the physician's time and medical decision making, these elements alone should not determine the appropriate code level. The AMA and CMS workgroup revised the code descriptors to state that providers should perform a "medically appropriate history and/or examination," so next year the individual elements of the history taken won't matter with respect to the level of office visit.

To illustrate, the 2021 definition of a 99203 eliminates specific language about component requirements and leaves things more in the physician's hands. The current definition states: office or other outpatient visit for the evaluation and management of a new patient, which requires these three components: *a detailed history*; a detailed examination; medical decision making of low complexity. Counseling and/or coordination of care with other providers or agencies are provided consistent with the nature of the problem(s) and the patient's and/or family's needs. Usually, the presenting problem(s) are of moderate severity. Physicians typically spend 30 minutes face-to-face with the patient and/or family.

The 2021 definition is: office or other outpatient visit for the evaluation and management of a new patient, which requires a *medically appropriate history and/or examination* and low level of medical decision making. When using time for code selection, 30 to 44 minutes of total time is spent on the date of the encounter.

Case history will always remain the foundation for type and level of services performed and a key element to determine the scoring of your professional services. Changes in 2019 and those coming in 2021 will give you more control of what you do and how you do it, while still being compliant with CPT guidelines and rules. ■

Send your coding questions to rocodingconnection@gmail.com.

PEER • REVIEWED • PRA • ARTICLE

How Do OCT Devices for Glaucoma Compare?

An in-person review of four unique instruments helps provide insight on what each can offer glaucoma patients. **By Ryan Schott, OD**

To manage glaucoma, OCT is a must-have technology and one of the biggest hurdles practitioners face when adding care of this disease to their practices. The devices provide sophisticated detail on anatomical status and disease risk, but the sheer volume and complexity of the data can be daunting, as there are features and reports galore that can easily overwhelm us. Having numerous technology platforms to choose from only complicates matters further. Each OCT device performs the core components needed for glaucoma care admirably well, but they can differ in scanning capabilities and data output. Which distinctions between instruments matter clinically, and which don't?

Last fall, *Review of Optometry* invited a patient from our practice and me to the 2019 American Academy of Optometry meeting to survey four spectral-domain OCT (SD-OCT) manufacturers—Heidelberg, Optovue, Topcon and Zeiss—together in one location. Due to the close proximity of our office, the venue was perfect for a comparative demonstration. This patient accompanied us into the exhibit hall to be tested on each instrument with all of the glaucoma modules available: optic nerve head (ONH), retinal nerve fiber layer (RNFL), macula and OCT angiography (OCT-A). We also obtained fundus photos from each company using separate retinal cameras or on-board fundus imaging within the OCT (*Figure 1*).

Standardizing the patient removed one key variable from the task of comparing technology platforms, as company literature will of course use patients unique to each manufacturer. Here's what we found.

Scan Acquisitions

Our patient was a 65-year-old white male recently diagnosed in our clinic with pre-perimetric low-tension glaucoma OD, with no repeatable defects yet on either his 24-2 or 10-2 testing. He reported his father had glaucoma and had to use medications for years to maintain his sight. He is active and healthy, with no comorbidities or medication usage and an average normal resting blood pressure of 113/75mm Hg. His intraocular pressure (IOP) ranges from 15mm Hg to 19mm Hg, and he had a slightly thinner cornea of 515µm. He has larger than average vertical cup-to-disc ratios of 0.65/0.60 OD/OS with a noticeably thinner superior temporal rim OD and RNFL wedge defect visible on fundus photography and confirmed with our in-house OCT.

Aside from the most commonly used RNFL analyses to aid in the detection and progression of glaucoma, we are fortunate to have optic disc and macular thickness (more specifically, retinal ganglion cell) analyses as well. For the clinician new to OCT and glaucoma, historically, close attention was always given to the RNFL average and inferior/temporal and superior/temporal (S/T) RNFL thicknesses. These were, respectively, the most sensitive and reproducible data points in regards to glaucomatous nerve fiber layer damage.[1] More recently, research is showing that macular ganglion cell loss may be just as sensitive as RNFL loss in diagnosing pre-perimetric glaucoma and may be especially useful in highly myopic eyes where traditional RNFL scans suffer.[2]

The introduction of OCT-A has also given us another method of analyzing structural loss in glaucoma, which

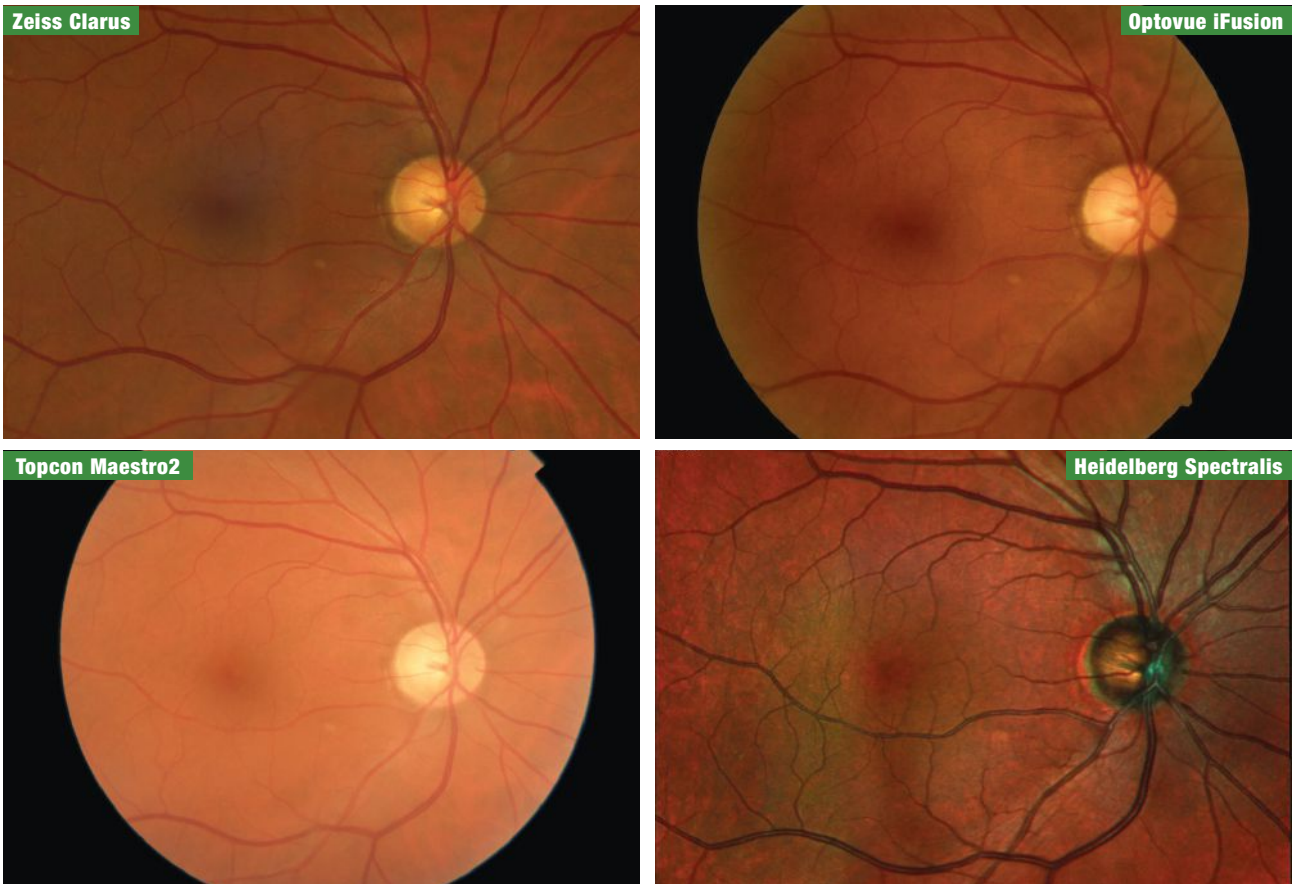


Fig. 1. Fundus images of the patient from the four manufacturers, taken at the 2019 Academy of Optometry meeting in Orlando. From top left clockwise are Zeiss Clarus, Optovue iFusion, Heidelberg Spectralis and Topcon Maestro2.

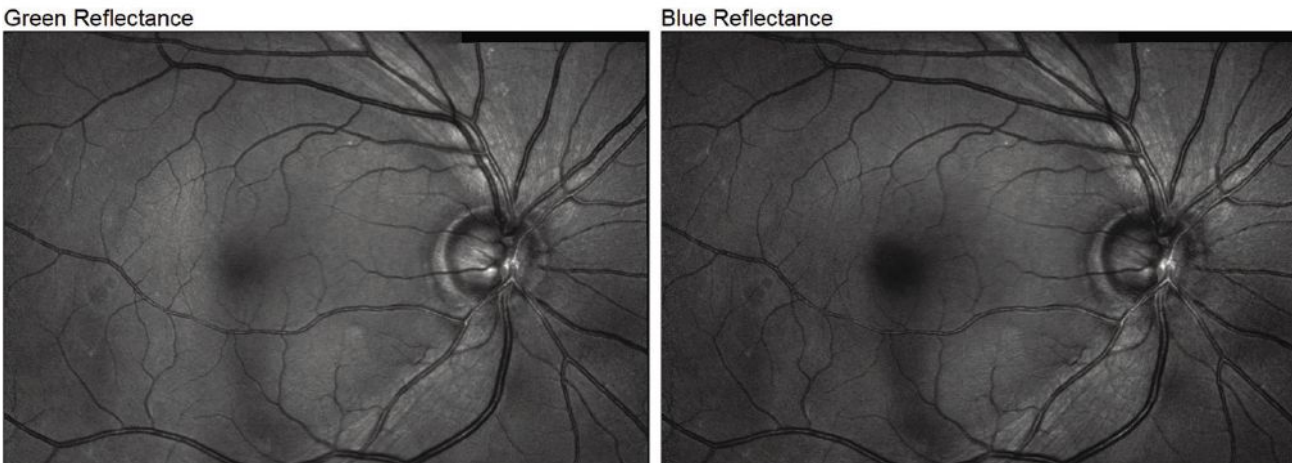


Fig. 2. The Spectralis's multicolor imaging mode demonstrates the S/T defect in our glaucoma patient. It can sometimes be difficult to appreciate NFL defects in fundus photos, especially for early-stage patients such as this one, but the above example highlights it exceptionally well.

looks promising as another key indicator in diagnosing glaucoma. The four manufacturers we tested now have the capability of providing OCT-A as well (Topcon is working on clearance from the FDA for OCT-A capability on its swept-source OCT Triton, but it is unknown at this time if it will be included on the Maestro).

Given the findings of our show floor test—in which all four manufacturers had excellent agreement in their analyses with no statistically significant difference in detecting glaucomatous loss, your unique needs in your practice should determine which platform you select.[2] In short, I found all four instruments equally valid tools for evaluating our test patient. Nevertheless, they do have some unique ways of acquiring and presenting data, which I'll review below. The opinions are entirely my own, and in fact you may look at the very same scans and arrive at different conclusions.

Heidelberg Spectralis

The images produced by the Heidelberg Spectralis are of outstanding quality. The ability to package in multiple modes of imaging including Blue Peak (fundus autofluorescence; FAF), multicolor high-contrast fundus imaging, anterior and posterior OCT and angiography into one platform is extremely convenient for the disease-focused practitioner. With regards to fundus imaging, of all the instruments we tested, I think the multicolor imaging module demonstrated the s/t defect in our glaucoma patient the best (*Figure 2*).

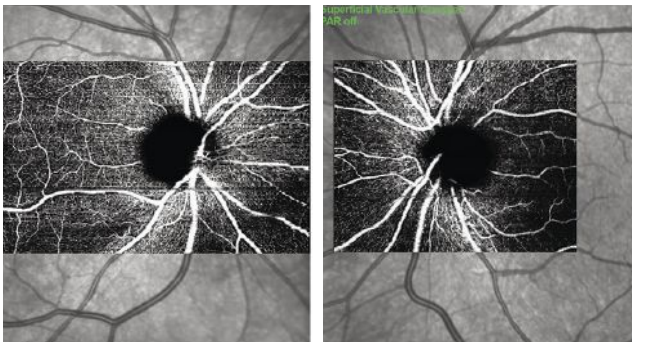


Fig. 3. OCT-A scans OD/OS from the Spectralis showing mild superotemporal capillary dropout OD.

The OCT-A images also showed some mild superotemporal dropout consistent with our clinical findings and expectations (*Figure 3*)

One caveat about the angiography module I noticed during our demonstration—the image capture of the

Table 1. Comparison of Commonly Used OCT Devices *(includes others not tested)*

	Optovue Avanti	Zeiss Cirrus 6000	Topcon Maestro2	Topcon Triton	Heidelberg Spectralis	Nidek RS-3000 Advance	Canon Xephilio OCT-A1
Imaging platform	SD-OCT	SD-OCT	SD-OCT	SS-OCT	SD-OCT	SD-OCT	SD-OCT
Optical source	840nm superluminescent diode (SLD)	840nm SLD	840nm SLD	wavelength-swept laser at 1,050nm	880nm SLD	880nm SLD	855nm SLD
Scan speed (A-scans/sec)	70,000	100,000	50,000	100,000	85,000	53,000	70,000
A-scan depth	3.0mm	2.0 – 2.9mm (in tissue)	2.3mm	2.6mm	1.92mm	2mm	2.0mm
Axial resolution	5µm	5µm (in tissue)	6µm	8µm (optical), 2.6µm (digital)	7µm (3.9µm/pixel)	7µm	3.4µm (optical) 1.6µm (digital)
Transverse resolution	15µm	15µm (in tissue)	20µm	20µm	14µm (5.7µm/pixel)	20µm	20µm
Imaging modes	Widefield *en face* OCT, OCT-A	Posterior segment, anterior segment, OCT-A, fundus imaging	Fundus (color, red-free, infrared), anterior and posterior segment (color, infrared), anterior and posterior OCT	Fundus (color, red-free, infrared), anterior segment (color, infrared), anterior and posterior OCT, OCT-A (only available in Europe)	Anterior segment, widefield, fundus imaging, IR, blue-reflectance (red-free), multicolor, scanning laser angiography, OCT-A, ultra-widefield, high magnification	Fundus surface imaging, anterior segment OCT (optional module), posterior segment OCT, EDI-OCT	Fundus (SLO), anterior segment, posterior segment
Normative database	442 eyes	284 eyes	399 eyes	410 eyes	368 eyes	N/A	520 eyes

angiography scan has multiple sizes. For our patient's right eye, the scan taken included the entire papillomacular bundle, with a horizontal scan width of 8.3mm. The sheer size and detail of the scan required an image acquisition time of minutes, not seconds. We opted for a smaller scan size for the left eye to minimize our patient's discomfort. The smaller scan still gives a thorough evaluation of the peripapillary angioplex with a much shorter acquisition time.

Even ignoring the OCT-A module, it is my personal opinion that this would be the most difficult instrument of the four tested to delegate to your technicians. That being said, the Spectralis has such a fantastic array of configurations and high level of detail that make the steeper learning curve easily justifiable. Of all the instruments we tested, it was the only one to offer a FAF module. Though we don't use FAF in our clinic for glaucoma monitoring, we use it regularly to monitor AMD, differentiate optic disc drusen and screen for drug-induced maculopathies.

The configuration during our demonstration of the instrument and separate PC unit did make for a larger footprint, which should be considered in a smaller clinic where space is at a premium.

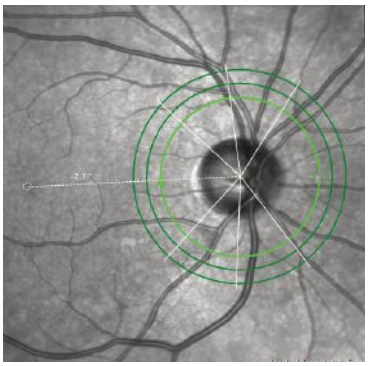
The quality of the OCT images from the Spectralis are fantastic, and the reports are plentiful (*Figures 4 and 5*). The Hood report and anatomic positioning system (APS) can be purchased with the Glaucoma Module Premium Edition (GMPE). The APS feature maps the anatomic position of the center of Bruch's membrane opening and the center of the fovea to establish an angle of reference for future scan comparisons as well as a consistent horizontal delineation between the superior and inferior structures for comparative analyses.

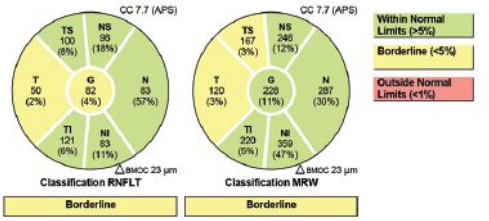
The Hood report rearranges some of the data on the typical temporal, superior, nasal, inferior, temporal (TSNIT) curve, so that the curve starts on the nasal region and the temporal region thickness, which is more likely to be compromised in glaucoma than nasal RNFL, is located centrally (*Figure 6*). It also overlays a visual field simulation plot for both 24-degree and 10-degree chords.

Interestingly, the clear thinning and RNFL defect found S/T with our patient was not flagged as borderline or outside normal limits on the S/T RNFL map sector report, but it was found to be borderline on the minimum rim width report.

Scenes from the Show Floor

During the Academy 2019 meeting in Orlando, I recruited Ed Searl— a recently diagnosed glaucoma patient who also happens to be the optician at my practice—to join me in the exhibit hall and be scanned by all the OCT devices available. New to the disease (I diagnosed him about six weeks prior to this event), he was still adapting to the knowledge of the possible course of therapy ahead of him. Still, he gamely undertook several sessions of photography and OCT scanning as we made the rounds that day. Here he is putting the devices through their paces.





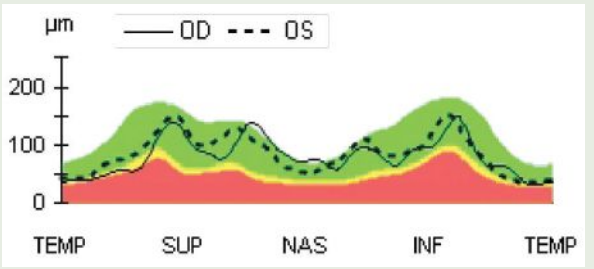
Figs. 4 and 5. Left: the ONH/RNFL scan indicating the Anatomic Positioning System on the Spectralis. Above: the Garway-Heath sectors from the Hood report, as determined by the Spectralis.

Optovue Avanti

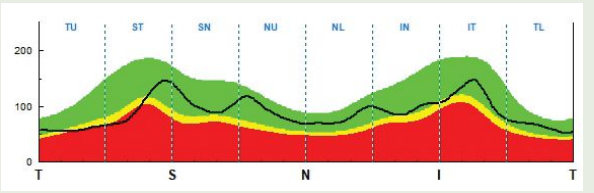
When using the Optovue Avanti with Angiovue software, in a matter of just a few minutes we had captured ONH, RNFL, macular ganglion scans, as well as some beautiful angiography images. The ease of use was simple enough. The RNFL and ONH report are similar to the other manufacturers' OCT instruments, with a typical TSNIT

Fig. 6. TSNIT vs. Hood for RNFL Analysis
OCT devices use either conventional graphs of RNFL thickness in the TSNIT sequence, or the alternative approach championed by Donald Hood, MD, which places the temporal segment (more vulnerable in glaucoma) in the center of the graph. Here's how the four devices rendered this data.

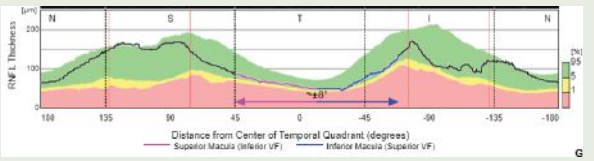
Zeiss Cirrus:



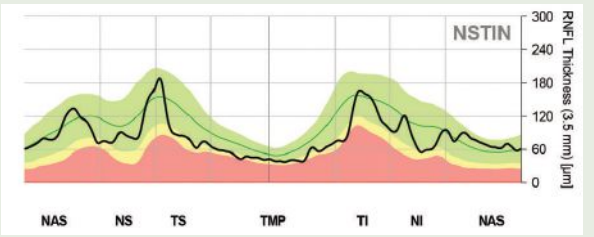
Optovue Avanti:



Topcon Maestro2:



Heidelberg Spectralis:



plot, an RNFL scan broken into eight segments, a line scan showing the contour of the disc as well as average thicknesses of the RNFL and ONH parameters and their corresponding relation to the age-matched normal indices. The RNFL and ONH scan on our patient confirmed our suspicions and agreed nicely with our clinical findings showing a thickness in the superotemporal region found in <1% of the normative database.

The ganglion cell complex (GCC) scan and report from Optovue is unique in comparison with all the other manufacturers. A cursory glance will give the impression the scan is off center from the macula, which is intentional. The scan is moved temporally, as this provides a higher concentration of ganglion cells to include in their global loss volume metric for early detection. A large review of SD-OCT also found that the nasal macular ganglion cell thickness to be less reliable than the superior and inferior temporal thicknesses.[2]

A report offered by Optovue includes the RNFL, ONH and macular GCC, encompassing all the parameters the typical practitioner could want (*Figure 7*). However, I do prefer having a more bird's-eye view of the ONH map image, as it can be difficult to subjectively appreciate the RNFL defect in the included map.

The OCT-A images provided by the Angiovue software were stunning, and in my opinion, the most detailed of all the instruments we tested (*Figure 8*). For our patient, the radial peripapillary capillary vessel density image shows capillary dropout corresponding to the RNFL defect superior temporal OD. Though we only performed angiography scanning of the ONH, there are benefits to angiography scanning of the macula in both glaucoma and various macular diseases.

The footprint of the instrument is reasonable, and the Avanti offers the addition of anterior segment maps and corneal OCT scans that round out an excellent package. Optovue also offers multiple NetView packages to allow the practitioner to view and manipulate the images from either their workstation, tablet or smartphone. Flow throughout the clinic is just as important as the data you receive. As reimbursements for OCT have decreased over the past decade, efficiency in analyzing the scans and making decisions come at a high premium, so it is considerate of Optovue to make their software as individualistic as possible by offering multiple options with NetView.

Topcon Maestro2

Topcon could not have made OCT capturing any easier by introducing the Maestro2, a near fully automated instrument combining fundus photography, anterior OCT, posterior OCT and possibly in the future (pending FDA clearance) OCT-A as well. Due to the automation of the Maestro2, it was by far the fastest instrument we tested, as the speed of the acquisition surprised both me and our patient. However, if you have an SD-OCT in your office now, you'll understand that the actual scanning time is negligible. It's the alignment, focusing and image plane selection that takes so much technician time. To have an automated instrument in a busy practice would be valuable, but I would wonder about reliability in patients who have fixation loss, such as a patient with

advanced macular degeneration with geographic atrophy. Would they be able to fixate where necessary? In such cases, manual fixation will need to be used.

Another nice feature: the rotatable control display makes the unit flexible to work in nearly any clinical setting and lets the tech and patient sit side by side if desired.

Including true color fundus photography with a nearly automated OCT in the same platform is enticing to the clinician who is looking to conserve space and save time. I would imagine that if I were in a corporate location with little space available to dedicate to imaging and had inconsistent staff or assistants with less imaging experience, the Maestro2 could be the perfect option.

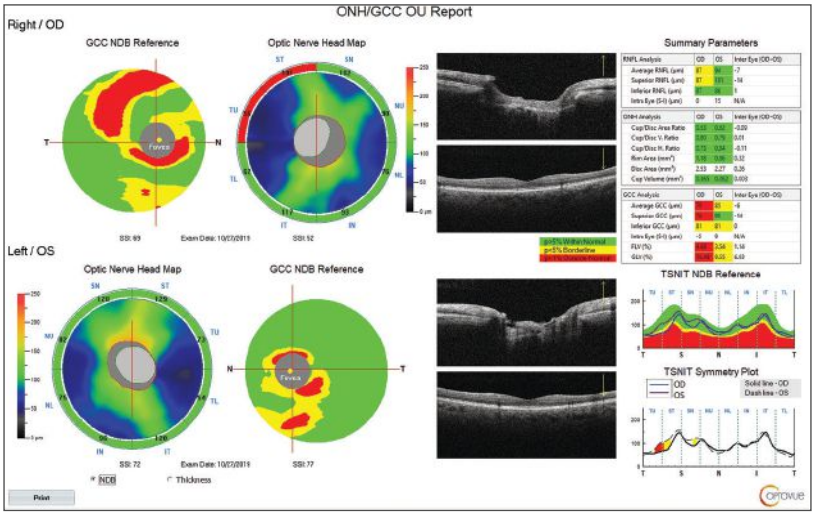


Fig. 7. This report from the Optovue Avanti includes numerous parameters that can help assess a glaucoma patient. One nice addition: an actual live OCT scan of the macula is visible, in addition to the contour map, to help the clinician recognize correspondence to anatomical structures.

Topcon's OCT platform offers a plethora of reports, but I especially like the one included here due to the abundance of data and inclusion of the color and red-free photography (*Figure 9*). The RNFL analysis is broken into a typical 12 o'clock and four quadrant setup like the Cirrus platform.

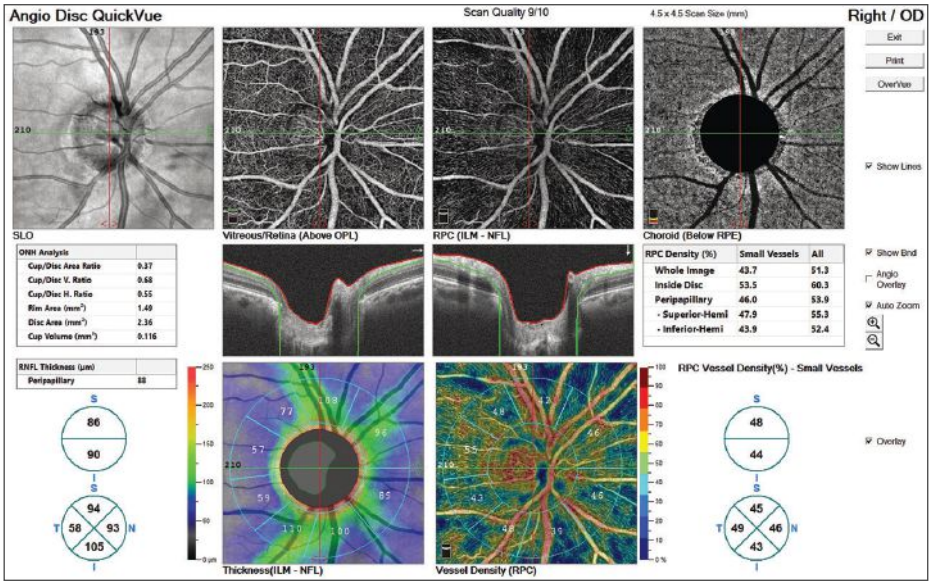


Fig. 8. The AngioVue software rendered the vascular network in rich detail. Note the capillary dropout in the vessel density map, which corresponds to the RNFL dropout. While the clinician would arrive at the same awareness of nerve fiber loss without OCT-A, it aids confirmation.

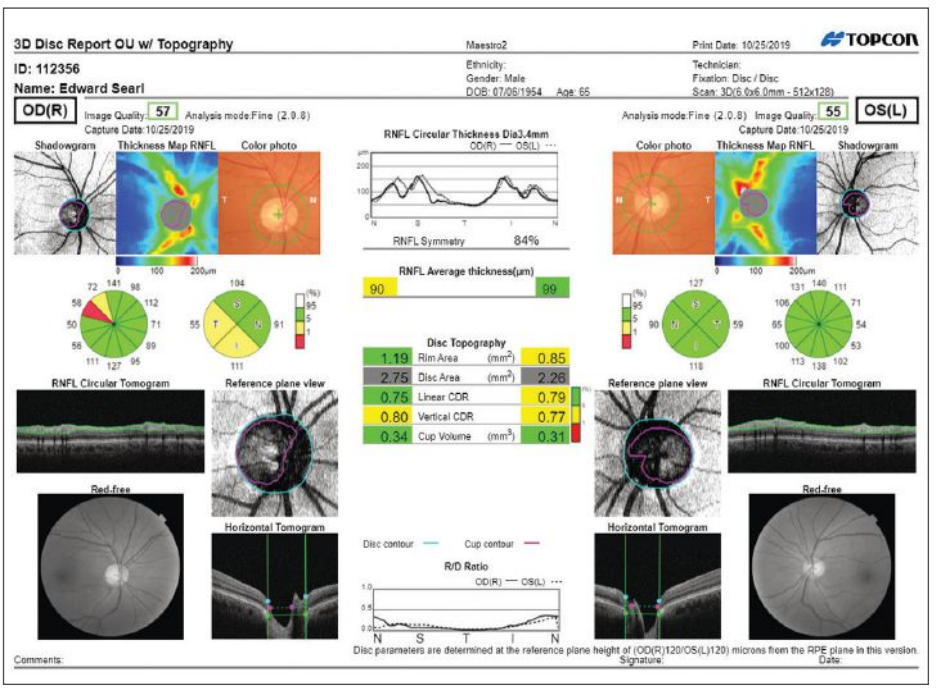


Fig. 9. Essentially every piece of diagnostic data a clinician might need is present in this Topcon Maestro2 report, including true color and red-free fundus photography.

The disc topography analysis offers age-matched normal comparisons as well. Topcon, like Heidelberg, also offers the Hood report for clinicians who prefer the shifted circumpapillary RNFL and simulated threshold map. Topcon's Hood report does differ slightly from Heidelberg's as a larger area of the retina is scanned, and the threshold overlay that is included on the Maestro differs from the Spectralis.

Unless the exclusion of angiography at present is a dealbreaker, the Maestro2 should be on the short list for the practitioner considering adding OCT in their office.

Zeiss Cirrus 6000

We've been using the Zeiss Cirrus 4000 in our office for almost eight years, so we are quite familiar with this platform. Nonetheless, I was still impressed with the upgrades Zeiss has added to its OCT family.

The Cirrus 6000 is still a remarkably easy platform to use and to train your technicians to operate, and certainly images and processes much faster than our current model. It boasts one of the smallest footprints yet to date. The layout of the instrument is similar to previous versions, with the technician sitting to the right of the patient. The speed of the instrument approaches that of the swept-source OCT units in the research facilities, with the company reporting speeds of nearly 100,000 scans/second.

I was pleased to see that the reports have changed little from our older-model OCT. This makes comparing data in the future when upgrading instruments seamless to the clinician and avoids any mental arithmetic when trying to compare apples to oranges.

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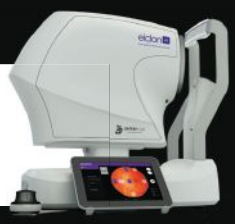

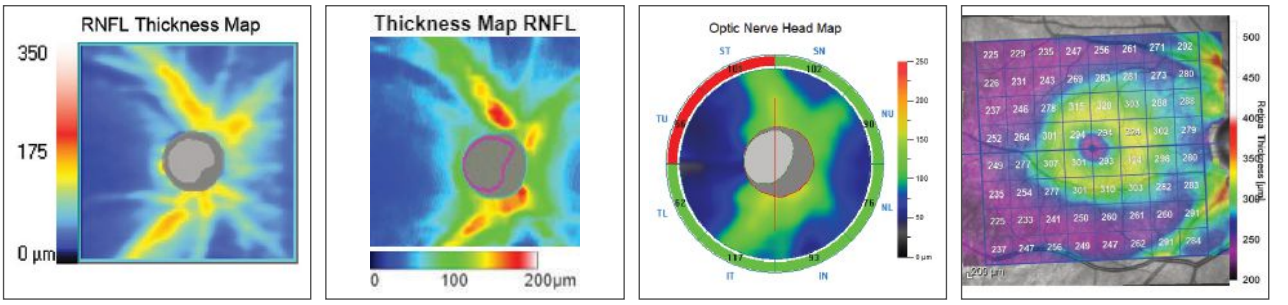

Fig. 10. RNFL thickness map comparison. Left to right: Zeiss Cirrus 6000, Topcon Maestro2, Optovue Avanti, Heidelberg Spectralis. The colors represent deviation from each device's normative database. The Topcon and Zeiss images highlight the RNFL wedge defect by the blue streaks superotemporally. All four instruments will ultimately detect the RNFL defect, but it comes down to personal preference in how you use the data and visuals to get there.

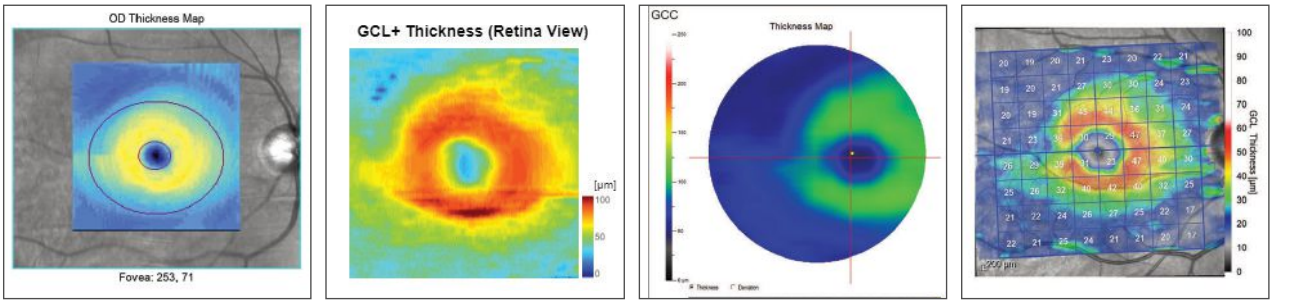

Fig. 11. Ganglion cell layer comparison. Left to right: Zeiss Cirrus 6000, Topcon Maestro2, Optovue Avanti, Heidelberg Spectralis. Note how all four instruments render the superior/inferior asymmetry.

Similar to Topcon's report layout, the Zeiss RNFL, ONH and ganglion cell report both break up the RNFL into an average thickness parameter, quadrants and clock hours (*Figures 11 and 12*). The divisions are then compared with the normative database also, with green representing 95% of normals, yellow <5% and red <1%. ONH analysis with normal comparison is also included.

I actually prefer Zeiss's simulated RNFL thickness map for RNFL defect visualization over the others. I fully admit this may be a habituation bias, but if you look closely at the similar images provided by the four manufacturers, it's my opinion that Zeiss offers a great image for the clinician looking to detect—not necessary quantify—RNFL defects (*Figure 10*). While the accuracy and repeatability of objective analysis is highly important, much of our day-to-day decision-making centers around the question, "Is this normal or not normal?" Often I'll have a patient with a blonde fundus, which complicates RNFL analysis on photographs and biomicroscopy, but the Cirrus platform demonstrates the visible RNFL dropout in these cases quite well.

Notice the visible dark blue slit on the Zeiss image S/T OD. This area can be difficult to appreciate on some fundus photography but is highlighted easily here. I find this a useful way to confirm or double check imaging results and funduscopic suspicions.

We have been using SD-OCT in our practice since 2011, so we have data going back nearly nine years. This has been extremely helpful in our clinic. The secondary but invaluable uses in illuminating macular pathology, angle anatomy and scleral lens fine tuning are wonderful, but they take a back seat to the primary function of diagnosing and managing primary open-angle glaucoma in our office.

Though our OCT is becoming dated, it holds thousands of scans that provide so much data that it would be hard to swallow a loss of the historical care by switching to another platform. When we do eventually

Thoughts on RNFL Thickness Measures

There was good correspondance among the four devices in RNFL thickness measurements in the affected eye: 82µm for Spectralis and Cirrus, 87µm for Avanti and 90µm for Maestro2. When comparing to age-matched normals, all but the Cirrus flagged it as below 5%. This highlights the importance of not making a decision on the red/yellow/green deviation measures alone. The RNFL thickness map on the Cirrus demonstrated the wedge defect with unambiguous clarity even though it reads as 'green' on deviation assessment.

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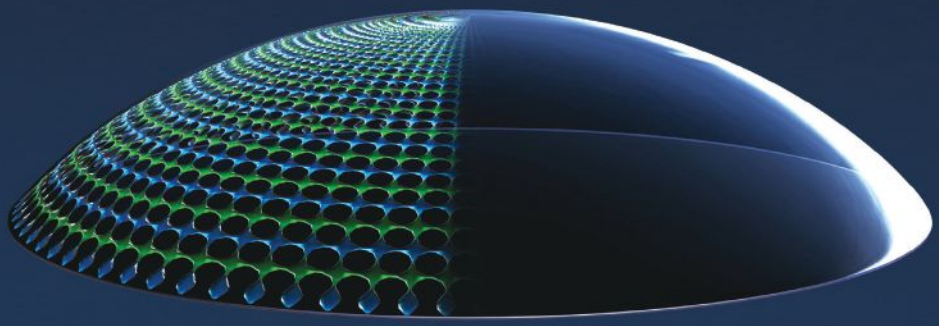

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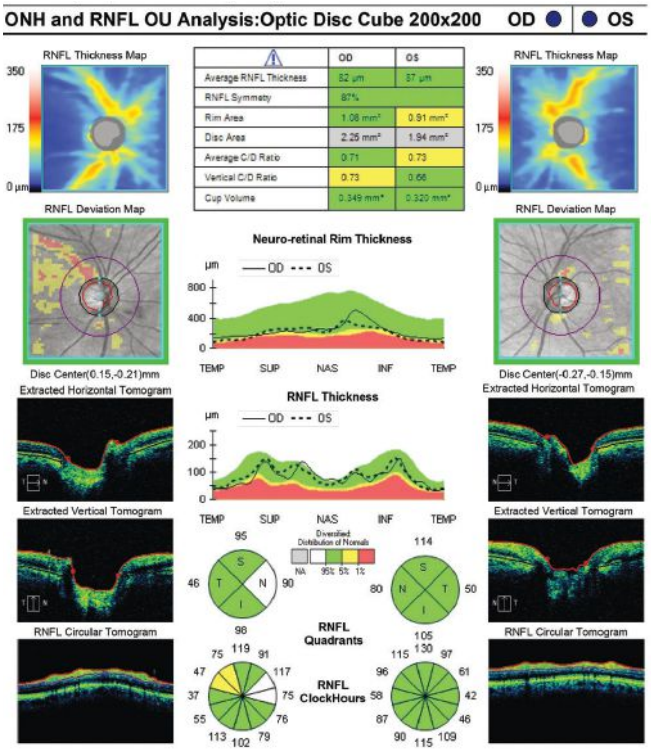

Fig. 12. This summary report from the Zeiss Cirrus 6000 retains the simplicity and familiarity of the company's long history in OCT applications for glaucoma. This report also flags the ONH disc area (gray line in the table) as outside normal parameters, which suggests a need for caution as this may be an anatomically larger disc.

upgrade our instrument, I anticipate we will stay with the Zeiss family due to their backwards compatibility. If you are using OCT primarily for other conditions besides glaucoma, I believe you are much more justified in switching platforms if this is your consideration. It must be repeated, however, that the data you have from one manufacturer is not transferrable to another. There is no continuity of care in glaucoma when switching platforms, so moving from one manufacturer to another will confound your progressive decision-making with your glaucoma patients.

Zeiss offers a separate picture archiving and communication system software called Forum. Our office has used this daily since adding our Cirrus platform. It must be considered, however, that this is a separate server-based imaging management software that must be purchased in addition to the instrument.

Final Thoughts

Our little show floor expedition helped me to see just how far OCT technology has evolved, especially in

glaucoma care, and to better understand the similarities and differences among the platforms. But clearly we only scratched the surface in this comparison.

For instance, we were largely unable to consider serial analysis of glaucoma patients to assess markers of disease progression, which is an absolutely critical element of care, since we only performed one scan on one day at each site. The beauty of today's SD-OCT tech is that you receive a repeatable and objective measurement of the optic disc and RNFL to help you isolate the suspects from the normals. The power these devices hold is the ability to repeat the measurements over time and further delineate the progressors from those with normal anatomical variation.

Progression analysis software and the integration of structure/function data were two important aspects of OCT we simply couldn't address in a single visit. Anyone interested in adding OCT or upgrading to a new device should do their due diligence with all the devices out there. We only tested one model from each of the big four manufacturers with most of the bells and whistles, but there are numerous models and configurations with each manufacturer—not to mention the two others we weren't able to try, Nidek and Canon, both of which offer high-performance OCT devices with custom glaucoma analytics, too.

It would also be wise to consider the longevity of the platform, repairability and warranty options offered by the manufacturers, as repairs can be costly. Though we've never had any direct issue with the PC driver of our Cirrus, I would imagine that the Spectralis and Optovue do have an advantage of having a separate and easily replaceable PC. Of course, the downside to a modular OCT system without an integrated PC is a much larger footprint, which could be a challenge in a smaller office. For those purchasing for the first time, I'd highly recommend doing a demonstration with all the manufacturers you are considering.

OCT has established itself as a technological pillar in eyecare, so if you're considering your first purchase, choose what fits your practice the best, as you will likely have continuity in the future with any of the manufacturers tested. ■

Dr. Schott owns and operates Kindred Optics at Maitland Vision in Maitland, FL. He is currently a member of the American Optometric Association and the Central Florida Optometric Society.

1. Park SB, Sung KR, Kang SY, et al. Comparison of glaucoma diagnostic capabilities of Cirrus HD and Stratus optical coherence tomography. Arch Ophthalmol. 2009;127(12):1603–9.
2. Kansal V, Armstrong JJ, Pintwala R, Hutnik C. Optical coherence tomography for glaucoma diagnosis: An evidence based meta-analysis. PLoS One. 2018;13(1):e0190621.

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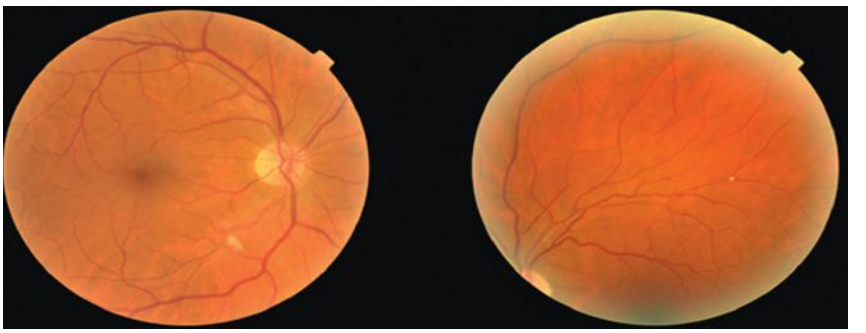
Diagnostic Skills and Techniques

Hints from the History

Be aware of the not-so-red flags in the medical history that should pique your interest about possible ocular associations. **By Sara Weidmayer, OD**

During every comprehensive eye examination, perusing the patient's medical history is a crucial first step, even when they present without any visual complaints. Many of our patients are being treated for a systemic condition, and either the disease itself or its treatment regimen—or both—could come with ocular manifestations the patient may not be aware of.

While the more common systemic issues become second nature (think diabetes), other details in the medical chart are easily overlooked because they may not immediately seem related to the eyes. When we notice these less-obvious flags in the medical history or during the review of systems, we should adjust our approach to ensure we are looking more closely for the (sometimes subtle) ocular associations. Even when the subsequent exam shows that the ocular system remains unaffected, we should ensure our patients are acquainted with the possible systemic and ocular manifestations that they need to watch for.



This patient had a Hollenhorst plaque in the superior-temporal arcade of each eye and a cotton-wool spot inferior-temporal to the right optic nerve. He had 70% to 99% stenosis of the right carotid artery and insignificant disease on the left. His echocardiogram was unremarkable. He underwent right carotid endarterectomy.

Cardiovascular & Cerebrovascular Disorders

This includes the common metabolic trifecta: diabetes, hypertension and hyperlipidemia. These are so routine in my daily practice that it's almost surprising when patients *don't* have at least one of them, but their ever-increasing frequency doesn't negate their importance. Every optometrist should already be well-versed in caring for patients with these comorbidities.

However, many other pertinent cardiovascular abnormalities might show up in your patient's medical record and give you pause. It's important that you educate these patients about cardiovascular optimization, including smoking cessation, and symptoms warranting emergency evaluation. Here are four of the more common ones:

Cerebral arteriovenous malformations (AVMs). These are a complex knot of directly fistulating arteries and veins, without a normal capillary bed in between. They can hemorrhage and cause significant morbidity and mortality associated with hemorrhagic stroke (cerebrovascular accident, CVA); in fact, they account for 2% of all hemorrhagic strokes.[1]

AVMs in or near the visual pathway can cause visual field defects associated with both mass effect from the AVM itself or from its bleeding, or from ischemia related to stroke. Occipital AVMs can cause headaches and visual symptoms similar to migraine auras. Brainstem AVMs may produce diplopia, abnormal extraocular function (cranial nerve III palsy, gaze palsy) and pupil abnormalities (anisocoria, light-near dissociation).[2] As congenital malformations, these predominantly affect younger and otherwise healthy patients.[1]

- *Think of:* stroke.
- *Look for:* visual field changes.
- *Educate patients about:* transient ischemic attack (TIA)/CVA symptoms, aura-like symptoms, visual field changes, headaches, diplopia and other neuro-ophthalmic findings.

Carotid artery stenosis or occlusion. Atheromatous plaques along the carotid arteries can lead to insufficient blood flow from the internal carotid to the ophthalmic and ultimately to both the inner retinal arterial and choroidal supply, which can also lead to general ocular ischemia (think hypoperfusion retinopathy, which can lead to ocular ischemic syndrome). These carotid atheromatous plaques can also ulcerate and lead to embolic strokes—both cerebral or intraocular (Hollenhorst plaques, which may cause retinal artery occlusions).

Optometrists must remember to educate patients on transient monocular vision loss (TMVL, also known as amaurosis fugax), particularly for these patients. This indicates transient ischemia to the eye and qualifies as a TIA, which should warrant *emergency* medical evaluation and stroke workup. Remember that the patient's highest risk of having a CVA is within 48 hours of a TIA, so TIA/CVA symptoms (including TMVL) should be reviewed regularly with patients who are at increased risk.

Atrial fibrillation (AFib). When the atria fibrillate, they do not pump normally; instead, blood pools, putting patients at a higher risk of clotting. This can cause thromboembolic vascular occlusions (ophthalmic, cerebral), which can occur even when the patient is anticoagulated. TIA/CVA/TMVL warnings are also pertinent in these patients.

Valvular heart disease. Calcific stenosis of the mitral or aortic valves can produce embolic artery occlusions, and prolapse of these valves can lead to thromboembolic occlusions.

- *Think about:* ischemia, TIA/CVA/TMVL.
- *Look for:* ischemic anterior segment findings (neovascularization); retinopathy (micro-aneurysms, intraretinal hemorrhages, cotton-wool spots, neovascularization); vascular occlusions (embolic, thromboembolic); neurologic manifestations (non-arteritic ischemic optic neuropathy (NAION), cranial nerve palsies, TIA/CVA).
- *Educate patients about:* TIA/CVA/TMVL, diplopia, vision/visual field loss.

Right the Record

Unless you work in a multidisciplinary hospital setting or integrated healthcare system, you may not have access to the patient's entire medical record, and you must instead rely on the patient's memory. Unfortunately, patients are often terrible historians. They may know their medications, but not their medical conditions/history—or vise versa. We all have those patients who say they don't have hypertension because it's controlled with medication, right?

Thus, we have to sleuth out the diagnosis based on the medication prescribed, or the likely medication based on the diagnosis. In addition to hypotensives, here are two more examples:

Levothyroxine. Think of underactive thyroid function, which may be due to a host of issues (autoimmunity, thyroiditis, etc.), including iatrogenic causes such as surgical removal of the thyroid or radioactive iodine treatment for hyperthyroidism. In patients on this thyroid hormone replacement with a history of hyperthyroidism/Grave's disease, look for (most commonly) ocular surface exposure and EOM dysfunction and educate your patients about dryness and double vision as possible ocular manifestations of thyroid eye disease.

Anticoagulants. Think AFib (though they are used for other indications as well), look for and educate patients about ocular cardiovascular-associated sequala and education the patient on possible complications of AFib such as stroke, mini-stroke, TMVL or subconjunctival hemorrhages from the drug's use.

Obstructive Sleep Apnea

While this systemic condition certainly carries a higher risk of systemic morbidity and mortality related to hypoxia, it's also associated with ocular morbidity. Eye care providers often think only of floppy eyelid syndrome and dry eyes; however, though less common, several other ocular conditions are associated with obstructive sleep apnea

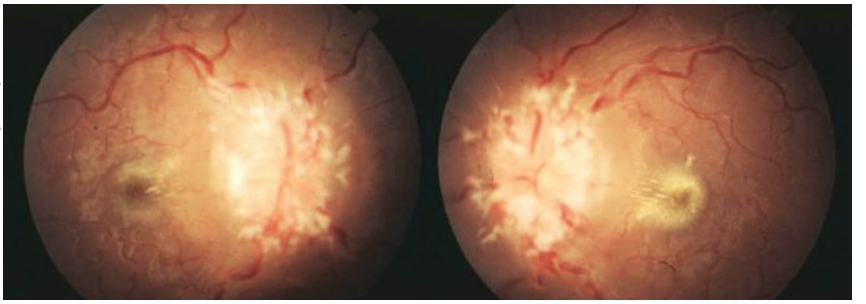


Photo: Brad Sutton, OD

Papilledema may be seen in patients who have sleep apnea.

(OSA), including NAION, primary open angle- and normal-tension glaucoma, retinal vein occlusions (RVOs) and central serous chorioretinopathy (CSCR).[3,4]

While the absolute risk of these ocular conditions is low, the relative risk is higher in OSA patients than it is in the general population.[3] Mechanical changes related to connective tissue weakness and vascular mechanisms related to hypoxia and impaired autoregulation of blood flow are all associated with OSA and these ocular manifestations.[4]

- *Think about:* floppy eyelid syndrome, dry eye, glaucoma, RVOs, CSCR.
- *Look for:* lid laxity, ocular surface/dry eye findings; optic disc edema or pallor; subfoveal fluid; retinal hemorrhaging, venous dilation or occlusion.
- *Educate patients about:* dry eye symptoms, acute vision/visual field loss, metamorphopsia.

Hematologic Disorders

Several conditions that affect the blood can have significant ocular manifestations:

Blood cancers. Leukemia can cause subconjunctival hemorrhages or retinopathy that appears similar to other common types of retinopathy (hypertensive, diabetic, ocular ischemic) with findings such as venous tortuosity and dilation, intraretinal hemorrhages, cotton-wool spots and Roth spots.

Hyperviscosity leading to RVOs is also possible. Ocular findings are present in up to 90% of patients with leukemia.[5]

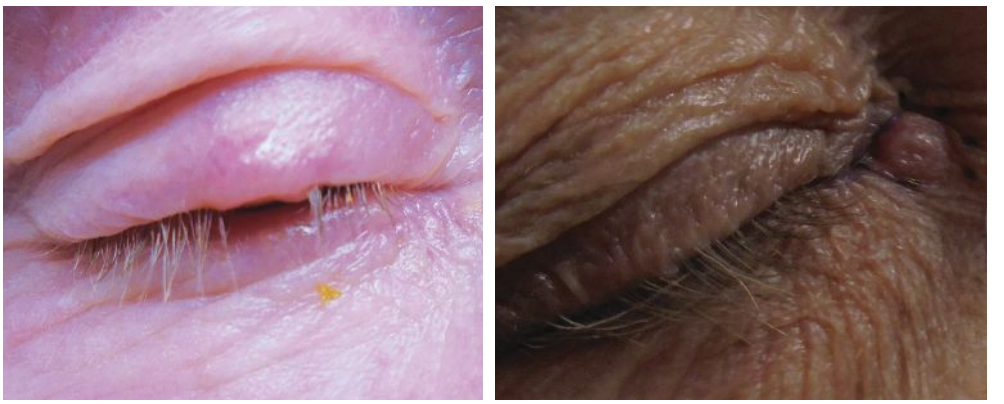
Lymphoma, a remarkable masquerader, can affect any compartment of the eye, including the eyelids, ocular surface, lacrimal gland, any part of the uvea, vitreous, retina or even can infiltrate the optic nerve or orbit.

Monoclonal gammopathies indicate an overproduction of plasma cell immunoglobins. Monoclonal gammopathies of undetermined significance (MGUS) are seen in 3% to 4% of adults older than age 50 and are considered premalignant because they can progress with a 1% annual risk of progression to myeloma.[6] Like lymphomas, MGUS and myeloma can affect nearly any part of the eye either by direct infiltration or related to the consequences of hematologic imbalance (hyperviscosity).

Deep stromal deposits of monoclonal proteins, crystalline keratopathy or copper deposits in Descemet's membrane, maculopathy, vitritis, vasculitis and findings associated with relative hyperviscosity have been reported with MGUS.[7-9] Similarly, myeoma can present as infiltration or deposition in various structures or solid plasmacytomas (plasma cell tumors). As with leukemias, hyperviscosity-related retinopathy can develop, and similar to lymphoma, myelomatous infiltration can occur throughout the eye and related structures.

- *Think about:* infiltration, hyperviscosity, masqueraders.
- *Look for:* lesions anywhere or indication of orbital lesions, deposition or infiltration, uveitis, retinopathy.
- *Educate patients about:* visible lesions in or around the eye, floaters, eye pain, diplopia, TIA/CVA/TMVL (hyperviscosity), visual/visual field changes.

Status-post bone marrow transplant (BMT). Patients may undergo BMT for a variety of hematologic



BCC of the upper eyelid, at left and the medial chanthus/nasal root, at right. Note the lesions' pearly elevated margins.

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Table 1. Common Medical History Findings and Their Associated Ocular Signs/Symptoms

	Ocular surface disease	Diplopia	Visual field loss	Optic neuropathy	Subconjunctival hemorrhages	Transient ischemic attack/ cerebrovascular accident/transient monocular vision loss symptoms	Extraocular muscle dysfunction	Metamorphopsia	Retinal hemorrhages, retinal emboli/thromboemboli	Retinal hemorrhages, cotton wool spots, venous dilation, arteriovenous nicking, Roth spots	Retinal vein occlusion	Glaucoma	Eye pain, redness	Other
Hyperviscosity			x			x			x	x	x			
Obstructive sleep apnea	x			x				x			x	x		Central serous chorioretinopathy
Parkinson's disease	x	x					x					x		Visual hallucinations
Atrial fibrillation, anticoagulants			x		x	x			x					
Cerebral arteriovenous malformations		x	x			x								Headaches, aura-like symptoms
Blood cancers, MGUS		x	x		x	x				x			x	Infiltrates, lesions
Multiple sclerosis		x		x			x						x	
Anemias, sickle cell			x	x	x	x				x				
Thyroid dysfunction	x	x					x							
Plaquenil								x						Toxic maculopathy
Anxiety/depression, antidepressants	x													
Carotid stenosis/occlusion, cardiac valve stenosis/prolapse			x	x		x			x	x				
PDE-5 inhibitors			x	x										Color vision changes
Fingolimod								x						Cystoid macular edema
Amiodarone, ethambutol			x	x										
Skin cancer														Skin lesions
Status-post bone marrow transplant	x												x	
Topiramate													x	Ciliary effusion/angle closure, palinopsia
Levothyroxine	x	x					x							

malignancies. If the patient receives allogenic bone marrow from a donor (rather than allogenic marrow), there is risk for the development of graft-vs.-host disease (GVHD).[10] Patients undergo conditioning to eradicate both tumor and host marrow cells prior to BMT, and this may include total body irradiation; those having had irradiation may also develop cataract.[10] GVHD can present acutely with a hemorrhagic pseudomembranous conjunctivitis, and chronically may appear as a significant autoimmune, Sjögren-like keratoconjunctivitis sicca with lacrimal gland findings, ocular surface and corneal findings and cicatricial scarring.[10]

- *Think about:* GVHD, cataract.
- *Look for:* keratoconjunctivitis sicca, corneal stromal infiltrates, pseudomembranous conjunctivitis, cicatricial changes to eyelids and conjunctiva, cataract.
- *Educate patients about:* Blurry vision, dry eye symptoms.

Anemias. These indicate a reduced level of red blood cells (RBCs) or hemoglobin; while iron deficiency anemia is most common, other forms are possible, such as pernicious anemia (vitamin B12 deficiency), aplastic anemia (inadequate RBC production) or hemolytic anemia (increased RBC destruction).[11] Anemia may lead to bleeding in or around the eye, such

as subconjunctival hemorrhage, and retinopathy similar to that seen in leukemia.[12] Pernicious anemia can lead to optic atrophy, which is seen as disc pallor.

Sickle cell. This genetic disorder causes RBCs to take on a sickle- or crescent-shaped morphology, which can lead to microvascular occlusion and local ischemia. In the eye, this may be seen as non-proliferative or proliferative retinopathy.[11]

- *Think about:* bleeding, ischemia, retinopathy, optic neuropathy.
- *Look for:* hemorrhaging, ischemia, retinopathy, disc pallor.
- *Educate patients about:* abnormal bleeding/bruising, changes in vision.

Skin Cancers

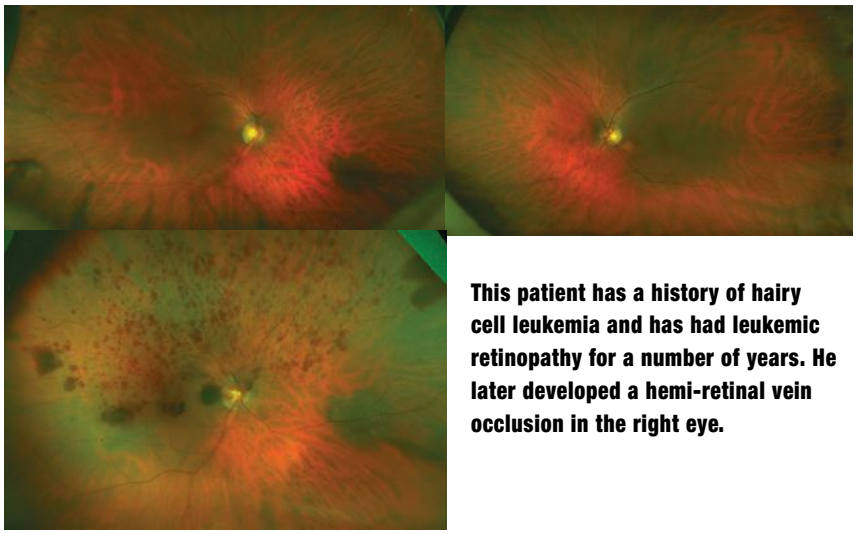
These are common and often will crop up on the head and neck. A patient's history of skin cancer should alert clinicians to screen for new concerning spots. Of all head/neck basal cell carcinomas (BCCs), 20% are periocular.[13] Squamous cell carcinomas (SCC) are second to BCC for eyelid malignancies, and melanoma and other malignancies can also present around the eye.[13] Patients should engage in regular self-monitoring and dermatologic evaluations to screen for potentially cancerous lesions, and optometrists are charged with monitoring not just the periorbital region, but any skin in sight.

- *Think about:* new cancerous or pre-cancerous lesions.
- *Look for:* pearly raised lesions (BCC), raised keratin patches or ulcerated lesions (SCC), irregular, asymmetric pigmented lesions (melanoma) or other suspicious lesions.
- *Educate patients about:* any new, symptomatic (itchy, scaly, etc.) or irregular skin lesions.

Parkinson's Disease

This is known to have many ocular and visual manifestations. Due to motor slowing, incomplete and delayed blink and lagophthalmos are common, along with a reduction in tear secretion, which all lead to dry eyes and exposure keratopathy. Dry eyes are seen in up to 60% of Parkinson's disease (PD) patients.[14] Given the slow progression, it's not uncommon for PD patients to present with significant signs but few symptoms.

Visual hallucinations are a frequent symptom, occurring in



This patient has a history of hairy cell leukemia and has had leukemic retinopathy for a number of years. He later developed a hemi-retinal vein occlusion in the right eye.

16% to 40% of PD patients, and up to 88% of PD patients who have dementia.[15] Visuospatial and visuoperceptual impairment also commonly develop as PD progresses. Clinical experience suggests PD patients by-and-large aren't informed that these things can, and likely will, happen to them, and many don't report these symptoms unless specifically asked. Uncovering these symptoms is important not only because it's treatable—with donepezil, for example, for visual hallucinations, or with neurorehabilitation—but also because it helps predict prognosis.[16]

Abnormal saccades, pursuits and extraocular movements are common, as is diplopia, particularly related to convergence insufficiency.[14] Additionally, research suggests an increased incidence of open-angle glaucoma and non-glaucomatous retinal nerve fiber (RNFL) thinning in PD patients.[14]

Carbidopa-levodopa is a common first-line agent for PD and its presence in the medical history will clue you in to PD as a diagnosis.

- *Think about:* ocular surface disease, extraocular muscle abnormalities, visuospatial/visuoperceptual abnormalities and visual hallucinations, glaucoma and/or non-glaucomatous RNFL thinning.
- *Look for:* lid and blink changes, lagophthalmos, dry

Medications to Watch For

Hydroxychloroquine

- Think about: toxic maculopathy ("Bull's eye maculopathy").
- Look for: parafoveal RPE and outer retinal disruption (parafoveal outer retinal and ellipsoid zone disruptions, central visual field abnormalities, paracentral hyperautofluorescence on imaging, central/paracentral mfERG depressions).
- Educate patients about: metamorphopsia, paracentral visual field changes.

Fingolimod

- Think about: Reports indicate that cystoid macular edema (CME) can develop, usually about three months after initiation, so a baseline evaluation then a three-to-four month follow up are warranted, followed by routine monitoring thereafter. Also think about multiple sclerosis signs such as optic neuritis and EOM dysfunction.
- Look for: CME, EOM dysfunction, optic disc changes.
- Educate patients about: metamorphopsia or vision changes; diplopia, eye pain or acute vision loss.

Topiramate

- Think about: ciliary effusion with secondary angle closure, palianopsia.
- Look for: large myopic shifts, ciliary effusion and secondary angle closure.
- Educate patients about: vision changes, afterimages; urgent return precautions with angle closure symptoms (hazy/foggy vision, eye pain, red eye).

Amiodarone

- Think about: vortex keratopathy (usually inconsequential though can cause irregular astigmatism), amiodarone-associated optic neuropathy, which is similar to NAION but with a more protracted course and generally fewer symptoms.
- Look for: optic disc edema, vortex keratopathy.
- Educate patients about: visual acuity or field changes.

Ethambutol

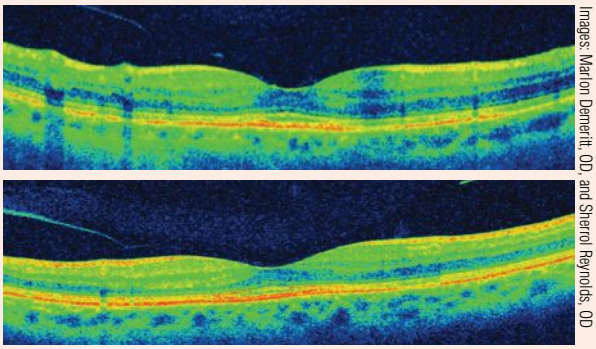
- Think about: Toxic optic neuropathy.
- Look for: optic disc pallor, papillomacular RNFL thinning, central visual field changes.[1]
- Educate patients about: central vision, visual field loss.

PDE5 Inhibitors

- Think about: NAION and color vision changes, co-morbid cardiovascular conditions associated with erectile dysfunction and their possible ocular effects.[2,3]
- Look for: optic disc edema or pallor.
- Educate patients about: short-term color vision changes, vision/visual field loss.[4]

Antidepressants

- Think about: dry eye symptoms.
- Look for: ocular surface disease.
- Educate patients about: dry eye symptoms, mental health care.



Images: Marlon Demeritt, OD, and Sherrol Reynolds, OD

High-resolution OCT demonstrating localized parafoveal thinning in a patient with early Plaquenil toxicity.

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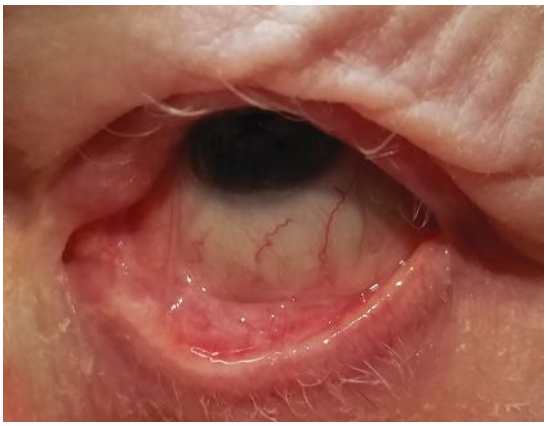
eye and exposure keratopathy, glaucoma, RNFL thinning.

- *Educate patients about:* dry eye symptoms, visuospatial/visuoperceptual symptoms, diplopia, visual hallucinations.



This patient is s/p BMT and has GVHD with symblepharon formation.

Depression and Anxiety

The link between the eye and these mental health concerns is not as obvious, but several studies have identified an association between depression, anxiety and antidepressant use and an increase in the prevalence of dry eye disease and subjective dry eye symptoms.[17,18] While objective findings are often lacking, these patients can be quite symptomatic. In addition to treating their ocular symptoms with dry eye therapies, be sure to support and encourage mental health care.

- *Think about:* dry eye symptoms, which often outweigh signs.
- *Look for:* ocular surface disease.
- *Educate your patient about:* dry eye symptoms, appropriate mental health care.

The conditions reviewed here are just a small sampling of the many things we see in our patient's medical record that should get our optometry wheels turning. Almost any systemic revelation can connect back to the eyes in some way. It's important that we embrace whole-body care to help our patients stay healthy and happy. ■

Dr. Weidmayer practices at the VA Ann Arbor Healthcare System in Ann Arbor, MI.

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Diagnostic Skills and Techniques

The Dry Eye Assessment: Start With the Lids

The real root cause of most dry eye is failure of the eyelids to protect the ocular surface. Here's how you know. **By Amy Nau, OD**

We have all experienced the frustration of caring for a dry eye patient who continues to be symptomatic despite maximal therapy. Before we toss in the towel, we must question if we are missing the underlying condition. This article suggests a new way to assess patients who still struggle with symptoms, and it all begins with a closer look at their eyelid function—and dysfunction.

In the setting of a compliant patient without severe structural damage, if a treatment is failing, it might not be because the recommended therapy, medication or device does not work. Rather, it might be a deficiency in how we are defining the patient's condition. The new definition set forth by the Tear Film and Ocular Surface Society's Dry Eye Workshop II is by far the most comprehensive one to date: "Dry eye is a multifactorial disease of the tears and ocular surface that results in symptoms of discomfort, visual disturbance, and tear film instability with potential damage to the ocular surface. It is accompanied by increased osmolarity of the tear film and inflammation of the ocular surface."[1]

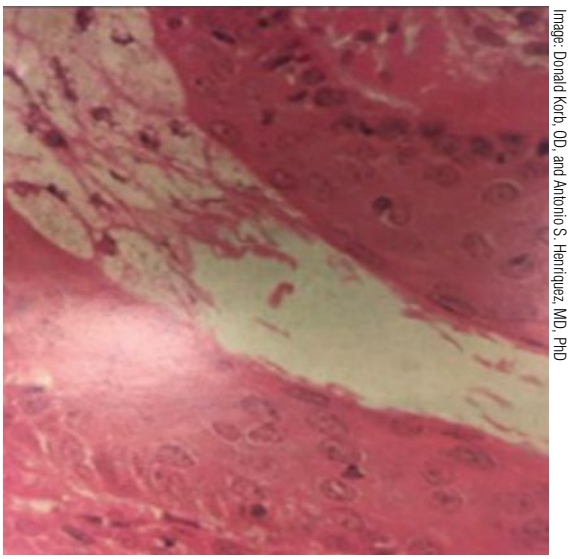

Image: Donald Korb, OD, and Antonio S. Henriquez, MD, PhD

Keratinized epithelium sheds from the lining of the terminal duct of the meibomian gland before the orifice. This creates a "spiderweb" that can trap meibum as it exits the gland if the force of the blink is insufficient to expel meibum.[11]

Still, none of the recent definitions for dry eye, including this one, emphasize the crucial role of the eyelids—when they do not function properly, the ocular surface is compromised. The mechanisms by which the eyelids fail to protect the eye are often ignored, and thus otherwise completely reasonable treatments do not improve signs or symptoms of dryness.

Take Cover

The eyelids are the primary protectors of the tear film, corneal nerves and conjunctiva. The fully closed eyelid protects and restores the cornea and conjunctiva during sleep, while the blink reflex and cilia protect the eye from harm and foreign objects. The eyelids contain the meibomian glands and some of the accessory lacrimal glands critical for tear film stability. Proper lid closure, innervation and

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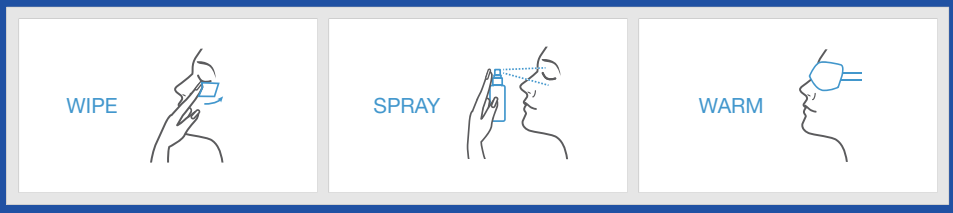

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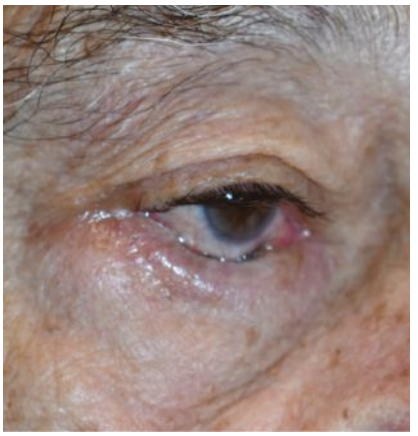

This patient's involutional entropion is pushing their lashes into conjunctiva, compromising the ocular surface.

Health, Compromised

Structural eyelid dysfunction isn't the only way the palpebral conjunctiva can become inflamed. Bacterial invasion causing meibomitis, airborne pollutants, *Demodex*, ocular rosacea and chronic exposure to allergens, pharmacological toxins, some cosmetics and other chemical exposures, are all possible causes.[1-4] In addition, mechanical stressors such as eye rubbing and contact lenses can cause inflammation.[5,6] Less commonly seen but well recognized causes of chronic and often severe inflammation include ocular graft-vs.-host disease, Stevens Johnson syndrome and chemical or thermal burns. Controlling any of these other factors is crucial before dry eye disease can be cured instead of just controlled.

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blink mechanisms are designed to produce, mix and spread the tear components across the ocular surface and guide the egress of tear film and debris through the nasolacrimal apparatus.

When the lids fail to do their job, a host of sequelae ensue, including inflammation and symptoms consistent with dry eye disease. Staring or partial blinking, as occurs during prolonged computer use, leads to evaporative (desiccating) stress. Research shows this initiates a dry eye cascade in a murine model exposed to scopolamine and drafty environments with low humidity.[2] The study suggests that under these conditions, an immune-mediated stress response occurs within six hours in conjunctival and corneal epithelial cells that leads to activation of natural killer (NK) cells. NK cells produce IFN-γ that signals upregulation of Th1-associated chemokines, which then recruit and activate other inflammatory cells.[2]

Because desiccating stress is one of the seminal events in the inflammatory cascade, our dry eye workup should evaluate whether the patient's eyelids are promoting tear film stability. Two critical questions to ask a patient is how many hours per day they use a screen and if their eyes feel dry when they wake up. If they are staring all day and/or not closing their eyes completely overnight, the tears will evaporate and inflammation is inevitable.

Meibomian gland dysfunction (MGD) is the most common way the eyelids can fail to do their job. MGD is caused by obstruction and poor blinking.

Obstruction. Epidemiologic evidence shows that aqueous and goblet cell deficiencies are less common than MGD.[3] The excitement surrounding meibomian gland rehabilitation as a potential silver bullet for dry eye therapy is evidenced by a host of new methods for rehabilitating the glands. Many clinicians now race to treat MGD as the root cause of dry eye but fail to understand why MGD starts in the first place. Obstruction from the inner aspect of the gland occurs because the keratinized epithelium in the terminal duct accumulates and then traps escaping meibum when the blink is not fully executed.

Obstruction from the outer aspect of the gland occurs when desquamated epithelium and biofilms cause inflammation of the lid margin, again because a partial blink does not naturally exfoliate the lid margin surfaces. These dead cells and biofilms eventually cause inflammation of the tissue.[4]

Both inner and outer obstruction is caused by partial or infrequent blinking, both of which often result from staring at computers, tablets and smartphones.

Dry Eye Etiologies

Primary Lid-related	Non-lid related
• Blink concerns (staring, often at digital devices)	• Immune (juvenile rheumatoid arthritis, Sjögren's syndrome, Graft-vs.-host disease)
• Lid seal and nocturnal lagophthalmos (20% of cases)	• Medications
• Aging (ectropion, entropoin, etc.)	• Hormones
• Ocular/lid surgery	• Contact lenses (controversial)
• Demodex	• Cosmetics
• Lack of hygiene	• Environmental and vocational
• Other lid abnormalities	• Diet
	• Smoking

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Dry Eye Assessment

Who	What	When	Why
Ophthalmic Technician	Comprehensive history form	Initial visit	To document the chronicity, past treatments and failures, severity.
	Dry eye questionnaire	Every visit	Keep the same survey from visit to visit to determine improvement.
	Meibography	Initial visit/yearly	Determine gland dilation and atrophy.
	Lipid layer thickness/blink assessment	Initial visit/follow-up	Determine meibomian gland function and blink dynamics.
	Osmolarity	Initial visit/follow-up until consistently negative	Tests sequelae of unstable tear film.
	Matrix metalloproteinase-9	Initial visit/follow-up until consistently negative.	Tests sequelae of unstable tear film.
	Evaporative stress test	Follow-up visit	When corneal hyperalgesia or neuropathic pain is suspected.
	Schirmer's test or dry eye test	Initial and occasional follow-up visits	To test for lacrimal gland function.
Doctor	Review history	Initial visit and follow-up visits	To understand the chronicity and severity, review amount of screen time, medications, cosmetic injections, contact lens use, allergies, cosmetic use.
	Review current dry eye regimen	Every visit	To make sure patient is following instructions.
	Observe blink	Every visit	How are the lids failing to protect the ocular surface? Does it match staining patterns?
	Gross eyelid assessment	Every visit	How are the lids failing to protect the ocular surface? Look for palpebral conjunctival and lid margin inflammation. May match staining patterns.
	Transillumination	First visit/yearly	If you don't have a meibographer.
	Vital dye staining (fluorescein, lissamine green, rose bengal)	Every visit	Helps assess line of Marx, cornea, exposure zone, superior limbic keratoconjunctivitis, lid wiper epitheliopathy, tear break-up time, tear lake assessment, tear drainage.
	Meibomian gland evaluator	Every visit	Determines meibomian gland function.
	KB light test	First visit	Determines whether eyelids are fully closed when sleeping.
	Reflex tearing/*tear lake*	Every visit	Can be affected by dehydration and medications as well as secondary aqueous deficiency from evaporation.

Tests that measure sequela are in italics.

Research shows smartphone use in particular can increase symptoms of dry eye.[5]

Lid closure. Complete eyelid closure is required for the meibomian glands to expel their contents, for the tear film to mix and for tears to spread and be removed from the eye. Without proper eyelid closure, the meibomian glands do not produce meibum and the aqueous components evaporate. The solid components of the aqueous phase become more concentrated, resulting in hyperosmolarity. Thus begins the cycle of inflammation and pain.

Myriad conditions can thwart the lids' ability to adequately protect the ocular surface, including nocturnal lagophthalmos and poor lid seal, age-related lid laxity, floppy eyelid syndrome, senile ectropion or entropion, blepharoplasty, Botox injections, thyroid eye disease, paralysis, high myopia, facial trauma and lid and craniofacial abnormalities.

Infrequent and/or partial blinks represent a primary failure of the lids to adequately protect the ocular surface.[6] When incomplete blinks occur over a period of hours, the ocular surface, subjected to chronic desiccating stress, becomes compromised.

Desiccating stressors are extremely prominent in modern society, mostly due to the ubiquity of digital screen use. Blink rate decreases during screen use,

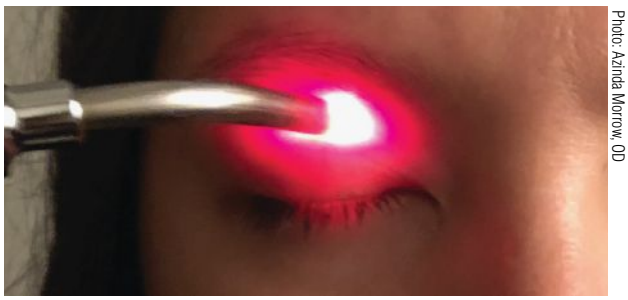

Photo: Azinda Morrow, OD

The Korb-Blackie test indicates incomplete eyelid closure when light is seen emanating from the lid margin of the eye.

and research shows incomplete blinks occur when looking at screens and reading. The tear film integrity should be sustained longer than the interblink interval; otherwise, the corneal nerves are exposed between blinks causing irritation and, ultimately, hyperalgesia.

Patients with poor blinking habits will invariably develop secondary MGD and exhibit inferior corneal staining and conjunctival injection nasally and temporally in the "exposure zone." Even patients with non-obvious MGD but significant dry eye symptoms can harbor increased numbers of inflammatory cells in the proximal tissue and conjunctiva.[7] This suggests that some of the dry eye symptoms may actually stem from the palpebral conjunctiva of the eyelids.

Any ongoing office and at-home treatments for meibomian gland health and ocular surface inflammation will have marginal and short-acting effects unless these root causes of dry eye disease are addressed first.

No Rest for the Weary

Exposure keratoconjunctivitis both during the day and at night is a problem for patients who have had aggressive lid surgery, paralysis, trauma, thyroid eye disease or other lid abnormalities. In this instance, the patient likely has incomplete habitual blinks during the day and nocturnal lagophthalmos—never providing the eye with a period of recovery. Some patients will experience corneal erosions and filamentary keratitis. Because the corneal nociceptors can become chronically irritated and hypersensitive to subthreshold stimuli, these cases are at risk for hyperalgesia and corneal neuropathic pain.[1] If this is suspected, clinicians can place a patient in a swim goggle for about 30 minutes or instill a drop of proparacaine to determine whether these conditions are present. The corneal nerves must be protected from desiccating stress at all costs for patients with exposure keratoconjunctivitis, and all other comorbid conditions need to be addressed.

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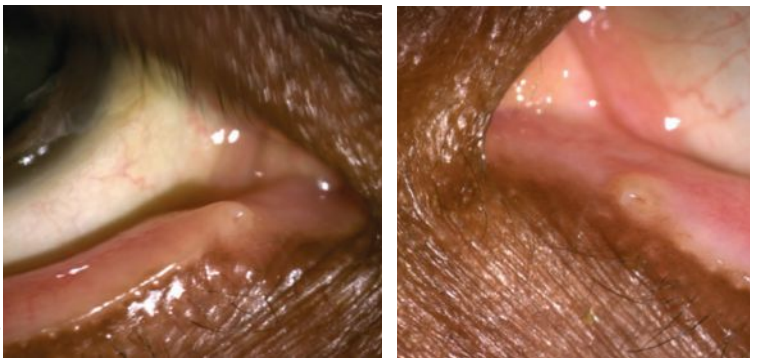


Photos: Marlon Dermeritt, OD, and Beata Lewandowska, OD

This patient displays punctal stenosis in both eyes.

Assessment, Step-by-step

Current recommendations for dry eye evaluation focus on longitudinal, quantitative metrics captured by questionnaires, vital dye staining, tear film and meibomian gland imaging and testing for inflammation, tear production and osmolarity. While these practice patterns are incredibly useful, they do not stress the importance of eyelid health. Thus, clinicians should re-evaluate their dry eye protocol to ensure it also incorporates careful lid assessment. This includes a determination of every reason that the lids may be failing to protect the surface of the eye by external and slit lamp evaluation using standard techniques.

Rooting out the multifactorial, primary causes of dry eye begins with a thorough case history, including systemic conditions, medications, prior successes and failures with dry eye treatments, use of contact lenses, screen time, exposure to pollutants and cosmetics and any prior lid surgeries or procedures (patients do not often think of cosmetic lid surgery as being relevant). Clinicians should review this comprehensive history at least annually and pair it with a list of all available interventions.

As part of the case history, it is vital to inquire when the patient experiences their dry eye symptoms. For example, if symptoms occur at the end of the day, clinicians should ask about how the patient is spending their time. The average American adult spends 11 hours per day in front of some type of screen, inevitably compromising their blink.[8]

If a patient reports that dry eye symptoms occur upon arising or when they get up in the middle of the night, it's likely that they have nocturnal lagophthalmos and the eyes are not sealed shut. In theory, Bell's phenomena with its upward movement of the globe during sleep should protect the cornea. However, in one study, fewer than half of the study population exhibited the expected upward rotation.[9]

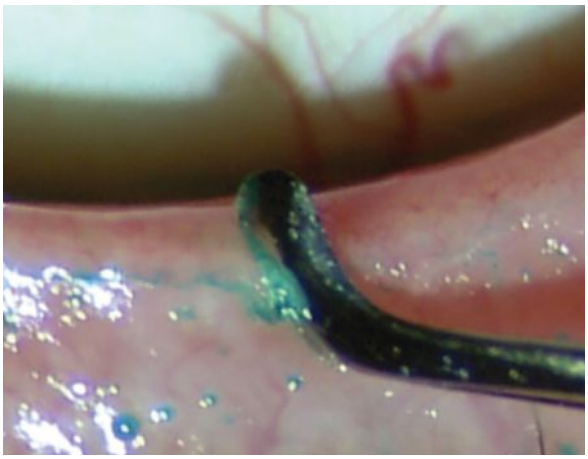
Testing for nocturnal lagophthalmos and poor lid seal is readily accomplished using the Korb-Blackie Light Test in which a Finoff transilluminator is applied to the superior sulcus over the closed eyelid in a darkened room.[10] The test is positive if light leaks down to the cheek and the patient reports morning symptoms. Some of these patients will manifest inferior corneal staining. In some cases the condition is unilateral, which could explain why they are more symptomatic in one eye. If the patient is having chronic exposure for six to eight hours every night, inflammation eventually occurs, which is further exacerbated by daytime

Slit Lamp Exam Considerations

MGD	Other Lid Concerns
Non-obvious: • Extremely common in teens and young adults • Lids look normal but glands are obstructed *Obvious - All sequelae/entrenched:* • Vascularization/telangiectasia • Lid thickening/serrated margins • Lid margin erythema • Gland blockage • Gland capping • Compromised lashes/follicles • Anterior progression of line of Marx	• Rosacea • Dermatitis • Acne (previous acne scarring) • Lacrimal drainage systems • Incomplete eyelid closure (Grave's disease, high myopia, etc.) • Ectropion/entropion • Trichiasis • *Demodex* • Punctal stenosis • Blepharitis • Nocturnal lagophthalmos (prominent globe) • Previous lid or brow surgery • Dermatochalasis

Any lid abnormality can result in failure to protect the ocular surface.

Image: Donald Korb, OD, and Antonio S. Henriquez, MD, PhD



Dead skin and biofilm can accumulate on the lid margin surface, causing external orifice obstruction.

proinflammatory activities such as contact lens wear, cosmetics and digital device use.

When evaluating the primary causes of dry eye, look to the definitions that help guide our management, but do not neglect the eyelids, which are the true guardians of the ocular surface. They play a pivotal role in protecting the eye from desiccating stress. Clincians should fully educate patients regarding the nature of their dry eye and overall prognosis for improvement. Some patients will not get better because of structural changes, and some patients may take a very long time to recover. Treating problems of eyelid function and structure are key to improving the signs and symptoms of dry eye disease. ■

Dr. Nau practices at Korb & Associates with a focus on medically necessary contact lenses, anterior segment disease and dry eye.

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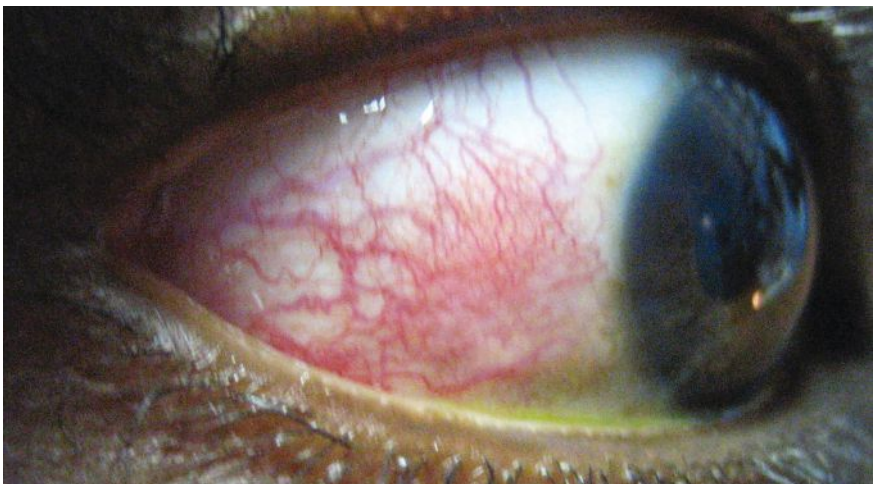
The Conjunctivitis Conundrum

"Pink eyes" are usually a sign of underlying infection or allergy. Here's how to get to the bottom of it to quickly resolve the issue. By Ernie Bowling, OD

Conjunctivitis is a general term that can refer to any in a spectrum of diseases and disorders that primarily affect the conjunctiva. Patients impacted will present complaining of redness or "pink eye" which is due to dilated conjunctival blood vessels.[1] They may also complain of pain, itching or discharge. While most cases are self-limiting and rarely result in vision loss, some may progress and can result in serious ocular complications, and vision loss is not out of the question. For this reason, it's essential to rule out the sight-threatening causes of red eye.



This patient demonstrates episcleritis, a presentation associated with complaints of discomfort or irritation (rather than true eye pain), redness and edema to the affected area over the sclera.

Additionally, conjunctivitis can stem from different roots, and distinguishing them can help identify treatment options. Conjunctivitis can affect people of any age, demographic group or socioeconomic status.[2] We can broadly categorize it as either acute or chronic and either infectious or non-infectious, but within those exist several subclassifications.[2]

This article provides an in-depth explanation of all the conjunctivitis types you're likely to encounter—and some that are quite rare. We review the protocol for differential diagnosis and managing each of the possible conjunctivitis cases.[2]

Etiology and Epidemiology

Conjunctivitis is the most common cause of eye redness and discharge.[2] The three most common types are viral, allergic and bacterial.[2] Allergens, toxins and local irritants are responsible for non-infectious conjunctivitis.[2]

Acute conjunctivitis of all causes is estimated to occur in six million Americans annually.[3] The highest rates are among children younger than seven years with the highest incidence occurring between birth and four years.[2] Another peak occurs in women at age 22 and men at age 28.[4] Overall conjunctivitis rates are slightly higher in women than men.[4] Peak seasonal incidence occurs in

children in March and in other age groups in May, and this seasonal occurrence is consistent in all geographic regions regardless of changes in climate or weather patterns.[4] Allergic conjunctivitis is the most frequent cause, affecting 15% to 40% of the population, and is seen most often in the spring and summer. Acute bacterial conjunctivitis is second most common and its rates are highest from December to April.[1,4-7]

Viral

Viruses cause up to 80% of all cases of acute conjunctivitis, with many cases misdiagnosed as bacterial conjunctivitis.[8,9] Between 65% and 90% of viral conjunctivitis (VC) are caused by adenoviruses and they produce the three most common presentations associated with VC; follicular conjunctivitis, pharyngoconjunctival fever and epidemic keratoconjunctivitis.[9,10]

Follicular conjunctivitis is the mildest form of a viral conjunctival infection. It has an acute onset, initially unilateral with the second eye becoming involved in a week. It presents with a watery discharge, conjunctival redness, follicular reaction and a preauricular lymphadenopathy on the affected side. Most cases resolve spontaneously.[10]

Pharyngoconjunctival fever is characterized by a high fever that comes on suddenly as well as a sore throat, periauricular lymph node enlargement and a bilateral conjunctivitis.

Epidemic keratoconjunctivitis, the more severe of the two, presents with an ipsilateral lymphadenopathy, conjunctival redness, swelling and watery discharge.[11] Lymphadenopathy is seen in up to 50% of VC cases and is more prevalent in VC than bacterial conjunctivitis.[9]

Labs and cultures are rarely necessary to confirm bacterial conjunctivitis and is generally reserved for severe or recalcitrant cases.[12] In-office rapid antigen testing is available for adenoviruses and can be used to confirm suspected viral causes of conjunctivitis to prevent unnecessary antibiotic use. The Quidel QuickVue (Quidel) adenoviral conjunctivitis test is a rapid, immunochromatographic test for visual, qualitative *in vitro* detection of adenoviral antigens directly from ocular fluid. A study comparing rapid antigen testing with polymerase chain reaction and viral culture and confirmatory immunofluorescent staining found rapid antigen testing to have an 89% sensitivity and 94% specificity.[13]

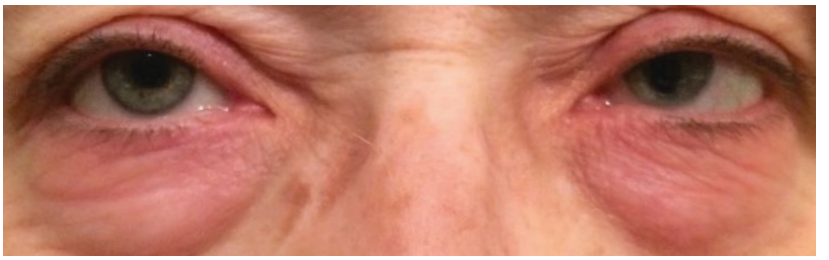
Adenoviral conjunctivitis is highly contagious, with the risk of transmission approximately 50%.[14,15] The infection is often termed epidemic keratoconjunctivitis due to the adenovirus' ability to rapidly infect family members, classmates or co-workers. The virus spreads through direct contact with fingers, swimming pool water and personal items and can be spread for up to 14 days.[16-18] With such high transmission rates, hand washing is imperative. One study found 46% of infected individuals had positive cultures grown from swabs of their hands.[18] Patients with possible adenoviral infection should be isolated from other patients in the office and all instruments and surfaces must be disinfected after potential exposure.[19]

Rapid antigen testing, using devices such as this, can prevent unnecessary antibiotic use. Studies show they have high rates of sensitivity and specificity.

While no effective treatment for VC exists yet, supportive measures to help with symptoms include artificial tears, topical antihistamines and cold compresses.[12] Available antiviral medications are not useful and topical antibiotics are not indicated.[20,21] Povidone iodine—a broad spectrum antimicrobial with high microbial kill rates—can be used in a 5% ophthalmic preparation off-label for management of adenoviral conjunctivitis.[22] In fact, a topical ophthalmic suspension of povidone iodine 0.6% and dexamethasone 0.1% is under clinical investigation.[23] This medication has the potential to treat both the viral and inflammatory components of adenoviral infection as well as immune-related sequelae such as subepithelial infiltrates.[23]

Bacterial

While viral conjunctivitis is more common, bacterial conjunctivitis (BC) can be more of a clinical challenge. It's the second most commonly occurring infectious cause in a conjunctivitis presentation.[5,17] BC is far more common in children than adults, and the pathogens responsible for BC vary depending on the age group. The most common cause of BC in children is *Haemophilus influenzae*, followed by *Streptococcus pneumoniae* and *Moraxella catarrhalis*.[24] Bacterial infection in adults are more often staphylococcal in origin, with *Staphylococcus aureus* more commonly found in adults, with an increase in conjunctivitis secondary to methicillin-resistant *Staphylococcus aureus* (MRSA).[7] Gram-negative infections are more prevalent in contact lens wearers, with *Pseudomonas aeruginosa* the most common cause in this group.[25] *Pseudomonas* is also the most likely



This patient is experiencing an allergic reactions on and around the eyelids as well as the conjunctiva. Upon allergen exposure the skin becomes red, tight and itchy.

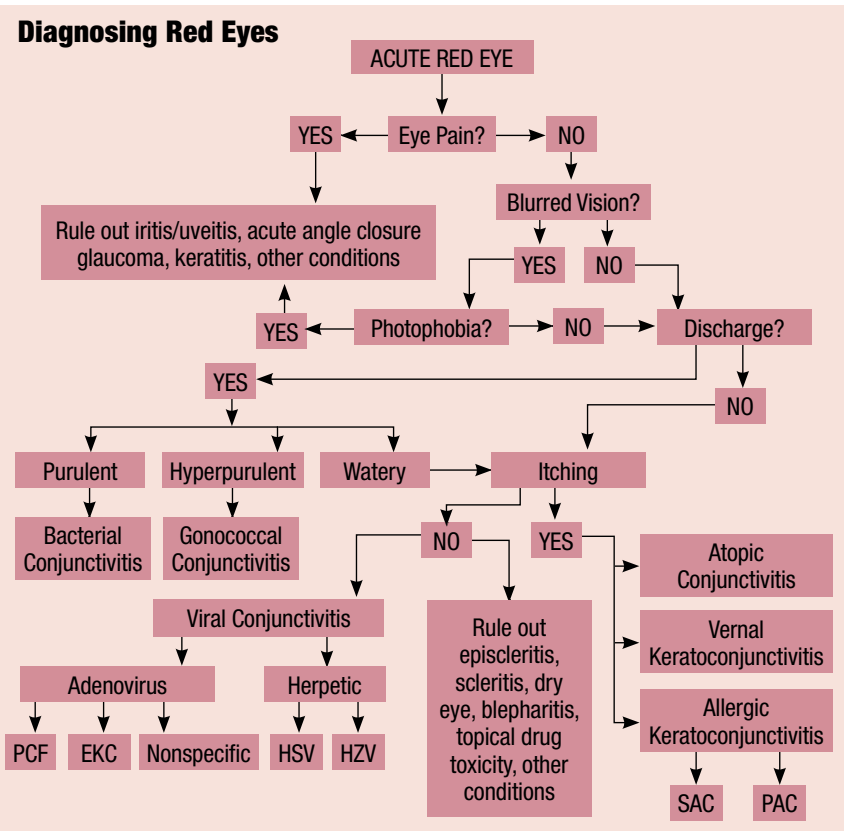
cause of BC in the critically ill and hospitalized patient.[7] Acute BC in newborns is typically the result of *Neisseria gonorrhoeae* and *Chlamydia trachomatis*.[1]

BC can be divided into three clinical presentations: acute, hyperacute and chronic.[10] The most common pathogens are the aforementioned *Staphylococcus aureus*, *Streptococcus pneumoniae* and *Haemophilus influenzae*.[10]

Signs and symptoms of acute BC include the rapid unilateral onset of a red eye, purulent or mucopurulent discharge, and conjunctival edema. The second eye typically becomes involved one or two days later.[10] Bilateral eyelid mattering and eyelids sticking together, a lack of itching, and no history of prior conjunctivitis exposure are strong positive predictors of acute BC.[26] Acute BC treatment consists of topical antibiotic drops or ointments. While BC infections are normally self-limiting within one to two weeks of presentation, antibiotic therapy speeds resolution and lessens disease severity.[17] A broad spectrum antibiotic may be used for five to seven days. No clinical evidence suggests one antibiotic is better than another.[12]

Hyperacute BC is most often caused by *Neisseria gonorrhoeae*.[27] The disease presents with a severe copious purulent discharge, decreased vision, often eyelid swelling, eye pain on palpation and preauricular adenopathy. The infection carries a high risk if corneal involvement and subsequent corneal perforation.[1] Conjunctival cultures are strongly recommended in this presentation. The treatment regimen for gonococcal conjunctivitis includes one gram of intramuscular ceftriaxone. If a corneal ulcer is present, hospitalization with one gram of IV ceftriaxone for three days is recommended.[10]

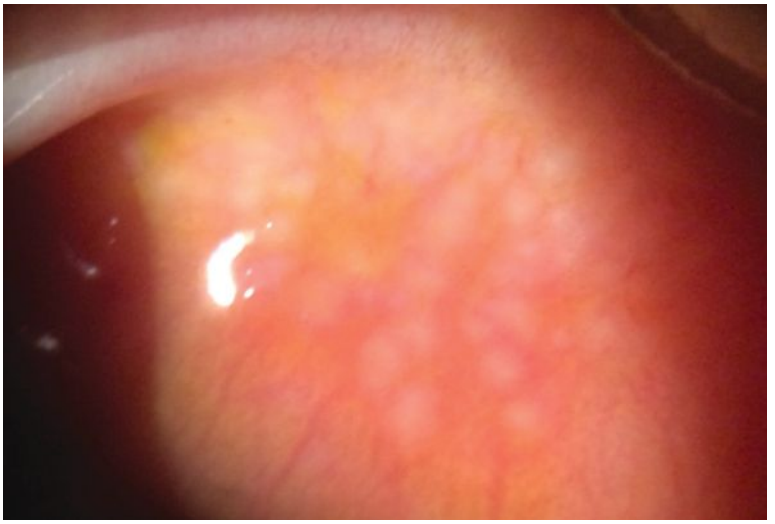
Chronic bacterial conjunctivitis is used to describe any conjunctivitis lasting more than three weeks, with *Staphylococcus aureus*, *Moraxella lacunata* and enteric bacteria being the most common culprits.[10] A chronic staphylococcal conjunctivitis may display signs including a diffuse conjunctival redness with minimal discharge. Papillae or follicles may be present as well as eyelid involvement which may show redness, lash loss, dilated small blood vessels, lid collarettes, recurrent hordeola and may lead to marginal corneal ulcers. The treatment of chronic BC includes antimicrobial therapy and good lid hygiene. Azithromycin drops, erythromycin and bacitracin ointments are effective topical antibiotics. Combination antibiotic/steroid drops or ointments can be rubbed into the lid margins if severe inflammation is present.[10] Oral tetracycline-class antibiotics may be needed for more severe presentations.[10]



This flowchart for the differential diagnosis of a red eye, adapted from various sources, explains an orderly approach to treating these irritated patients.[61,62]

Allergic

Ocular allergy is a common condition seen in clinical practice. Allergic disease have dramatically increased in the last decades, with the increase considered to be caused by a number of factors including genetics, increased air pollution in urban areas, pets and early childhood exposure.[28-30] Allergic conjunctivitis is a general term encompassing seasonal allergic conjunctivitis (SAC), perennial allergic conjunctivitis (PAC), vernal keratoconjunctivitis (VKC) and atopic conjunctivitis (AKC). Contact lenses or ocular prosthesis associated giant papillary conjunctivitis (GPC) are often included in this group, yet GPC is not a true allergic disease but a more chronic ocular micro-trauma disorder.[31]



Giant papilliary conjunctivitis causes this inflammation characterized by papillary hypertrophy of the superior tarsal conjunctiva.

SAC and PAC are the most common forms of ocular allergies, affecting 15% to 20% of the population.[29] Specific IgE antibodies can be documented in almost all cases of SAC and PAC.[30] The pathogenesis of allergic conjunctivitis is by and large an IgE-mediated hypersensitivity reaction, where allergens interact with IgE bound to sensitized mast cells, causing degranulation. This mast cell degranulation causes increased levels of histamine, prostaglandins and leukotrienes and also induces activation of vascular endothelial cells, which in turn express chemokines and adhesion molecules. This early-phase reaction clinically lasts 20 to 30 minutes.[32] The chemokine release initiates recruitment of inflammatory cells into the conjunctival mucosa, which leads to the late phase allergic reaction, characterized by infiltration of inflammatory cells, occurring a few hours after the initial mast cell activation.[33]

Signs and symptoms are the same in SAC or PAC. SAC is usually caused by airborne pollens occurring in the spring and summer and wane in the winter. PAC occurs throughout the year with exposure to allergens to which the patient is sensitive. Clinical features of both SAC and PAC consist of itching, redness and swelling. Itching is a consistent symptom of both SAC and PAC. An old saying applies here: "If it itches, it's allergy." Fortunately, corneal involvement is rare.

Contact allergic reactions usually occur on the skin including the eyelids, but the conjunctiva can also see contact allergic reactions. Upon allergen exposure the skin becomes red, tight and itchy. Treatment includes avoiding contact with the offending agent. Topical and oral antihistamines, topical steroids and cold compresses can help ease the symptoms.

Treatment Options for Allergic Conjunctivitis

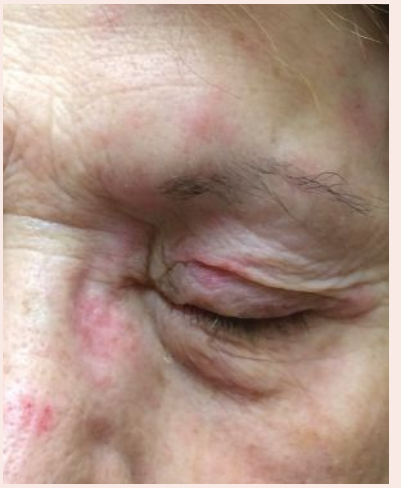
The first treatment for all types of AC is to avoid the offending allergen if possible, which is difficult if the agent is not readily known. Artificial tears provide a barrier between the offending agent and the conjunctiva, and help dilute and wash away the allergen from the ocular surface.

Keeping the tears refrigerated also provides soothing relief, as do cold compresses. Pharmacologic therapy consists of anti-allergic agents such as antihistamines, mast cell stabilizers and dual-action medications. Due to their short duration of action topical antihistamines require frequent dosing, up to four times daily. Combination antihistamine/decongestants are more effective than antihistamines alone.[34] Decongestants are primarily vasoconstrictors and while effective in reducing redness should only be used for short-term relief and are not recommended for use in narrow angle glaucoma patients. Mast cell stabilizers do not relieve existing symptoms and are used prophylactically to prevent mast cell degranulation.

The most commonly used medications for ocular allergy therapy are the multi-action agents that exert multiple pharmacological effects. These include olopatadine, ketotifen, azelastine, epinastine and bepostatine. These medications are the drugs of choice for providing quick symptomatic relief to patients suffering from AC. When these medications do not yield the control desired, the next step are anti-inflammatory agents. Non-steroidal anti-inflammatory drugs (NSAIDs) can be added to reduce symptoms.

Herpetic Conjunctivitis

If your patient's conjunctivitis isn't related to the adenovirus, it may one of the nearly 50,000 new or recurring cases of ocular herpes simplex virus (HSV) related conjunctivitis diagnosed in the United States every year.[1] HSV comprises 1.3% to 4.8% of all acute conjunctivitis cases.[2] You can begin to distinguish it from adenoviral conjunctivitis because HSV is almost always unilateral, whereas adenoviral is usually bilateral.[2] In HSV, follicular conjunctivitis, watery discharge and vesicular eyelid lesions may be present.



This patient displays herpes zoster virus of the forehead with ocular involvement. The eye is involved in approximately 75% of cases when the nasociliary nerve is affected.

Unlike adenoviral conjunctivitis treatment, topical and oral antivirals are used to shorten HSV disease duration.[3] Topical treatments include trifluridine one percent one drop every two hours, reduced to five time per day after three to seven days, or ganciclovir 0.15% gel five times a day. Oral treatments include acyclovir 400mg three to five times a day in adults, valcyclovir 500mg three times a day, or famciclovir 250mg three times a day.[1] Avoid topical steroid use in HSV, as they enhance the virus and can cause harm.[4]

The virus responsible for shingles, herpes zoster (HZV), is a reactivation of a varicella zoster (chickenpox) infection and can invade ocular tissues, with the eyelids most commonly involved followed by the conjunctiva.[5] HZV of the forehead involves the eye in approximately 75% of the cases when the nasociliary nerve is affected.[6]

Patients presenting with eyelid involvement, or those presenting with vesicles at the end of the nose (the Hutchinson sign), should be examined carefully as HZV is almost always accompanied by ocular involvement. While the Hutchinson sign is a biomarker, it is neither sensitive nor specific, and ocular involvement can occur even if the Hutchinson sign is absent.[7] Treatment usually consists of oral antivirals and topical steroids. Topical antivirals have no role in the treatment of HZV. Oral acyclovir can be used, 800mg five times a day, for seven to 14 days. Valacyclovir and famciclovir can also be used. For the treatment of HZV, a good rule of thumb is to double the doses of the medications used for HSV keratitis.[7] Oral antivirals started within 72 hours of symptom onset can reduce disease severity and long-term complications.[6]

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Steroids are the most potent medications used in AC and are effective in treating both acute and chronic presentations.[35] Yet as with any medication there are limitations with steroid use, including delayed wound healing, secondary infection, elevated intraocular pressure, and cataract formation. Due to these potential adverse events, a short course of steroid therapy is appropriate. Baseline intraocular lens evaluation and IOP measurements should be taken if an extended steroid course is needed.[36]

Immunotherapy can be effective in treating the ocular symptoms of AC and may be considered for long term AC control.[37] Oral antihistamines are commonly used to treat nasal and ocular allergy symptoms. The newer second generation antihistamines (cetirizine, fexofenadine, loratadine) are preferred as they produce fewer side effects, especially less drowsiness, but they can produce ocular dryness which may actually worsen ocular allergy symptoms, and researchers suggest the concomitant use of topical drops may more effectively treat ocular allergy symptoms.[38,39]

Vernal

VKC is a disease of warm climates (more common in the tropics) and warm months, but it is not unusual to see the occasional VKC patient in North America.[40] Mostly children and young adults, typically males, with a history of atopy are affected.[41] Symptoms include ocular itching (which may be quite intense), redness, swelling and discharge. Patients may often have photophobia. The most characteristic signs are giant, "cobblestone" papillae on the upper tarsal conjunctiva, easily seen on lid eversion, with mucous discharge.[42]

The cornea may be affected in VKC. Trantas dots (clumps of necrotic eosinophils) may appear at the limbus when VKC is active and wane when symptoms subside.[33] Noninfectious shield ulcers can present in the superior cornea. Corneal epithelial punctate keratitis may be present and can evolve into corneal macroerosions, ulcers and plaques.[28]

Atopic

AKC is a bilateral chronic inflammatory disease of the eyelids and ocular surface and the "ocular counterpart" of atopic dermatitis or atopic eczema.[42] Ocular findings can include mild to severe conjunctival injection and swelling. Giant papillae and Trantas dots may or may not be present, but conjunctival scarring is common. AKC patients may also develop atopic cataracts and it is not unusual for AKC patients to undergo cataract surgery at a young age.[43] Both VKC and AKC may present with giant papillae and Trantas dots. VKC resolves by age 20, while AKC can persist throughout life.[33]

Giant papillary conjunctivitis

GPC is a conjunctival inflammation characterized by papillary hypertrophy of the superior tarsal conjunctiva similar to VKC but without corneal involvement.[33] GPC may be caused by limbal sutures, ocular prostheses and contact lenses.[44] Hence, GPC is not a true allergic disease, as the impetus for the papillary conjunctival changes are inert material and not allergens.

Episcleritis

Any discussion of the red eye must include episcleritis. This is an acute unilateral or bilateral inflammation of the episclera, the thin layer of loose connective tissue between the conjunctiva and the sclera. Episcleritis can be diffuse, sectoral or nodular, is usually idiopathic and self-limiting, but is sometimes associated with systemic collagen vascular diseases and autoimmune diseases.

An underlying cause is found in about a third of cases.[45] These conditions include rheumatoid arthritis, Chron disease, ulcerative colitis, psoriatic arthritis, systemic lupus erythematosus, reactive arthritis, relapsing polychrondritis, anklylosing spondylitis, polyarteritis nodosa, Beçhet disease, Cogan syndrome and Wegener granulomatosis.[46] Some infections are also linked to episcleritis, including Lyme disease, cat scratch fever, syphilis and herpes virus, but are much less common than the collagen vascular and autoimmune diseases.[46]

Episcleritis is commonly diagnosed in young to middle-aged females and is rarely diagnosed in chil-

dren. Patients with episcleritis complain of discomfort or irritation (rather than true eye pain), redness and edema to the affected area over the sclera. Visual acuity is not affected and there is rarely discharge or photophobia. Episcleritis is described as diffuse, where all or part of the episclera is inflamed, or nodular, where the inflammation is confined to an area with the presence of a well-defined, non-mobile, red nodule. Diffuse episcleritis occurs in about 70% of cases, while nodular episcleritis occurs in approximately 30%.[47] Nodular episcleritis is often more uncomfortable than a diffuse episcleritis and takes longer to resolve.

The condition is self-limiting, generally running its course in a few days while the nodular form may last for weeks. Many patients may require treatment for the redness and discomfort. Cold compresses and artificial tears provide symptomatic relief. Topical nonsteroidal anti-inflammatory drugs (NSAIDs) and steroids are used for persistent symptoms. Rarely, systemic steroids may be necessary. Immunosuppressive treatment to control an underlying autoimmune disorder is the last resort for resistant cases.[48] Although episcleritis is usually a benign condition with a good prognosis, there may be some instances where communication with the patient's rheumatologist is in order.

Conjunctivitis encompasses a wide range of diseases occurring worldwide. It rarely causes permanent vision loss, but its impact on patients' quality of life can be considerable. It can cause them to miss work or school, not to mention its effect on their wallet.[49] Our clinical duty is to properly diagnose and, when necessary, treat this condition, whatever its origin, with a targeted approach. ■

Dr. Bowling is a past recipient of the Georgia Optometric Association's Bernard Kahn Memorial Award for outstanding service to the optometric profession.

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PEER • REVIEWED PRA ARTICLE

Differentiating Problems of the Eyelids and Ocular Adnexa

When patients ask about dermatologic concerns around the eyes, be prepared to address them in the office and refer when needed.

By Marlon J. Demeritt, OD, and Beata I. Lewandowska, OD

To the general population, lesions of the eyelid skin are often a cosmetic, not a medical, concern. While ophthalmologists, and optometrists in numerous states, can surgically address the cosmetic issues of eyelid lesions, the primary concern is whether the lesion in question is benign or malignant in nature and whether it warrants a referral.

Clinical decision making is guided by the pertinent information gathered about the patient's medical history; thus, effective communication is a critical aspect of a successful physician-patient encounter. Careful history taking and examination can reveal important clues when suspecting malignancy of eyelid lesions.

The most common risk factors for developing skin cancer include fair skin, previous skin cancer, smoking, excessive sun exposure, prior radiation and immunosuppression.[1] To reveal these risk factors in a patient's history, clinicians can ask these questions:

- When did you first notice the lesion?
- Has the lesion changed in color, size or shape?
- Has the lesion caused you any pain or irritation?
- Has the lesion caused bleeding or draining of fluid or purulent material?
- Have you noticed any other similar skin lesions on other

Release Date: February 15, 2020
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Educational Objectives: After completing this activity, the participant should be better able to:

- Recognize common dermatological conditions and how to differentiate between them.
- Take a good clinical history to help hone the diagnosis.
- Describe when and how to refer.
- Discuss the basics of treating these common dermatological conditions in the office.

Target Audience: This activity is intended for optometrists engaged in the care of patients with dermatological conditions of the eyelids and adnexa.

Accreditation Statement: In support of improving patient care, this activity has been planned and implemented by the Postgraduate Institute for Medicine and Review Education Group. Postgraduate Institute for Medicine is jointly accredited by the Accreditation Council for Continuing Medical Education, the Accreditation Council for Pharmacy Education, and the American Nurses Credentialing Center, to provide continuing education for the healthcare team. Postgraduate Institute for Medicine is accredited by COPE to provide continuing education to optometrists.

Faculty/Editorial Board: Marlon Demeritt, OD, and Beata Lewandowska, OD.

Credit Statement: This course is COPE approved for 2 hours of CE credit. Course ID is **66533-GO**. Check with your local state licensing board to see if this counts toward your CE requirement for relicensure.

Disclosure Statements:

Drs. Demeritt and Lewandowska have nothing to disclose.

Managers and Editorial Staff: The PIM planners and managers have nothing to disclose. The Review Education Group planners, managers and editorial staff have nothing to disclose.

parts of your body?
- Have you had similar lesions on your eyelids in the past?
- Do you have a history of skin cancer?

When to Refer

The decision to refer depends on various factors, such as the risk of malignancy and the cosmetic concern of the patient. Malignancy is and should be the primary concern of the provider and the patient. Any of the following signs and symptoms are suspicious and warrant referral:

- The lesion violates the ABCDE rule (*Table 1*).
- The lesion becomes ulcerated, bleeds or loses eyelashes.
- The lesion changes color.
- The patient notices growth after onset.
- The borders of the lesion assume an irregular shape.
- Risk factors exist that indicate suspicion for malignancy.

Non-malignant Issues

Once malignancy has been ruled out, there are a number of common non-malignant dermatologic findings you might encounter in your practice (*Table 2*). Many of these conditions have similar presentations. Here's a look at how to differentiate each one (*Table 3*).

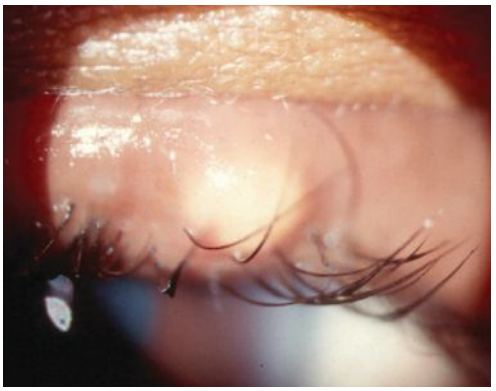

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Fig. 1. Localized swelling secondary to an external hordeolum pointing anteriorly through the eyelid skin.

Cysts are benign, painless focal blockages of the glands that can occur on the palpebral conjunctiva or the skin of the eyelid. Cysts of Moll, dome-shaped papules or nodules filled with clear fluid that transilluminates, are the result of blocked apocrine sweat glands. The secretory cells of Moll can give rise to bluish adenomas, which are known as apocrine hidrocystomas. Blocked sebaceous glands will give rise to a cyst of Zeiss, which, unlike cysts of Moll, will not transilluminate, as they are filled with a yellow oil-like secretion. Epidermal inclusion cysts, which tend to be elevated, round, keratin-filled, firm lesions, arise from an occlusion of the infundibulum of the hair follicle. These cysts are often slow-growing and may have a central pore. They require surgical excision.

Hordeola, which usually present as swollen, red and painful lesions, are secondary to acute bacterial infections. According to recent studies, 90% of hordeola are associated with *Staphylococcus aureus*.[2] Internal hordeola originate in the meibomian glands while external hordeola originate in the glands of Zeiss or Moll.[3] Patients at risk for developing a hordeolum are those with a history of poor lid hygiene or inflammatory diseases of the eyelid such as blepharitis, rosacea and meibomitis.[4,5]

The common approach to managing hordeola involves observation in cases where the lesion is smaller in size and the patient is asymptomatic (*Figure 1*). Daily lid hygiene as well as topical and systemic antibiotics can be used in situations where size, cosmesis or discomfort are a problem. Topical steroids can be effective in reducing healing time and symptoms associated with inflammation.[6,7]

Recurrent hordeola can occur due to failure of complete bacteria eradication.[8] A recurrent hordeolum or chalazion—a similar lesion caused by a clogged oil gland—in the same area should prompt the clinician to suspect sebaceous gland carcinoma. One study reported sebaceous gland carcinoma as the most commonly missed malignancy, oftentimes misdiagnosed as a chalazion.[9]

Sebaceous gland carcinoma, an aggressive neoplasm with possible local and distant metastasis, has a

Table 1. The ABCDEs of Melanoma[55]

A = asymmetry	Malignant lesions tend to be asymmetric.
B = border	Malignant lesions tend to have irregular, scalloped and poorly defined borders.
C = color	Malignant lesions tend to vary in color from one area to another.
D = diameter	Melanomas are usually greater than 6mm at the time of diagnosis.
E = evolution	Malignant lesions tend to evolve and will change over time in aspects of shape, size and color.

Table 2. Dermatological Terms

Cyst	A closed sac that contains liquid/semisolid material, usually resilient on palpation.
Papule	A solid, elevated lesion with no visible fluid; up to 0.50cm in diameter.
Nodule	A larger and deeper papule that may be in the dermis or subcutaneous tissue, or in the epidermis; usually 0.50cm or more in diameter.
Pustule	A circumscribed elevation containing a purulent exudate (white, yellow or greenish-yellow).
Vesicle	A circumscribed epidermal elevation containing clear fluid; less than 0.50cm in diameter.

Table 3. Dermatological Differential Diagnoses	
Papilloma	Verrucae vulgaris, seborrheic keratosis, verrucous carcinoma
Hordeola	Preseptal cellulitis, sebaceous gland carcinoma, pyogenic granuloma, chalazion
Molluscum contagiosum	Papilloma, verruca vulgaris, keratoacanthoma, herpes simplex, herpes zoster
Contact dermatitis	Rosacea, atopic eczema, seborrheic dermatitis, blepharitis, psoriasis
Preseptal cellulitis	Orbital cellulitis, chalazion, hordeola, viral conjunctivitis, eyelid abscess
Orbital cellulitis	Preseptal cellulitis, orbital tumors, orbital pseudotumor, thyroid eye disease, mucormycosis
Ocular rosacea	Posterior blepharitis, contact dermatitis
Impetigo	Herpes simplex dermatitis, discoid lupus erythematosus, cutaneous candidiasis
Herpes zoster ophthalmicus	Herpes simplex, contact dermatitis, rosacea

higher rate of morbidity and 5% to 38% mortality.[10] Recurrent hordeola or chalazia should be considered suspicious in nature and should be referred for biopsy.

The term ***papilloma*** encompasses a group of various benign epithelial skin lesions. Most commonly, a benign squamous papilloma is an elevated, smooth, rounded, sessile or pedunculated, flesh-colored lesion often found on the eyelid skin. These lesions may be treated with either cryotherapy or surgical excision.

Verruca vulgaris, also known as a viral wart, is a common benign cutaneous lesion caused by the human papilloma virus (HPV).[1] These lesions have different shapes, sizes and levels of pigmentation. They appear as hyperkeratotic papules with a rough, irregular surface. Filiform warts are long, slender growths often found on the eyelid skin. Verrucae affecting the eyelids are usually asymptomatic and can be observed (*Figure 2*). In some cases, they may cause cosmetic disfigurement, which would warrant surgical excision.

Molluscum contagiosum (MC) is caused by viral infection of the skin (*Figure 3*). On clinical exam, expect to see a solitary lesion or multiple lesions that appear as umbilicated papules that are pink or skin-colored.[11,12] Small, solitary lesions are generally seen in immunocompetent patients, while the immunosuppressed patient will have larger (greater than 1cm) and more extensive papules.[13-15] Transmission of the virus occurs through direct contact with infected skin, contaminated towels or even swimming in a pool.[16] Although you can see molluscum contagiosum in an immunocompetent patient, the molluscum poxvirus is more common in immunocompromised patients, with the prevalence in HIV patients close to 20%.[17] Ophthalmic lesions from MC are common on the lid margin and can lead to chronic follicular conjunctivitis due to the shedding of viral particles into the tear film.[18-20]

While evaluating a patient with conjunctivitis, these lesions can be overlooked, especially when the patient seems healthy. Because these lid lesions can be implicated as causes of follicular conjunctivitis, a careful dermatological evaluation of the eyelids is essential. Patients with the molluscum virus may develop eczematous plaques around one or more lesions, which is known as molluscum dermatitis. Approximately 9% to 47% of patients with molluscum will develop this condition; however, unlike allergic contact dermatitis, there is no clear consensus if the application of topical steroids facilitates resolution of the molluscum lesions.[21-23]

Molluscum contagiosum lesions are usually self-limiting, with a duration of six to nine months. Still, in some cases the duration can span three to four years.[24] In immunosuppressed patients, the condition is usually refractory to treatment.[15]

Treatment options include observation, antiviral medications, curettage, immunomodulators and specific chemicals, such as cantharidin and 10% potassium hydroxide, targeted at destroying the lesion.

In an immunocompetent patient with a solitary lesion, it is likely safe to monitor the patient for up to nine months. Once the patient develops a follicular conjunctivitis secondary to the lesion, a referral to an oculoplastic surgeon is warranted.

Contact dermatitis is an inflammatory condition of the skin that is precipitated by exposure to an

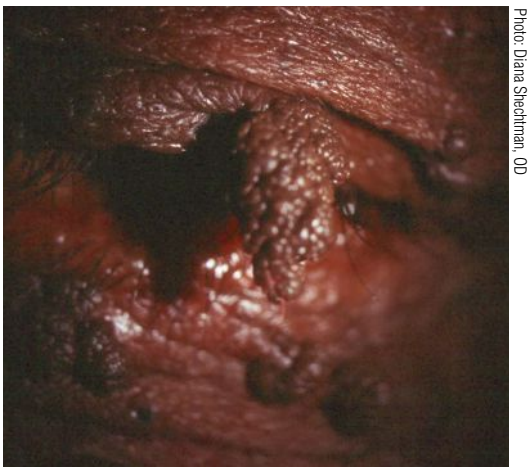

Photo: Diana Shechtman, OD

Fig. 2. Asymptomatic filiform verruca with a typical irregular surface.

offending agent that produces an allergic response.[25]

Two types exist: an irritant contact dermatitis and an allergic contact dermatitis (ACD). Here, we will focus our attention on ACD. The mediation of skin inflammation occurs due to activation of antigen-specific acquired immunity. The activation of the acquired immunity leads to the development of effector T cells.[26] Activation of effector T cells leads to the inflammatory process responsible for the cutaneous lesions and occurrence of ACD.[26,27]

The patient may complain of itching and swelling of the eyelids and around the eyes. The case history will uncover recent exposure to the offending allergen responsible for the reaction. The periorbital area will reveal swelling and erythema of the eyelids and, in some cases, a scaly appearance on the ocular adnexa. If all four eyelids are involved, allergic contact dermatitis is very likely the diagnosis.[28] If uncertain of the diagnosis, you may refer your patient to a dermatologist for a patch test.

If the offending agent is known, eliminate contact with it, and if no contraindications are present, initiate a topical steroid cream for management of the dermatitis. It is important to keep in mind that, although rare, some patients may be allergic to the preservatives used in the base of the steroid cream. In addition, topical steroids carry a risk of skin atrophy as well as hypo- and hyperpigmentation. For cases unresponsive to conservative management, consider referral to a dermatologist.

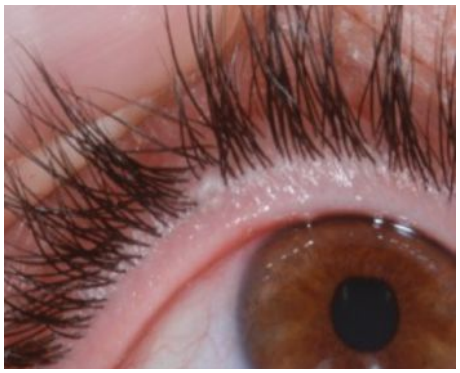

Fig. 3. This is an example of a single solitary skin colored Molluscum contagiosum lesion with an umbilicated center.

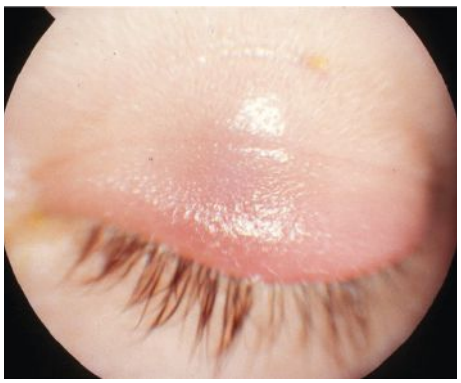

Fig. 4. This child had swelling of the upper eyelid in a case of preseptal cellulitis, which can generally be managed with broad-spectrum oral antibiotics.

Preseptal and ***orbital*** are the two types of ***cellulitis*** you may encounter in clinical practice. While the former involves an infection of the anterior portion of the eyelid, the latter is an infection of the contents of the orbit, which include periorbital fat, extraocular muscles and neurovascular bundles.[29,30] Initially, the two conditions may appear indistinguishable. Fortunately, preseptal cellulitis is more common than orbital cellulitis. Preseptal can progress to orbital cellulitis, which is usually associated with high mortality rates.[31,32]

To properly distinguish between preseptal and orbital cellulitis, clinicians should obtain a good history, evaluate the patient and order laboratory testing and imaging studies.[33] During the history, inquire about when symptoms started and whether the patient is experiencing any reduced vision, double vision, pain or fever. Patients with orbital cellulitis may present with a recent acute sinusitis or a history of upper respiratory tract infection.[33]

While patients with preseptal usually present with sudden-onset redness, swelling, eye pain and mild fever, patients with orbital cellulitis may have limited extraocular movements, pain, decreased vision, diplopia and proptosis.[34,35] Imaging studies in the form of a CT scan should be performed within 24 hours, if there is no improvement with antibiotic therapy or if there is a deterioration of the patient's condition.[29,36]

Generally, empirical treatment is initiated with broad-spectrum oral antibiotics to manage the preseptal cellulitis (*Figure 4*). Orbital cellulitis can be life- and vision-threatening, so prompt diagnosis and management are crucial. Most patients require hospital admission when the following signs are present: periorbital swelling, decreased vision, double vision, abnormal pupillary responses, proptosis, restricted ocular motility, headache, seizures, vomiting and drowsiness.[36] Management of orbital cellulitis involves an intravenous antibiotic for one to two weeks, followed by oral antibiotics for four weeks.[37]

Impetigo is a term used to describe an infection caused by *Streptococcus pyogenes* or *Staphylococcus aureus* in the superficial keratin layer of the skin. It often presents in socioeconomically disadvantaged children between the ages of two and five as discrete purulent lesions of the eyelid skin.[38] These lesions begin as papules, then rapidly evolve into vesicles surrounded by skin erythema. The lesions gradually enlarge into pustules before breaking down over four to six days to form thick crusts.

Treatment involves systemic penicillinase-resistant penicillins or first-generation cephalosporins.

Altabax (retapamulin) 1% topical antibiotic ointment is FDA approved for children nine months or older and should be applied BID for five days.[38] Improved personal hygiene and improved living conditions are also recommended.

Rosacea is a chronic inflammatory condition of the skin that affects the facial and ocular regions (*Figure 5*). Typically, rosacea affects patients between 40 and 59 years of age.[39] The National Rosacea Society estimates that 16 million Americans have acne rosacea, and 58% to 72% of acne rosacea patients also suffer from ocular symptoms.[40] There are four distinct subtypes describing varying degrees of inflammation. Subtypes I, II and III are classified as erythematelangiectatic rosacea, papulopustular rosacea or phymatous rosacea, respectively.[41] Ocular rosacea is classified as the fourth subtype.[41]

Ocular involvement can occur concomitantly or independently of the facial features of redness overlying the patient's nose and cheeks.[42] The dermatologic reaction associated with rosacea results in meibomian gland dysfunction, scarring of the meibomian gland orifices and telangiectasia of the eyelid margin.[43-47] This can lead to the reduction in the efficiency of the lipid layer, which causes the patient's vision to become compromised due to an unstable tear film, thus contributing to symptoms of ocular dryness. It is not uncommon for severe cases of rosacea to lead to ocular surface conditions, such as recurrent corneal erosions, ulceration and corneal perforations.[43,46,48]

Various approaches to the management of rosacea exist, such as lifestyle modification, conservative therapies, medical management, surgical management and laser therapy. Conservative treatments such as lid hygiene facilitate improved outflow through the meibomian glands and stabilization of the ocular surface.[49]

Medical management includes the use of artificial tears, nutritional supplements, immunosuppressive agents, antibiotics and antimicrobials. Symptoms of dry eye are common with rosacea because of its effect on the meibomian glands. Artificial tears are helpful in ameliorating the symptoms of dry eye due to ocular rosacea, but twice-a-day instillation of cyclosporine A has proven to be more efficacious than artificial tears.[50]

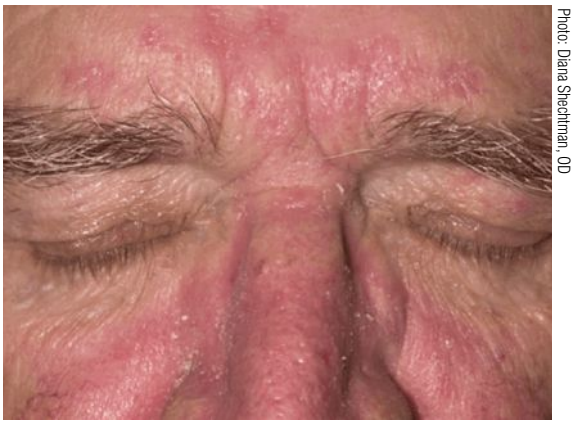

Photo: Diana Shechtman, OD

Fig. 5. This patient presented with facial features of rosacea, including redness of the nose and cheeks extending into the forehead.

Included in our armamentarium of treatment options are antibiotics, specifically oral doxycycline, with several authorities suggesting that antibiotics become a constant in the management of ocular rosacea.[49,51] Laser and surgical treatment options are also possible, and patients should be referred to dermatology for these. Intense pulsed light is an emerging treatment for rosacea and, off-label, meibomian gland dysfunction. The procedure involves direct application of light of appropriate wavelengths to the skin; research shows that it improves tear breakup time and patient symptoms as well as reduces skin erythema and telangectasia of rosacea.[52,53]

Careful evaluation and proper treatment of patients with ***herpes zoster ophthalmicus*** are crucial to decrease long-term morbidity and incidence of postherpetic neuralgia. When human herpesvirus type 3 reactivates in the ophthalmic division of the trigeminal nerve, pain and a breakout of cutaneous pustules will often cause the patient to rush to the office. This can occur in one or multiple areas that include the forehead skin, eyebrow, eyelid and the skin at the side of the tip of the nose (Hutchinson's sign). Between 50% and 72% of patients will demonstrate ocular involvement with eyelid rash or, in more severe cases, corneal epithelial and stromal involvement, optic neuritis, uveitis or retinal necrosis.[54]

Initially, the pain is often a mild burning and tingling sensation, known as a prodrome, but as the body's inflammatory response becomes more severe, the pain becomes more intense. The eyelids will typically manifest a rash that may develop a secondary bacterial infection resulting in yellowing, crusting and discharge.

A corneal pseudodendrite of heaped-up epithelium that exhibits "negative staining" by collecting the fluorescein stain at its edges may be present. Using rose bengal or lissamine green vital dyes can assist in differentiating between a pseudodendrite and dendritic lesion secondary to herpes simplex virus.

Anterior stromal keratitis with stromal infiltrates may coalesce to form a nummular keratitis. Infection of the corneal endothelium may present with a stromal keratitis and elevated intraocular pressure due to a trabeculitis.

Table 4. Systemic Antiviral Medications for the Treatment of Herpes Zoster[56,57]

Acyclovir*	800mg five times per day PO
Valacyclovir*	1,000mg TID PO
Famciclovir*	500mg TID PO

*seven-day dosage

If the patient is immunocompromised, rapid necrotic inflammation of the retina may develop, often leading to permanent vision loss. Therefore, a thorough medical history and a complete ophthalmic examination with a dilated retinal fundus exam is indicated in these patients. The most common chronic complication of herpes zoster infection is postherpetic neuralgia, consisting of severe, debilitating pain that may persist indefinitely and is seen in 9% to 45% of cases.[54]

All patients presenting with herpes zoster ophthalmicus require treatment with systemic antiviral medications (*Table 4*). Evidence shows that therapy with oral antiviral medications started within 72 hours after the onset of rash can reduce the duration of viral shedding, promote resolution of skin lesions and limit the duration of pain.[54] Artificial tears to lubricate the ocular surface and a topical ophthalmic antibiotic ointment used four times a day to prevent a superinfection should be considered. Topical steroids are helpful in stromal keratitis, uveitis and episcleritis/scleritis, but caution should be exercised if an epithelial defect is still present. Immunocompromised patients as well as patients with retinal involvement should be referred to a retinal specialist.

Many of our patients' concerns regarding the eyelid skin and the ocular adnexa can be addressed in the office. However, in cases of suspected malignancy or when patients are not responding to the appropriate treatment, a consultation with an oculoplastic specialist or dermatologist may be indicated. ■

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OSC QUIZ

You can obtain continuing education credit through the Optometric Study Center. Complete the test form and return it with the $35 fee to: Jobson Healthcare Information, LLC, Attn.: CE Processing, 395 Hudson Street, 3rd Floor New York, New York 10014. To be eligible, please return the card within three years of publication. You can also access the test form and submit your answers and payment via credit card at *Review Education Group* online, www.revieweducationgroup.com.

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Please check with your state licensing board to see if this approval counts toward your CE requirement for relicensure.

1. The following type of medication has been shown to reduce healing time and symptoms in patients with hordeola:
a. Oral fluoroquinolones.
b. Topical anti-infective drops.
c. Topical NSAID drops.
d. Topical steroid drops.

2. Recurrent hordeolum should be referred out due to suspicion of the following:
a. Basal cell carcinoma.
b. Squamous cell carcinoma.
c. Sebaceous gland carcinoma.
d. Seborrheic keratosis.

3. In patients affected by the molluscum poxvirus with ophthalmic involvement, the lesion is most commonly located in the following:
a. Conjunctiva.
b. Cornea.
c. Eyelid.
d. Retina.

4. The following is on average the duration of a self-limiting molluscum lesion:
a. Two to four months.
b. Six to nine months.
c. Three months.
d. Five years.

5. Patients with allergic contact dermatitis will present with complaints of itching and swelling of the eyelids as well as around the eyes. The following clinical signs help determine if an allergic contact dermatitis is the likely diagnosis:
a. Right upper lid and left upper lid affected only.
b. Right lower lid and left lower lid affected only.
c. Right upper lid and left lower lid are affected only.
d. All four eyelids are affected.

6. Patients with preseptal cellulitis should have an orbital CT scan within _____ hours if they do not show signs of clinical improvement:
a. Two.
b. Eight.
c. 24.
d. 72.

7. A patient with preseptal cellulitis may present with which of the following signs:
a. Proptosis.
b. Relative afferent pupillary defect.
c. Sudden-onset diplopia.
d. Sudden-onset redness.

8. Ocular rosacea is classified within the following subtype of inflammation:
a. I.
b. II.
c. III.
d. IV.

9. Severe cases of untreated ocular rosacea can lead to all of the following ocular complications, *except*:
a. Anterior uveitis.
b. Corneal perforations.
c. Corneal ulcerations.
d. Recurrent corneal erosions.

10. All of the following are treatment options for ocular rosacea, *except*:
a. Oral doxycycline.
b. Intense pulsed light.
c. Cyclosporine.
d. Oral acyclovir.

11. Apocrine hidrocystomas are adenomas that arise in which of the following glands:
a. Lacrimal.
b. Meibomian.
c. Moll.
d. Zeiss.

12. A fluid-filled cyst that does *not* transilluminate is most likely located in the following gland:
a. Lacrimal.
b. Meibomian.
c. Moll.
d. Zeiss.

13. Verruca vulgaris is most likely associated with the following virus:
a. Herpes simplex.
b. Human papilloma.
c. Molluscum contagiosum.
d. Varicella zoster.

14. Impetigo is an infection in the skin most commonly caused by which pathogen:
a. *Staphylococcus albus*.
b. *Staphylococcus epidermidis*.
c. *Streptococcus pneumoniae*.
d. *Streptococcus pyogenes*.

15. The treatment of impetigo includes all of the following, *except*:
a. Retapamulin 1% ung.
b. First-generation cephalosporins.
c. Improved personal hygiene.
d. Valacyclovir 1g.

16. The presence of a cutaneous pustule at the side of the tip of the nose in patients with herpes zoster is known as the following sign:
a. Hutchinson's.
b. Munson's.
c. Rizzuti's.
d. Vogt's.

17. All of the following are appropriate for the treatment of herpes zoster, *except*:
a. Famciclovir 500mg TID PO.
b. Valacyclovir 1,000mg TID PO.
c. Acyclovir 400mg five times per day PO.
d. Acyclovir 800mg five times per day PO.

18. The following is the most common complication of herpes zoster:
a. Encephalitis.
b. Pneumonia.
c. Postherpetic neuralgia.
d. Vision loss.

19. To reduce the duration of viral shedding, promote resolution of skin lesions and limit the duration of pain in patients with herpes zoster, treatment with oral antivirals should be initiated within:
a. 24 hours.
b. 48 hours.
c. 72 hours.
d. 96 hours.

20. The following is a characteristic of malignant skin lesions:
a. Evolution.
b. Symmetry.
c. Sharp borders.
d. Uniform color.

Examination Answer Sheet

Differentiating Problems of the Eyelids and Ocular Adnexa

Valid for credit through February 15, 2023

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Answers to CE exam:

1. Ⓐ Ⓑ Ⓒ Ⓓ
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19. Ⓐ Ⓑ Ⓒ Ⓓ
20. Ⓐ Ⓑ Ⓒ Ⓓ

Post-activity evaluation questions:

Rate how well the activity supported your achievement of these learning objectives:
1=Poor, 2=Fair, 3=Neutral, 4=Good, 5=Excellent

21. Recognize common dermatological conditions and how to differentiate between them. ① ② ③ ④ ⑤
22. Take a good clinical history to help hone the diagnosis. ① ② ③ ④ ⑤
23. Describe when and how to refer. ① ② ③ ④ ⑤
24. Discuss the basics of treating these common dermatological conditions in the office. ① ② ③ ④ ⑤

25. Based upon your participation in this activity, do you intend to change your practice behavior?
(choose only one of the following options)
Ⓐ I do plan to implement changes in my practice based on the information presented.
Ⓑ My current practice has been reinforced by the information presented.
Ⓒ I need more information before I will change my practice.

26. Thinking about how your participation in this activity will influence your patient care, how many of your patients are likely to benefit? (please use a number): ☐

27. If you plan to change your practice behavior, what type of changes do you plan to implement? (check all that apply)

ⓐ Apply latest guidelines ⓑ Change in pharmaceutical therapy ⓒ Choice of treatment/management approach ⓓ Change in current practice for referral ⓔ Change in non-pharmaceutical therapy ⓕ Change in differential diagnosis ⓖ Change in diagnostic testing ⓗ Other, please specify: _____

28. How confident are you that you will be able to make your intended changes?

ⓐ Very confident ⓑ Somewhat confident ⓒ Unsure ⓓ Not confident

29. Which of the following do you anticipate will be the primary barrier to implementing these changes?
ⓐ Formulary restrictions
ⓑ Time constraints
ⓒ System constraints
ⓓ Insurance/financial issues
ⓔ Lack of interprofessional team support
ⓕ Treatment related adverse events
ⓖ Patient adherence/compliance
ⓗ Other, please specify:

30. Additional comments on this course:

Please retain a copy for your records. Please print clearly.

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Rate the quality of the material provided:
1=Strongly disagree, 2=Somewhat disagree, 3=Neutral, 4=Somewhat agree, 5=Strongly agree

31. The content was evidence-based. ① ② ③ ④ ⑤
32. The content was balanced and free of bias. ① ② ③ ④ ⑤
33. The presentation was clear and effective. ① ② ③ ④ ⑤

By submitting this answer sheet, I certify that I have read the lesson in its entirety and completed the self-assessment exam personally based on the material presented. I have not obtained the answers to this exam by any fraudulent or improper means.

Signature _____ Date _____

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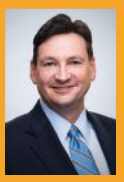

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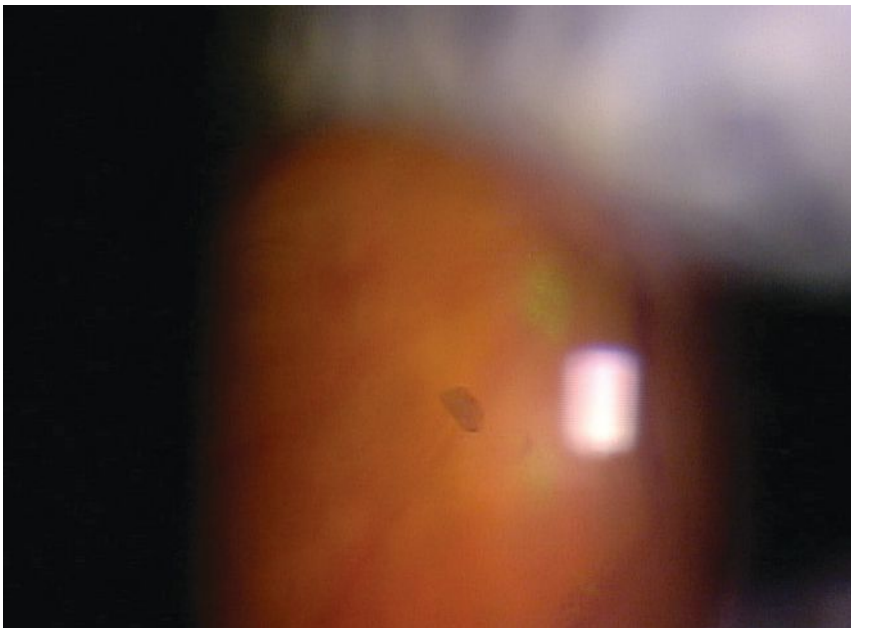
Sinking the Floaters

With laser indications proliferating, optometry can do more to address patients troubled by the benign debris. **By Myranda Partin, OD, and Nathan Lighthizer, OD**

Optometrists understand that floaters are usually not indicative of a serious health issue, but for patients—particularly those experiencing posterior vitreous detachment (PVD)—this annoying phenomenon can be a source of irritation and stress. And patients experiencing an influx of floaters are not rare.

Research shows by the age of 50, 50% of patients will have had a PVD, by the age of 65, 65%, and so on.[1] Although these visual disturbances don't permanently damage visual acuity, patients may complain that they have deleterious effects on their visual functioning and affect their abilities to read or drive, for example.[2]

In addition to keeping the eye healthy, optometrists are tasked with keeping patients happy with their vision—a significant influence on their overall quality of life.[3,4] And so, when a patient's primary complaint is an influx of floaters, it's necessary that optometrists take patient concerns seriously, even for a rather benign condition.



This patient is about to undergo nd:YAG vitreolysis which will address an influx of floaters. For some patients, this procedure can relieve an irritating visual disturbance.

It's on us to not only describe what's happening within the vitreous of their eyes but also to explain what technologies and techniques eye care physicians have in their toolbox to address floaters and explain whether they're appropriate patient candidates for these procedures.

This article describes the development of these pesky floaters, what patients can achive from undergoing a procedure to address them, how to co-manage patients through the process and, with an eye toward the ODs, expanding scope of practice, how to remove floaters using nd:YAG lasers.

Weiss Rings

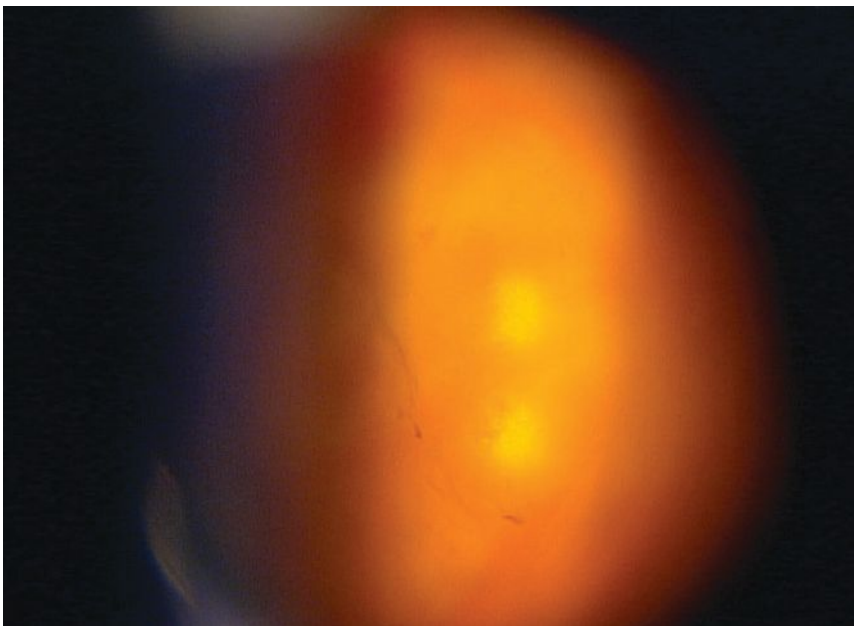
When a patient first presents with a symptomatic floater, education is vital. First, carefully examine the macula and peripheral retina to rule out any underlying issues, such as retinal detachment, that could threaten vision. If no such condition is found, discuss the neural-adaptation process along with the recommendation that they wait four to six months before considering treatment.[5]

When we are young, hyaluronan keeps collagen fibrils separated in the vitreous cavity, maintaining transparency of the vitreous.[6] As we age, the process of vitreous liquefaction starts a breakdown in the collagen molecules causing those fibrous structures to clump into networks.[6] The resulting process, PVD, is often marked by fibroglial tissue that's left free-floating over the optic nerve, called a Weiss ring. This ring and other vitreous opacities cast shadows onto the retina that are perceived as floaters.[7]

The histopathological make-up may continue to change, pushing the floater out of the symptomatic line of sight, another reason to wait approximately six months to consider referring for treatment of symptomatic vitreous floaters.[5]

It's also important to note that the risk of a retinal tear or detachment after an acute PVD is highest in the condition's first six weeks and diminishes at six months.[8]

Thorough patient education is key to their understanding of the need to wait before considering treatment since a fresh PVD can be a very dramatic experience for a patient. This discussion must also assure the patient that floaters do not cause blindness or permanent vision loss. Most times when patients return a month later for follow up, they usually are not as anxious or bothered as the initial visit. Therefore, treating all symptomatic patients for their floaters from a PVD would lead to overtreatment, creating unnecessary risk of surgical complications.



The patient, mid-procedure, is undergoing nd:YAG vitreolysis.

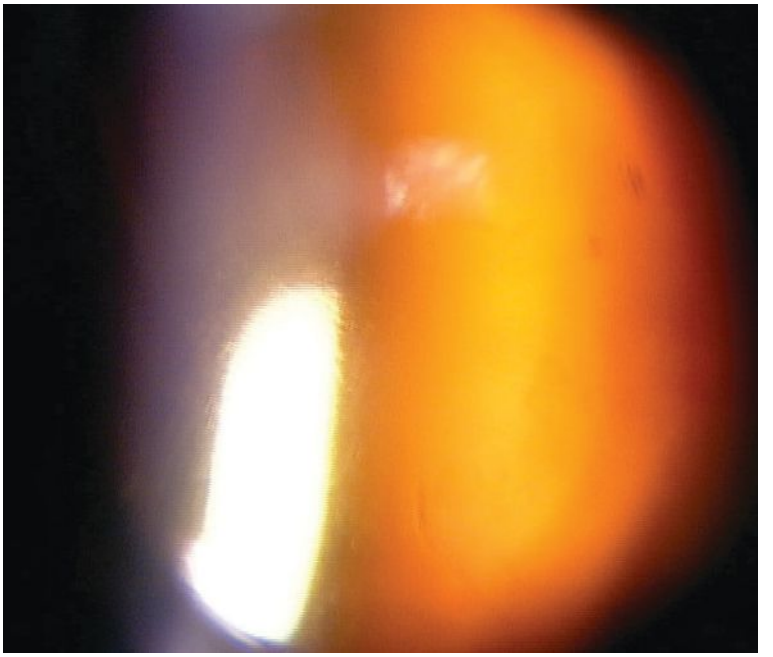
Treatments and Risks

Standard of care obligates us to review risks, benefits and alternatives of any procedure with patients before proceeding. Until nd:YAG vitreolysis, pars plana vitrectomy (PPV) was the only option. Although vitrectomy is essentially a "cure" for floaters, retinal tears or detachments, hypotony, postoperative infection and cataract formation are all risks.[9] These risks have been reduced with the advent of small incision techniques, which employ 27g wounds.[7]

Another option to discuss with patients suffering from persistent floaters is nd:YAG vitreolysis, a laser technique commonly used for opacity of the posterior capsule following cataract surgery. This procedure is currently within optometry's scope of practice in a few states. Like any procedure, patient selection is key. Whether an optometrist is performing it or referring to a surgical center, they must take into account the specific pathology of the patient's vitreous and retina. The most important test for this decision is a complete exam with dilated funduscopy.[10] Before performing any laser procedure, it is recommended that the patient be free of active retinal pathology or chronic ocular inflammation.[10]

During the posterior exam, it is helpful to document any large floaters, syneresis or a Weiss ring suspended in the vitreous.[10] Factors to consider are: the floaters' proximity to the nerve and macula, phakic vs. aphakic anatomical lens, the symptomatic floaters' depth in the vitreous (more anterior or posterior), the floater's size and density, and the amount of liquefaction of (or "the percentage of liquid in") the vitreous.[10] Additionally, literature states the importance of documenting loss

in contrast sensitivity over decreased vision.[10] Lastly, you also want to proceed with caution on a patient who has strong lenticular astigmatism or keratoconus because either of these can make focusing your target beam very difficult.[10]



This photo shows our patient's eye immediately following the procedure. At this point, the patient should wait in the office for at least 30 minutes so doctors can recheck their IOPs.

Selecting for YAG Vitreolysis

First of all, the patient must be symptomatic. These patients will report persistent moving shadows or vision-obstructing clouds of accumulated vitreal networks. Floater selection is critical, too. For best success with YAG vitreolysis, the patient must have a well defined floater—such as a Weiss ring or opaque amorphous cloud. Patients with web-like floaters present all throughout the vitreous are not ideal candidates for YAG vitreolysis.

It's also the optometrist's job, whether they perform the procedure or refer, to prepare patients who are selected with reasonable expectations. In our pratice, we tell these patients that they may need more than one treatment. Often, Weiss rings can be treated in one session, with larger opaque floaters or amorphous clouds taking multiple treatment sessions with the laser.

Limit any treatment to pseudophakic patients to avoid any risk of cataract formation from the abundant amount of laser energy used in the process. Avoid patients with multifocal lenses, because the lens's multiple focal points will divide the nd:YAG laser beam.

Performing the Procedure

For the laser vitreolysis itself, maximum pupillary dilation is of the utmost importance. In our clinic we use 1% tropicamide and 2.5% phenylephrine to help achieve that goal. When you look into a vitreolysis lens, your view shrinks tremendously, as if looking through a straw. Therefore, having maximum dilation will save time and effort while locating the floater in question.

Using the right vitreous laser lenses is crucial to success with nd:YAG vitreolysis. Unlike a non-contact fundus lens whose image is inverted, the Ocular Karickhhoff 21mm or 25mm Vitreous Lens (Ocular Instruments) or the Singh Mid-Vitreous Lens (Volk Optical) create the necessary upright, virtual image.

Next, sit the patient as comfortably as possible behind the laser with a technician holding their forehead against the headrest. The patient could also be strapped in, but not all are comfortable with this. If performing the procedure, it's helpful to locate the floater with a 90D lens behind the laser to orient yourself prior to placing the vitreolysis lens on the patient's eye.

Then, just like any other contact lens, use proparacaine and goniosol or genteal gel prior to placing the lens on the patient's eye. Maximum illumination is recommended to aid in your detection of the floater. Similar to a nd:YAG capsulotomy moving the light tower orientation can sometimes provide you with different views of the floater. Generally speaking, the illumination tower off-axis facilitates better viewing of anterior floaters, while on-axis facilitates better viewing of the floater in the mid- to posterior vitreous. We also find doing this important in locating the floater to orient the patients field of gaze to improve the field of view.

Once the floater is in focus through the optics, verify that your aiming beams are lined up prior to firing. The offset on your laser should be zero for mid-vitreous floaters. The more anterior the floater gets, the more posterior offset should be used, and vice versa.

The nd:YAG laser works by vaporizing floaters caused by plasma formation. Therefore, at

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lower power levels, the vitreous would be fractionated but not vaporized. Research shows that 2mJ to 6mJ of energy will lyse vitreal membranes in rabbits safely if the membranes are at least 2mm in front of the retina.[12] To discern a floater's distance from the retina, investigators recommend first bringing the floater into focus. If the retina is also in focus simultaneously, the floater is most likely too close to the retina to treat. But if the retina is not in focus, it's likely that floater is located anterior enough to avoid retinal damage during treatment.[13] Higher energy is needed to reach more posterior floaters, which increases your risk of damage to retinal structures.

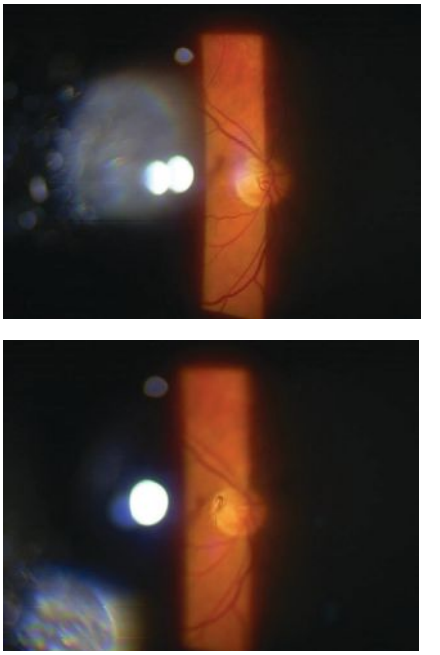
It is recommended that the patient wait in the lobby for at least 30 minutes to recheck pressure before sending them home. A small percentage of patients do experience intraocular pressure (IOP) spikes after treatment. It's common to instill two drops of an aqueous suppressant, such as brimonidine or dorzolamide immediately after the treatment for IOP spike prevention. Some doctors will prescribe a topical steroid such as Pred Forte 1% (prednisolone, Allergan) QID for a week or Durezol (difluprednate, Novartis) BID for a week. Because the laser delivers energy to inert proteins, not living tissue, patients theoretically experience no induced inflammation. Therefore, the topical steroid is not medically necessary (though we still prescribe it at our office).[14]

After the treatment is finished, the patient does not have any restrictions. Notify them that they may see a few dark spots in their vision, from the gas bubbles created during the procedure. Follow-up visits are typically scheduled at one week, one month then four months. Dilate the patient at each of these visits to monitor the retina for any breaks. It's best to wait close to six months before ever repeating vitreolysis to fully evaluate the success of the initial procedure.

Laser Dynamics

The treatment spot size and pulse width are fixed at 8µm and 4 nanoseconds. The only parameters that vary are the energy of the pulse and the number of pulses fired in one shot (i.e., single, double or triple). The Ultra Q Reflex (Ellex) and Tango Reflex (Ellex) lasers are optimized for viewing the vitreous because they allow for coaxial illumination, giving the operator the ability to view the floater and retina at the same time, providing the necessary spatial context needed for safe and efficacious treatments.[15] Most treatments can be performed at between 2.5mJ and 6.5mJ energy and typically take 150 to 200 pulses to accomplish for symptomatic Weiss rings. More shots may be required for amorphous cloud floaters. More energy will be required if the floater is located deep in the posterior vitreous.[16]

The vitreous strand/opacity may move or become mobile during the laser treatment due to the shock wave introduced with each shot fired. It is best to wait until the floater settles back down before firing again. This is why it can take up to 30 minutes for just one procedure, depending on the nature of the floater and the treating doctor's experience. These lasers also have an air-cooled cavity that enables thousands of shots to be fired without overheating or causing the laser to reset.



This patient has a partial Weiss ring with PVD that displays a shadow on their retina.

Measuring Results

One thing no doctor wants is an unsatisfied patient, which is why it's vital to understand whether an objective measurement can be established for a patient's subjective complaint. In fact, the anatomical improvement may not directly correlate with the treatment's success.

Recently published research shows vitreolysis can, in fact, decrease both the amount of vitreous floater opacities seen on color fundus imaging and improve related symptoms.[16] That research employed the National Eye Institute Visual Functioning Questionnaire 25 (NEI VFQ-25) responses of 32 patients to establish subjective successes.[16] The investigators established a statistically significant improvement in the near visual function following laser vitreolysis.[16] Also, the visual disturbance rate improvement

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showed a statistically significant reduction after the treatment.[16] However, they note that distance visual function did not show a statistically significant difference after the procedure.[16]

On the objective side, the researchers used color fundus photography to determine whether vitreous opacity improved over time.[16] Using this method, complete vitreous opacity improvement was observed in 18 eyes (56.2%), partial improvement in 12 eyes (37.5%) and no change in vitreous opacities in only two eyes (6.3%).[16]

That study also shows no elevation in IOP, retinal tears or detachment, significant visual decline or a recurrence of floaters during the follow-up period.[16]

A 2017 study shows that performing vitreolysis can subjectively improve Weiss ring-related symptoms and objectively improve Weiss ring appearance.[17] That research shows 52 subjects split into two groups: one that received the laser procedure and a sham control group.[17] A total of 19 patients in the nd:YAG laser group reported significantly or completely improved symptoms vs. zero in the sham group.[17]

As mentioned, the patient's expectations must be managed beforehand. The 2017 research showed seven of the study's patients who had residual postoperative floaters believed that those were just as bothersome as their original Weiss ring.[17] Eight reported zero improvement in symptoms, even while seven of them had significant objective improvement based on before and after color photography as determined by a masked grader.[17] This means that, for some patients, although a surgeon can vaporize most of a Weiss ring, some residual particles can bother the patient subjectively as much as the initial floater.[18]

Vitreolysis is a relatively new procedure. For some optometrists, knowing about what it can achieve will help them guide their patient and avoid complications. For those optometrists in the few states where it is within the scope of practice, directly providing them relief is possible. In either case, patient education before and after the procedure is critical. Remind patients that, depending on the floater, it may take multiple treatment sessions over multiple visits to significantly improve their symptoms. Patients are always reminded that YAG vitreolysis may not completely eradicate their symptoms but likely will offer a significant improvement. ■

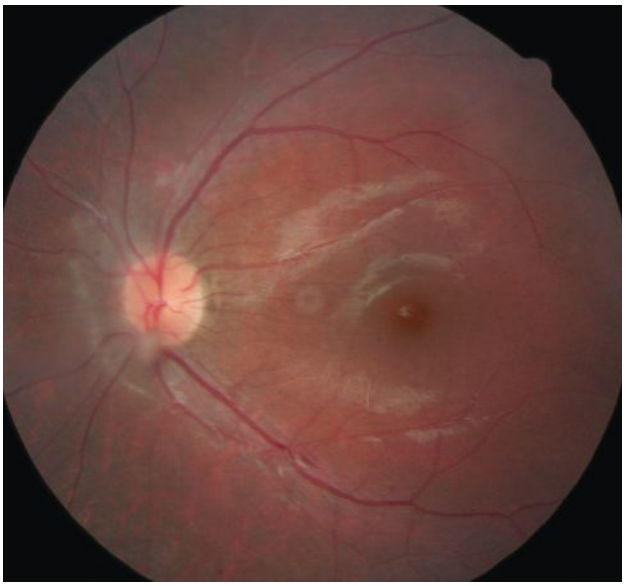

Photo: Dr. Katie West

Here, a floater can be easily seen just above the bottom part of the patient's optic nerve head. This particular floater was too close to the retina, which contraindicated laser treatment.

Dr. Partin is residency trained and currently works at Oklahoma Eye Surgeons as a glaucoma and dry eye specialist.

Dr. Lighthizer is the Associate Dean, Director of Continuing Education and Chief of Specialty Care Clinics at the NSU Oklahoma College of Optometry.

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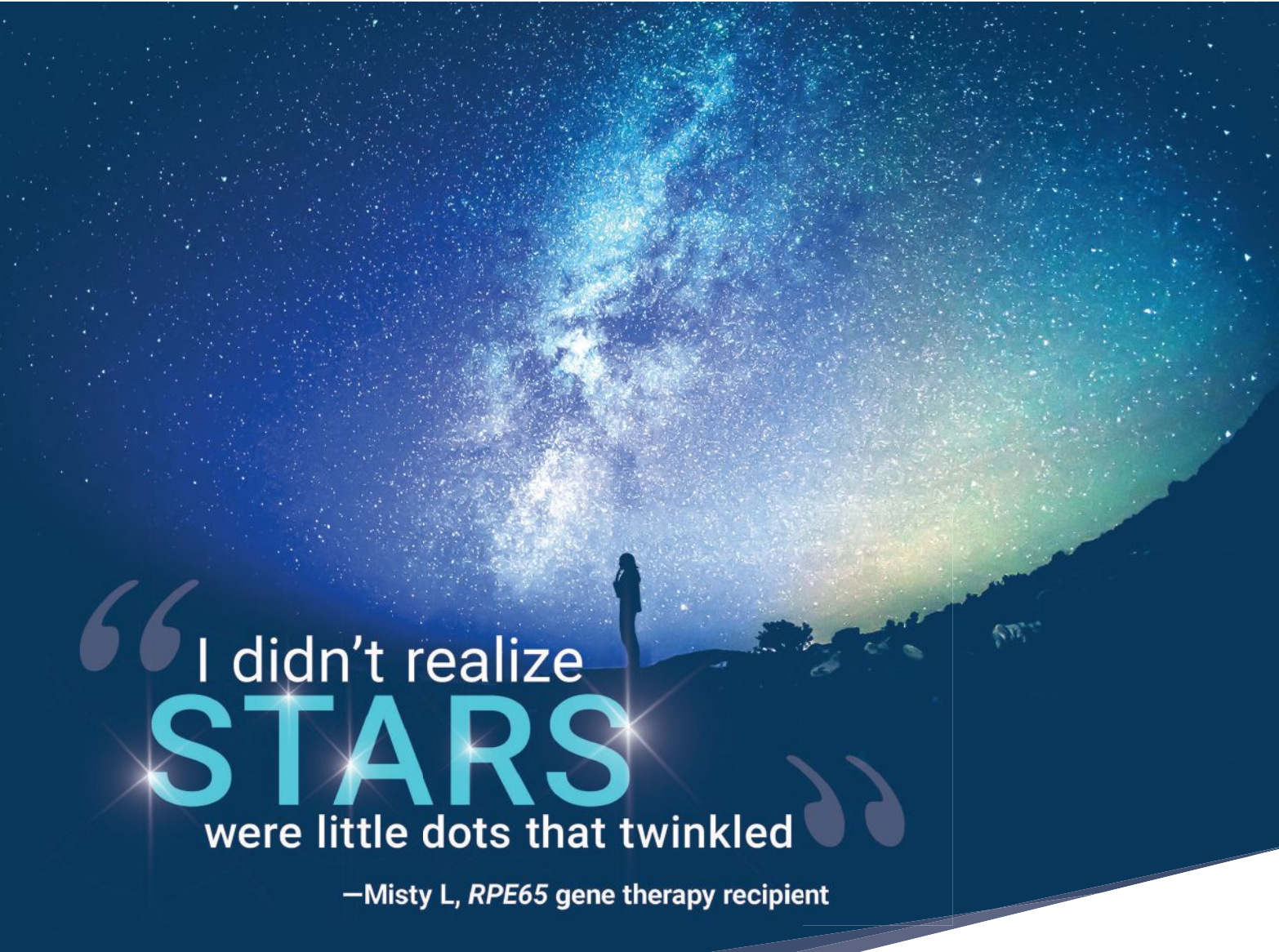

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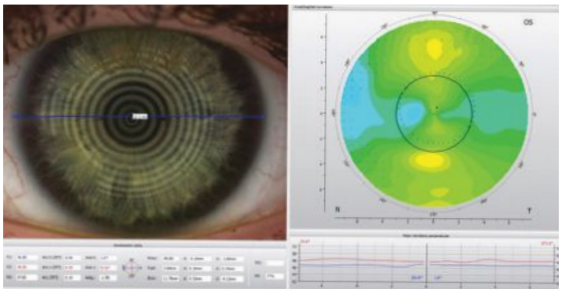
Fighting Flexure

Here's how to manage reduced visual acuity sometimes associated with scleral lens wear.

Edited by Joseph P. Shovlin, OD

Q I recently started fitting scleral lenses on irregular corneas and have run into a few cases where I'm not sure what role flexure is playing when a patient experiences less-than-ideal vision. Sometimes, even a careful spherocylindrical over-refraction fails to improve vision significantly. Could the poor vision be due to another cause?



Topography over a scleral lens shows 1.7D of lens flexure.

A Over the last several years, scleral lenses have proven to be life-changing devices for countless patients who need the protection they afford the corneal and limbal surfaces or who need gas permeable (GP) optics but can't tolerate GP lenses, according to Jason Jedlicka, OD, associate professor at the Indiana University School of Optometry and chief of the school's Cornea and Contact Lens Service. However, Dr. Jedlicka notes that visual acuity can be reduced with scleral lens wear for several reasons, including residual astigmatism due to lens flexure.

The Root of the Problem

Lens flexure occurs when the shape of the corneal surface is regularly toric to the point that the lens bends when it lands, allowing some of the shape of the eye to be manifest in the shape of the lens. Dr. Jedlicka recommends performing keratometry or topography to evaluate lens shape on eye. If the lens is flexing, the results will indicate astigmatism. Sclerals do not land on the cornea, so lens flexure only occurs when there is regular scleral toricity. Additionally, scleral shape does not always correlate with corneal shape, so you cannot necessarily predict flexure based on corneal toricity, he cautions.

Studies show that 30% of eyes demonstrate regular toricity of at least 300µm and 40% demonstrate asymmetric toricity, which means the potential for flexure exists in a number of patients.[1] Dr. Jedlicka suggests the best way to fit scleral lenses to reduce the risk of flexure is to make sure the lens is in optimal alignment with the sclera. If the lens aligns with the sclera, be it toric or not, it should not flex.

If your lens fit is optimal but your refraction is still indicating regular astigmatism, Dr. Jedlicka says the only way to pinpoint the source is with keratometry or topography. If either yields astigmatism and it matches the refraction, he notes that the diagnosis is flexure. He recommends re-evaluating your fit and seeing if you can add toricity to the haptic to improve alignment. If this is not possible, he notes that increasing center thickness, while being mindful of corneal oxygen demand, can help. If this doesn't solve the problem, he says additional front surface toricity may offset the flexure. He warns that this can impact lens thickness, and, as flexure is not always consistent, covering it up with front surface cylinder may not be the best visual solution.

Not all cases of reduced acuity with a scleral lens are due to flexure or residual astigmatism, so other factors must be ruled out. Many irregular cornea patients have higher-order aberrations due to the back surface of their cornea—such as coma in cases of keratoconus—and GP and scleral lenses can do little to address that, Dr. Jedlicka says. He suggests performing retinoscopy to detect irregular optics that persist through a scleral and help determine if this is the problem. Poor wettability, which can happen at any time, and debris in the fluid reservoir, which happens with time, can also reduce acuity.

Helping scleral lens wearers attain the best possible vision means taking all factors into account. With the increased ease of prescribing toric haptics and performing scleral topography to help determine scleral shape and haptic necessity, avoiding or managing flexure should fall well within our wheelhouse. ■

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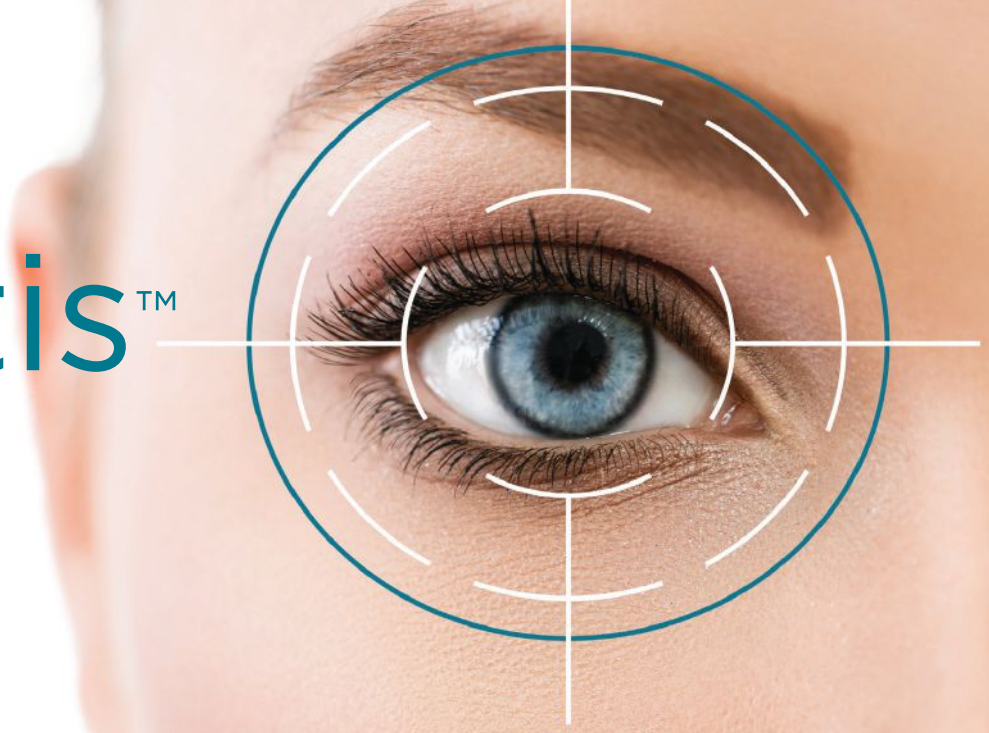

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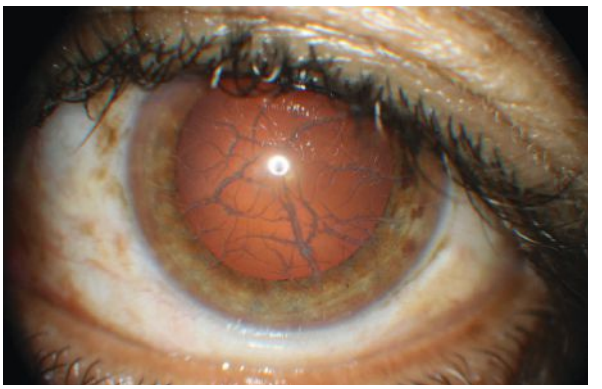
Genetic Test For Corneal Risk

New technology can help identify which patients are likely to develop post-LASIK dystrophies. **By Paul M. Karpecki, OD**

Over the years, we've become increasingly comfortable with the safety and predictable outcomes of modern refractive surgery. Indeed, many of the concerns we once had are less worrisome today because our screening protocols and pre-treatment routines have evolved. This is certainly true of our enhanced capacity to pretreat dry eye and ocular surface disease prior to laser-assisted *in situ* keratomileusis (LASIK) surgery. Doing so improves outcomes as well as the experience for the patient, the optometrist and the surgeon.



This patient's anterior segment shows clear signs of lattice dystrophy. This potential consequence of LASIK can be avoided with a proper evaluation that now includes genetic testing.

But ocular surface disease isn't the only thing we need to watch out for when examining refractive surgery candidates. We also need to diligently screen for corneal degenerations and dystrophies. These conditions contraindicate surgery due to the likelihood of progression as in the case of keratoconus or exacerbation as in the case of transforming growth factor beta-induced (TGFB-I) dystrophies. Yet, these can be difficult to identify based on family history and clinical exam alone.[1]

Fortunately, optometrists have newer tools at our disposal to help identify patients before it's too late. Furthermore, optometry's genetic screening strategies have recently evolved from testing five types of genetic mutations that result in corneal dystrophy to 70. This technology, the AvaGen (Avellino) test, identifies whether a patient has the genetic mutation responsible for type II granular corneal dystrophies (GCD2). The test uses a simple buccal or cheek swab that is sent to a laboratory for molecular testing. The results are then sent back to your office within 24 to 48 hours. This swab can establish more than 1,000 variants across 75 genes for keratoconus, allowing us to proactively diagnose corneal disease before symptoms ever appear.

Establishing Risk

In determining a patient's suitability for refractive surgery, AvaGen tests for keratoconus can be extremely useful.

If the swab tests positive, refractive surgery should not be considered, since it could lead to accelerated disease exacerbation characterized by bilateral opacity in the anterior corneal stroma, leading to a severe decrease in best-corrected visual acuity.[14]

One way to understand who might be at the greatest risk of developing more advanced disease is to understand the natural progression of keratoconus. In a systematic review and meta-analysis of 41 publications reporting various outcomes over 12 months on 11,529 untreated eyes with keratoconus, three factors were found to be statistically significantly associated with the risk of progression.[2]

The first significant factor was ethnicity.[2] When comparing the risk of progression between East Asians, Europeans and Middle Eastern populations, Middle Eastern populations showed a significantly greater risk, followed by Europeans and then East Asians.[2]

The second factor was age. Younger patients had more aggressive progression and steepening of Kmax at 12 months.[2] In fact, the authors predicted that for every 10-year increase in age they'd see 0.8D less Kmax steepening.[2] Their models predicted that patients younger than age 17 are likely to have more than 1.5D of Kmax progression at one year.[2]

Finally, the third factor associated with progression was a high Kmax at baseline.[2]

"I was only seeing light flashes early on, but light FLASHES when you've not seen anything for so many years—it was wonderful"

—Keith H, retinal prosthesis recipient

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Granular Corneal Dystrophy

Corneal dystrophies are bilateral, progressive, genetically determined, noninflammatory diseases restricted to the cornea.[1]

According to the 2008 IC3D classification system, five types of corneal dystrophies are caused by the mutation of TGFB-I gene.[3,4] These include lattice corneal dystrophy type I and variants, granular corneal dystrophy type I (GCD1), GCD2 (e.g., Avellino corneal dystrophy), Thiel-Behnke corneal dystrophy and Reis-Bückler corneal dystrophy.[3]

Initially, researchers knew of no distinction between types I and II GCD.[5] However, in 1988 investigators presented the histopathology of corneal buttons from four patients whose origins could be traced to the Italian province Avellino. They each underwent unilateral keratoplasty because of decreased vision.[6,7] These patients were clinically diagnosed as GCD2. The cause was found in a mutation of the TGFB-I gene.[8,9]

In patients with GCD2, vision slowly decreases with age as the central visual axis becomes progressively opaque and painful, recurrent epithelial erosions are common.[9,10]

GCD2 and Refractive Surgery

Due to the likelihood of recurrence and exacerbation, corneal dystrophies, including GCD2, contraindicate refractive surgery.[11] Many cases of post-laser surgery exacerbation have been reported worldwide since GCD2 was first identified.[1,3,5,12] But how can we most effectively determine the presence of disease in refractive surgery candidates?

Traditionally, the diagnosis and classification of corneal dystrophies were based on corneal signs and clinical symptoms.[13] However, depending on the precise mutation of the disease, patients can be clinically asymptomatic and "dystrophy-free" prior to LASIK, yet they can develop post-op deposition as a result of surgically-induced trauma.[1,11] Injury to the central cornea results in exacerbation of the GCD2 with dramatic acceleration of corneal deposition, opacities, and consequent visual loss.[3]

Because LASIK is strongly contraindicated in GCD2 and surgical candidates with the TGFB-I gene mutations may be asymptomatic, it has been proposed that preoperative genetic screening for TGFB-I mutations should be mandatory for refractive surgery candidates with risk factors.[3]

Since a lack of clinical signs does not mean the absence of corneal dystrophy-causing mutations and several reports highlight that LASIK treatment induces moderate-to-severe early exacerbations of GCD2 and a marked acceleration in disease course with rapidly worsening visual outcome, genetic testing is a welcome safeguard that can be easily added to a standard examination routine.[1,5] ■

Note: Dr. Karpecki consults for companies with products and services relevant to this topic.

This Luneau VX120 image shows the topography of an eye with keratoconus.

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Inflammation Swindler

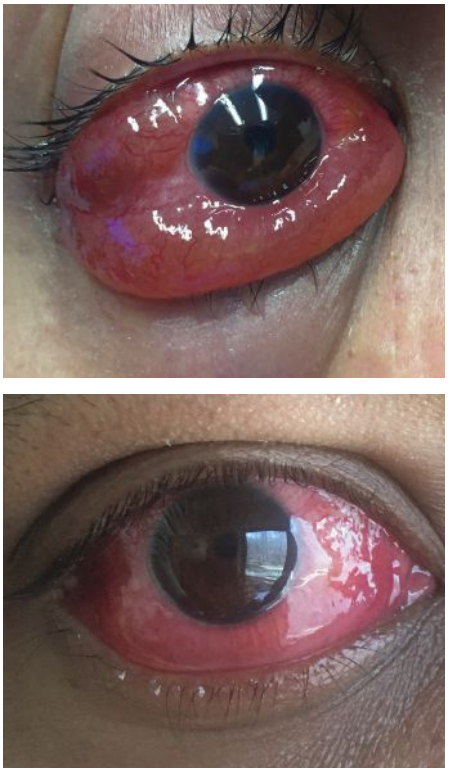
Despite being similar to other orbital diseases, diagnosing IOI requires a through neuro work-up. **By Michael Trottini, OD, and Michael DelGiodice, OD, PhD**

A 40-year-old male presented complaining of progressive swelling of his right eye. An ophthalmologist saw him one week prior and gave him unknown drops; however, his swelling continued to worsen. A different ophthalmologist then diagnosed him with epidemic keratoconjunctivits (EKC) and started him on Tobradex (tobramycin/dexamethasone, Alcon) drops and erythromycin ophthalmic ointment. His swelling progressed even further, which prompted him to go to the emergency department. He denied any double vision, pain or blurry vision.

On exam, his visual acuity was 20/25 OD and 20/20 OS. No afferent pupillary defect was noted and his intraocular pressures via Tonopen (Reichert) were 11mm Hg OD and 12mm Hg OS. There was marked swelling and chemosis of the right eye. There was lateral restriction of his ocular motility on the right eye, and, although he did not report diplopia during the initial history, he reported seeing two images when instructed to look to the right. Other than the swelling and reduced ocular motility, the rest of his exam was unremarkable. There was no retinal abnormality noted and no disc edema was present.

Guessing Game

The initial differential list included: idiopathic orbital inflammation (IOI), thyroid associated orbitopathy (TAO), orbital lymphoma, orbital cellulits, carotid cavernous fistula and EKC. His lack of pain made orbital cellulitis unlikely, and the degree of swelling made EKC unlikely; however, they were still considered in the initial differential list. A complete blood count (CBC) was ordered along with computed tomography CT of his orbits and CT angiography (CTA) of his brain. His CBC and CTA were unremarkable. The orbital CT demonstrated enlargement of the right lacrimal gland and right lateral rectus muscle with proptosis. There appeared to be swelling of both the belly and tendinous insertion of the lateral rectus.



One dose of prednisone led to significant improvement of the patient's injection and severe chemosis of the right eye.

After reviewing his imaging, orbital cellulitis, carotid cavernous fistula and EKC were excluded from the differentials. Additionally, because both the lateral rectus muscle belly and tendon were enlarged, TAO was unlikely to be the cause. Lastly, granulomatosis with polyangiitis and sarcoidosis can rarely affect the extraocular muscles (EOMs) and lacrimal gland, so serology studies for these disorders were ordered and were negative.

Diagnosis

The clinical presentation and CT findings were most consistent with IOI, and the patient was given 80mg of prednisone and instructed to follow up 24 hours later.

On exam the following day, there was significant improvement of the chemosis. Given the rapid response to just one dose of prednisone, IOI continued to seem the most likely diagnosis. The patient was instructed to continue 80mg of prednisone for three more days then cut to 60mg daily. On follow up one week later, he showed continued improvement of the eye swelling. The EOM restriction had resolved, and he did not notice the diplopia when looking to the right anymore. I slowly tapered the prednisone over the next six weeks, dropping 10mg each week.

On follow-up after he finished

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the prednisone taper, his clinical exam was completely normal with no proptosis, EOM restriction, chemosis or diplopia. As of his last exam, four months after finishing the prednisone, there was no recurrence.

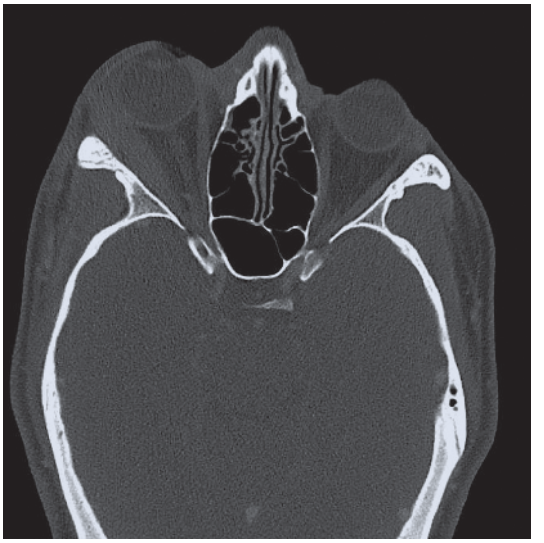
Discussion

IOI is a noninfectious, inflammatory disorder of the orbit without any identifiable local or systemic cause.[1-4] The syndrome can be categorized depending on which structure(s) are involved: myositis, dacryoadenitis, anterior, apical or diffuse inflammation.[3] Rarer types of IOI include periscleritis and perineuritis.[4]

Anterior IOI causes inflammation of the globe, conjunctiva and eyelids generally without proptosis, while diffuse IOI affects the same structures but tends to be more severe and more often seen with proptosis.[4] Myositis involves single or multiple EOMs where the medial rectus is most commonly affected, followed by the superior, lateral and inferior rectus muscles.[4]

Dacryoadenitis is the most common subtype of IOI and causes inflammation of the lacrimal gland. With apical IOI, inflammation of the orbital apex is seen, which can spread intracranially affecting the cavernous sinus and middle cranial fossa.[4] Lastly, periscleritis involves inflammation of the scleral, uvea or tenon's capsue. Perineuritis is defined by inflammation of the optic nerve sheath. This is in contrast to optic neuritis, which affects the axons of the nerve.[4]

In general, the presenting symptoms of IOI include pain, periorbital edema, chemosis, proptosis, diplopia and reduced vision. It is typically a unilateral disease with only rare bilateral involvement. Diagnosing IOI can be challenging, as there can be similarities between IOI, TAO and orbital lymphoma. Certain features, however, can help to distinguish them from one another. With IOI myositis, both the muscle belly and tendon tend to be inflamed, as seen in this patient, in contrast to TAO, where only the muscle belly is involved and the tendinous insertion is typically spared.



CT findings revealed swelling of right lateral rectus muscle and lacrimal gland, which helped narrow down the diagnosis to IOI.

Also, TAO most commonly affects the inferior followed by the medial and then the superior rectus. Isolated lateral rectus muscle involvement as seen in this patient is extremely rare for thyroid orbitopathy. Additionally, IOI is commonly unilateral where TAO is usually bilateral. Lastly, TAO is generally unresponsive to steroid therapy—which wasn't the case in treating this patient.

Orbital lymphoma can also be difficult to differentiate from IOI; however, this patient's dramatic response to just one dose of prednisone with no recurrence since stopping treatment makes that an unlikely scenario. A biopsy isn't indicated initially if the suspicion for IOI is high, but it is necessary if there is a higher suspicion for another etiology or if patients aren't improving on steroid treatment. Serology studies testing can also be helpful to role out orbital sarcoidosis and granulomatosis with polyangiitis.

Treatment

IOI treatment largely involves oral steroids, and more than 75% of patients show significant improvement within 24 to 48 hours.[4] An initial dose of 1mg/kg/day to 2mg/kg/day is recommended, and I almost always start with 80mg to 100mg per day. A slow taper, generally over six to eight weeks, is necessary to prevent a flare up of the inflammation. For patients who don't respond to oral steroids, radiotherapy, antimetabolies, T-cell inhibitors, alkylating agents, lymphocyte inhibitors and TNF-alpha inhibitors have all been reported as alternatives.[4]

IOI can be a difficult syndrome to diagnose, especially early in its course. Treatment is largely successful with oral steroids, and patients typically respond quickly. Because biopsy can be difficult to obtain when the inflammation is deeper within the orbit, it isn't always recommended initially. Differentiating IOI from other orbital disorders can take some experience, but certain radiologic, serologic and clinical differences aid the diagnosis. ■

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Edited By Derek N. Cunningham, OD, and Walter O. Whitley, OD, MBA

Anti-VEGF Injections: Start to Finish

Here's a step-by-step guide to how it's performed.

By Scott Bauer, BA, Christina Tran, BS, and Leonid Skorin Jr., DO, OD, MS

Intravitreal injections of anti-VEGF are FDA-approved for a number of ocular conditions and have been shown to decrease macular edema and improve vision.[1-5] The most common are aflibercept (Eylea), ranibizumab (Lucentis) and bevacizumab (Avastin).

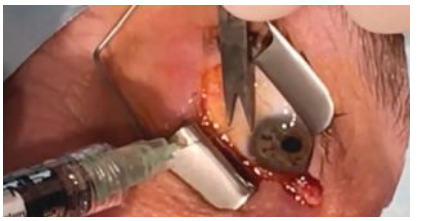
Aflibercept acts as a decoy receptor that binds to and inhibits the activation of VEGF receptors on endothelial cells. It can treat wet AMD, macular edema from vein occlusion, diabetic macular edema and diabetic retinopathy. Ranibizumab binds to active VEGF and prevents interaction with receptors on endothelial cells. The drug treats the same conditions as aflibercept and is also indicated for intravitreal use in myopic choroidal neovascularization. While bevacizumab's use is considered off-label, the drug has achieved similar success rates as aflibercept and ranibizumab.[6]

The Process

Though optometrists cannot perform these injections, it's helpful to understand it for comanagement purposes.

On the day of the procedure, the patient's visual acuities and intraocular pressures will be measured and the eye receiving treatment will be noted by marking the forehead. The patient will put on a hair net and be put in the supine position. A lid speculum will be inserted to prevent blinking. The patient will be instructed to look up and away and be still.

For a video of this procedure, visit www.reviewofoptometry.com or scan the QR code.



A patient undergoes an anti-VEGF injection.

The injection site is inferotemporal to the limbus due to its ease of accessibility. Using a caliper, the retina specialist administering the injection will measure the distance between the two; it should be 4.0mm in phakic eyes to prevent inadvertent puncture of the natural lens and 3.5mm in aphakic and pseudophakic eyes. The injection site will be prepared using topical proparacaine 0.5%, povidone-iodine 5.0% and lidocaine hydrochloride 3.5%; povidone-iodine swab sticks will be used to sterilize the eyelids and surrounding tissue.

The patient should not speak, cough or sneeze, as streptococcal isolates in saliva are three times more likely to cause endophthalmitis post-intravitreal injection than post-intraocular surgery.[7] Everyone in the room should be wearing a mask.

One drop of povidone-iodine 5.0% will be instilled directly onto the injection site, then the surgeon will insert the needle perpendicular to the sclera and through the underlying pars plana, and inject the medication into the vitreous chamber. The patient's gross visual acuity will be checked to ensure the increase in vitreous chamber volume did not cause a significant decrease in retinal profusion. The patient should be able to accurately count fingers within seconds of the injection. After the speculum is removed, the eyelid and eye will be rinsed with saline to remove any residual povidone-iodine to prevent superficial punctate keratitis.

Intravitreal injections may cause a patient to see a black spot due to an air bubble in the medication. This is not serious, and the air bubble should reabsorb in about a week. A subconjunctival hemorrhage may occur but should also resolve spontaneously and without complication in a week. Significant side effects following intravitreal injections are rare.[8] The patient should avoid dusty environments and eye-rubbing and return to the doctor if they experience decreased vision, pain and redness or flashes and floaters. ■

Mr. Bauer and Ms. Tran are fourth-year optometry students at Pacific University in Forest Grove, OR.

Dr. Skorin is a consultant in the Department of Surgery, Community Division of Ophthalmology in the Mayo Clinic Health System in Albert Lea, MN.

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Eyelid Health

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Bruder now offers a handy kit that includes an entire home care regimen for patients, the company says.

Each kit includes the Moist Heat Eye Compress mask, a 10-pack of cleaning sheets, a 30-pack of eyelid wipes and a bottle of antimicrobial spray solution.

Packaging all the necessary items for an effective lid care regimen in one kit may help improve patient compliance and motivation for lid hygiene, Bruder says.

Diagnostics

Headset Simplifies Dark Adaptation Testing

If you want to help identify AMD patients sooner so that management can begin before substantial vision loss occurs, a new product from Maculogix aims to help. The company's AdaptDx Pro device is a headset worn by patients for impaired dark adaptation screening, which Maculogix describes as the earliest biomarker of AMD pathology.

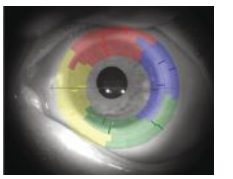
The new product improves upon its previous generation by addressing several logistical hurdles that can limit patient and practitioner motivation for the test. Practices no longer have to tie up a room and a tech while the test is performed—the head-mounted device uses light-blocking eyepieces that allow the patient to be seated anywhere—and the product uses a digital assistant, named Theia, to guide the patient during the test. The device monitors patient fixation and prompts the patient if they exhibit signs of inattention. The result, Maculogix says, is greater independence for the office staff administering the test.

Soon to follow the launch of AdaptDx Pro will be a software dashboard called Meye Practice that pulls data from the practice's EHR to help clinicians and administrators track the clinical and financial performance of AMD care in the practice, according to Maculogix.

Existing customers who own the original AdaptDx will have the first opportunity to update to the Pro device at a discount, the company says.

Company Aims to Simplify Scleral Lens Design

If you fit scleral contact lenses, you may be interested in two new features for the Eye Surface Profiler from Eaglet Eye. The topographer already maps both the cornea and sclera. Now, a new feature called Scleral Profiler allows practitioners to quickly and easily determine if they should pursue a spherical, toric, quad or fully custom scleral periphery, the company says. The second new feature is the ability to enter the over-refraction to allow for more seamless interaction with the lab of choice, to help reduce complications when ordering sclerals.



Vision Care

New Myopia Management Starter Program

Clinicians ready to dive into myopia management but unsure where to start can consider a new comprehensive approach developed by CooperVision.

The Brilliant Futures program promotes what the company touts as a new standard of care for children with myopia. Patients and parents will receive educational materials about myopia and their prescribed therapy via a mobile app, as well as free product shipping and returns for MiSight 1 day lenses. Doctors will receive online clinical education, specialist support and a myopia management toolbox that includes patient and parent education resources, staff training tools, practice integration support and community outreach and engagement resources, according to CooperVision.

Keep Low Vision Care Close to Home

For your low vision patients, instead of referring them out right away, consider a new lens product line from EnChroma called the Lx Series intended to help by reducing the effects of glare, improving contrast and enhancing color vision, the company says.

A trial kit includes: a category 1 lens (80% transmission) for low-light and night-time use, category 2 and 3 lenses (12% and 36% transmission, respectively) for combined glare reduction and color vision enhancement and an ultra-dark category 4 lens (3% transmission) for extreme glare management.

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■ **4-8.** *Art & Science of Optometric Care: A Behavioral Perspective*. Timonium, MD. Hosted by: OEPF. Key faculty: Virginia Donati. CE hours: 35. For more information, contact Karen Ruder at 410-561-3791, karen.ruder@oepf.org or go to www.oepf.org.

■ **4-8.** *SECO*. Atlanta. Hosted by: SECO International. Key faculty: Jack Parker, Mark Dunbar, Harry Quigley, Tammy Than, Danica Marrelli, Charlie Sacarelli. CE hours: total: 400, max. per OD: 54. For more information, contact Elizabeth Taylor at 770-451-8206, info@secostaff.com or go to www.attendseco.com.

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■ **7.** *AZ-AAO Spring Meeting*. Glendale, AZ. Hosted by: Arizona Chapter–AAO. CE hours: 6. For more information, contact Florencia Yeh at arizona.aaopt@gmail.com or go to www.aaopt.org/membership/us-and-international-chapters/azchapter.

■ **7-8.** *Continuing Education Seminar*. Vaughan, Ontario, Canada. Hosted by: Academy of Ophthalmic Education. Key faculty: Carolyn Majcher, Rich Trevino, Melissa Vitek, Brian Rogoff. CE hours: 14. For more information, contact Claudia Marks at 905-731-6022, cmarks@aoece.com or go to www.aoece.com.

■ **7-8.** *IOA Winter CE Series*. Alsip, IL. Hosted by: Illinois Optometric Association. Key faculty: Blair Lonsberry. CE hours: 6 TQ. For more information, contact Katie Lewis at 217-525-8012, katie@ioaweb.org or go to illinois.aoa.org.

■ **8.** *MOA CEXpress*. Detroit, MI. Hosted by: Michigan Optometric Assn. Key faculty: Chris Cakanac. CE hours: 7. For more information, contact MOA at 517-482-0616, info@themoa.org or go to www.themoa.org.

■ **8-9.** *Great Lakes Optometric Congress*. Northbrook, IL. Hosted by: OEP Foundation/Kovach Eye Institute. Key faculty: Cathy Stern, Glenn Steele. CE hours: 13. For more information, contact John A. Loesch at 708-917-5353 or drjohnod1@gmail.com.

■ **10.** *PECAA Selling Strategies for Success Workshop*. San Diego. Hosted by: Professional Eye Care Associates of America. Key faculty: Doug Martin, Samantha Toth. CE hours: 3 COPE, 3 ABO, 3 NCLE, 3 CPC. For more information, contact Cathi Zerba at 503-670-9200 or cathi@pecaa.com.

■ **11-15.** *Ocular Therapeutics in Cancun*. Cancun, Mexico. Hosted by: Ocular Therapeutics CE. Key faculty: Tony Litwak, Jim Thimons, Eric Conley. CE hours: 20. For more information, contact Tony Litwak at 443-895-1682, info@otce.net or go to www.otce.net.

■ **12-13.** *SUNY Residents Day*. New York, NY. Hosted by: SUNY College of Optometry. Key faculty: SUNY residents. CE hours: total: 14, max.: 7 per day. For more information, contact Betsy Torres at 212-938-5830, ce@sunyopt.edu or go to www.sunyopt.edu/cpe.

■ **12-14.** *2020 OAOP Vision Summit*. Norman, OK. Hosted by: Oklahoma Association of Optometric Physicians. Key faculty: Peter Cass, Sharon Carter, Joe DeLoach, Lynn Lawrence, John Rumpakis. CE hours: total 29, max. per OD: 19. For more information, contact Heatherlyn Burton at 405-524-1075, heatherlyn@oaop.org or go to www.oaop.org.

■ **13.** *Binocular Vision and Pediatrics Forum*. Columbus, OH. Hosted by: Ohio State College of Optometry. Key faculty: Katherine Weise. CE hours: 7. For more information, contact Denise Turner at 614-292-4451, turner.1545@osu.edu or go to optometry.osu.edu/continuing-education.

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■ **15.** *Dynamic Duos*. Elkins Park, PA. Hosted by: Salus University. Key faculty: Clark Chang, Stanley Hatch, Erin Jenewein, Chad Killen, Tracy Offerdahl-McGowan, Carlo Pelino. CE hours: 8. For more information, contact Natalie Standig at 215-780-1381, nstandig@salus.edu or go to www.salus.edu/events.

■ **15-16.** *Netherlands Contact Lens Congress*. Veldhoven, Netherlands. Hosted by: ANVC/NAC. CE hours: total 48, max. per OD: 16. For more information, contact Hans Kloes at hans@kloes.nl or go to www.ncc2020.com.

■ **20-22.** *SCOPA 2020 Spring Meeting*. Greenville, SC. Hosted by: South Carolina Optometric Physicians Association. For more information, contact Jackie Rivers at jrivers@sceyedoctors.com or go to www.sceyedoctors.com.

■ **21-22.** *Massachusetts Education Conference*. Springfield, MA. Hosted by: Mass. Society of Optometrists. Key faculty: Jerome Sherman, Joe DeLoach, Larry Oxenham. CE hours: 16. For more information, contact Jessica Bertrand at 508-875-7900, jessica@maoptometry.org or go to maoptometry.org.

■ **21-22.** *Ocular Disease: Part One*. Fullerton, CA. Hosted by: MBKU-SCCO. CE hours: 16. For more information, contact Bonnie Dellatorre at 714-449-7495, ce@ketchum.edu or go to www.ketchum.edu/ce.

■ **22.** *Vancouver Specialty Lens Symposium*. Vancouver, BC, Canada. Hosted by: Pacific U. College of Optometry. Key faculty: Matt Lampa, Beth Kinoshita, Mari Fujimoto, Pat Caroline, Mark Andre, Randy Kojima. CE hours: 7. For more information, contact Miki Buckingham at 503-352-2985, mikibuckingham@pacificu.edu or go to www.pacificu.edu/academics/colleges/college-optometry/continuing-education.

■ **26-28.** *Nebraska Primary EyeCare*. Lincoln, NE. Hosted by: Nebraska Optometric Assn. CE hours: 16. For more information, contact Emily Wilcox at 402-476-7716, ewilcox@assocoffice.net or go to nebraska.aoa.org/education-and-training/spring-conference.

■ **26-29.** *Conference for Comprehensive Eyecare*. Niagara Falls, NY. Hosted by: PSS EyeCare. Key faculty: Ron Melton, Randall Thomas, Jerome Sherman. CE hours: 18. For more information, contact Sonia Kumari at 203-415-3087, education@psseyecare.com or go to www.psseyecare.com.

■ **26-29.** *Vision Expo East*. New York, NY. Hosted by: Reed Exhibitions/Vision Council. Key faculty: Mark Wright, Stephanie Woo, Murray Fingeret, Joseph Pizzimenti, Paul Chous, Whitney Hauser. CE hours: total 320, max. per OD: 28. For more information, contact Julia Moore at 703-740-2248, jmoore@thevisioncouncil.org or go to east.visionexpo.com.

■ **27-28.** *UOA Annual Spring Conference*. St. George, UT. Hosted by: Utah Optometric Association. CE hours: 6. For more information, contact Alyssa White at 801-364-9103, alyssa@utaheyedoc.org or go to www.utaheyedoc.org.

■ **27-29.** *IOA 2020 Annual Congress*. Des Moines, IA. Hosted by: Iowa Optometric Association. CE hours: 14. For more information, contact Jill Gonder at 515-222-5679x3, jillg@iowaoptometry.org or go to www.iowaoptometry.org.

■ **29.** *Los Angeles County Optometric Society Winter CE*. Los Angeles. Hosted by: Los Angeles County Optometric Society. CE hours: 5. For more information, contact Gary D. Polan at 310-459-0055, dr.polan@verizon.net or go to www.lacos.net.

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Diagnostic **Quiz**

Ring in the New Year

A patient asked for new glasses, but doctors noticed another problem.

By Andrew S. Gurwood, OD

History

A 27-year-old Caucasian female reported to the office with a chief complaint of "needing new glasses." She had no other ocular or systemic issues, took no medications, was in good health and denied allergies of any kind.

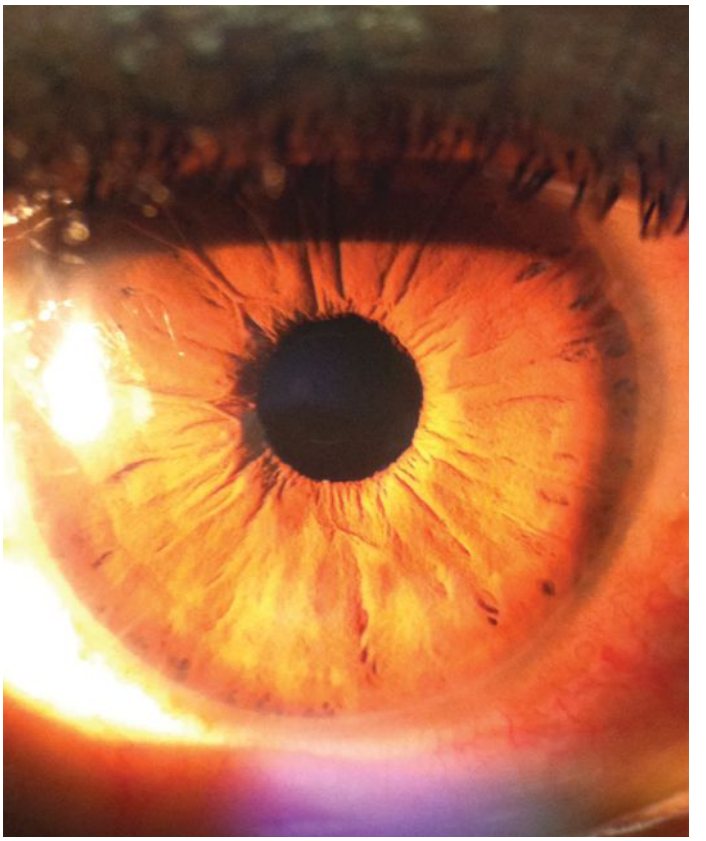
Diagnostic Data

Her best-corrected entering visual acuities were 20/20 OD and 20/25 OS at distance and near. External examination was normal and no evidence of afferent pupillary defect was seen. Refraction corrected the issue, yielding improved vision at distance of 20/20 in both eyes.

Biomicroscopy of the anterior segment uncovered unusual ring-shaped opacities in both peripheral corneas (the pertinent clinical findings are demonstrated in photograph). Goldmann applanation tonometry measured 15mm Hg OU. Peripheral pathologies were seen OU.

Your Diagnosis

Does the case presented require any additional tests, history or information? Based on the information provided, what would be your diagnosis? What is the patient's most likely prognosis? To find out, please visit us online at www.reviewofoptometry.com. ■



This photo of the patient's anterior presentation shows her unusual ring-shaped opacities in the peripheral cornea.

Next Month in the Mag

Coming in March, *Review of Optometry* will present its 13th Annual Pharmaceuticals Report.

Topics include:

- *Steroid Wars: Will New Drugs Challenge Old Habits?*
- *How—and Why—to Choose a Dry Eye Drug*
- *When to Change Up Your Glaucoma Regimen*
- *The Do's and Don'ts of Oral Medication Use* (Earn 2 CE Credits)

Also in this issue:

- *Can You Meet the Demands of Today's Cataract Patient?*
- *Use Anterior Segment OCT for Better Contact Lens Fits*

REVIEW OF OPTOMETRY (ISSN 0147-7633) IS PUBLISHED MONTHLY, 12 TIMES A YEAR BY JOBSON MEDICAL INFORMATION LLC,395 HUDSON STREET, 3RD FLOOR FLOOR, NEW YORK, NY 10014. PERIODICALS POSTAGE PAID AT NEW YORK, NY AND ADDITIONAL MAILING OFFICES. POSTMASTER: SEND ADDRESS CHANGES TO REVIEW OF OPTOMETRY, PO BOX 81, CONGERS, NY 10920-0081. SUBSCRIPTION PRICES: US: ONE YEAR $56; TWO YEARS $97, CANADA: ONE YEAR $88, TWO YEARS $160, INT'L: ONE YEAR $209, TWO YEARS $299. FOR SUBSCRIPTION INFORMATION CALL TOLL-FREE (877) 529-1746 (USA); OUTSIDE USA, CALL (845) 267-3065. OR EMAIL US AT REVOPTOMETRY@CAMBEYWEST.COM. PUBLICATIONS MAIL AGREEMENT NO: 40612608. CANADA RETURNS TO BE SENT TO BLEUCHIP INTERNATIONAL, P.O. BOX 25542, LONDON, ON N6C 6B2.

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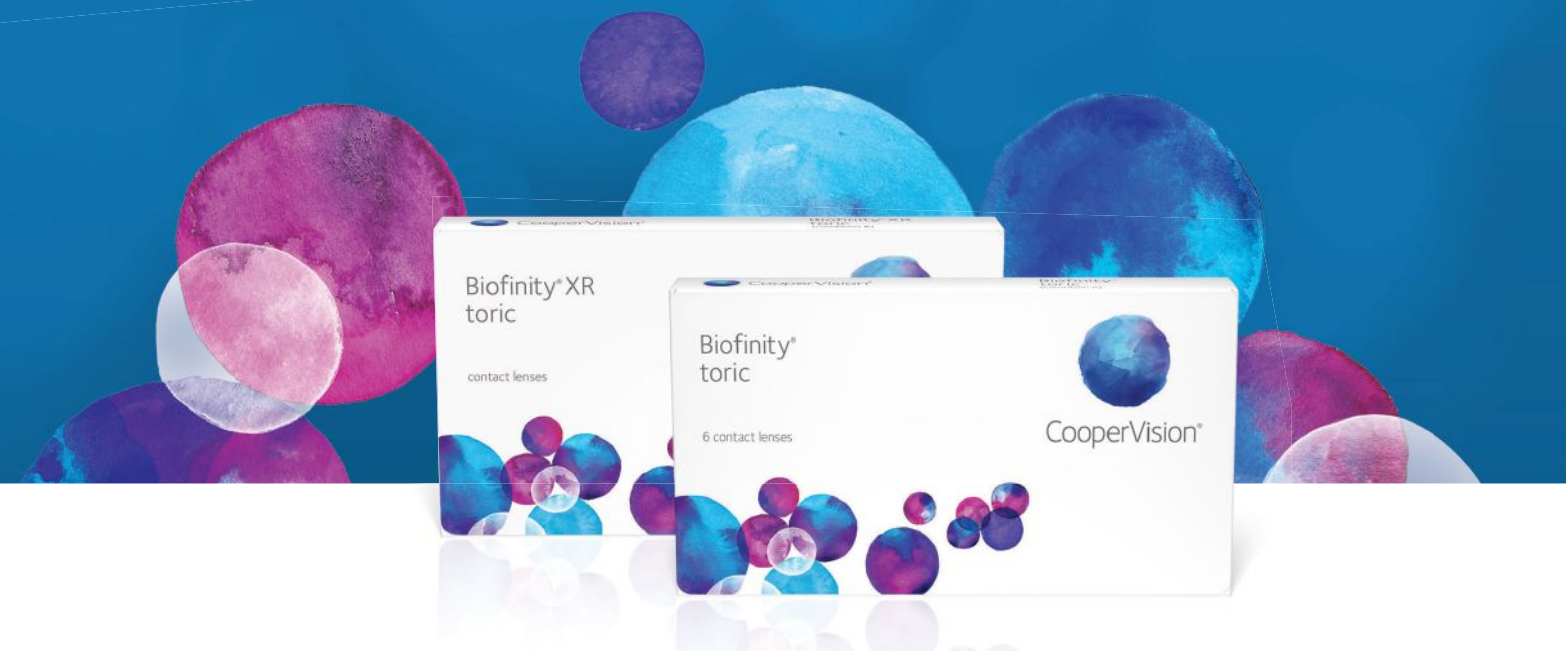

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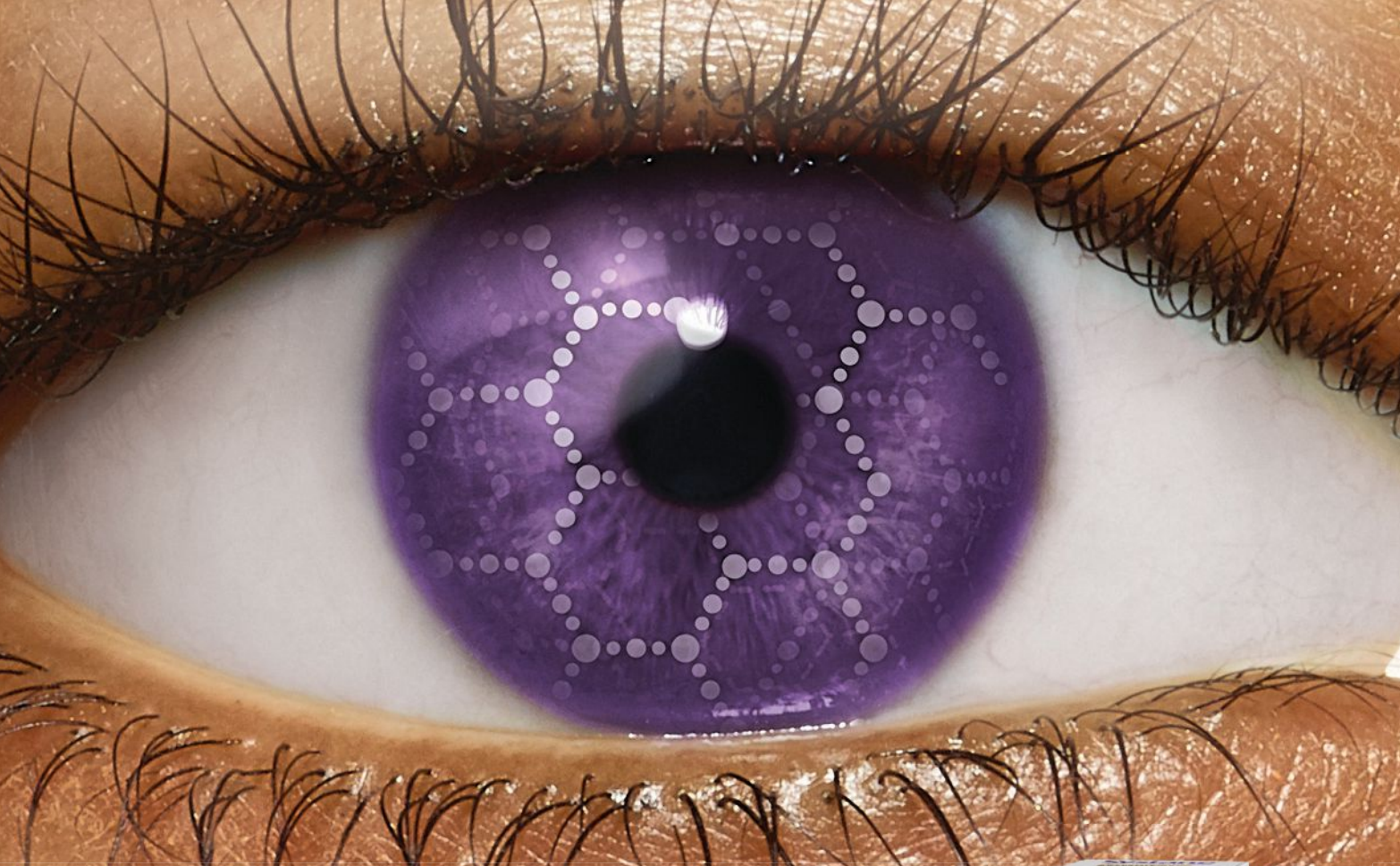
1. US industry data on file. **2**. The results of an online survey involving patients who wear Biofinity® toric contact lenses. January 2018 Biofinity® toric wearer online survey. Data on file.



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