EARN 2 CE CREDITS: When Drugs Cause Dry Eye, p. 91

REVIEW® OF OPTOMETRY

October 15, 2019

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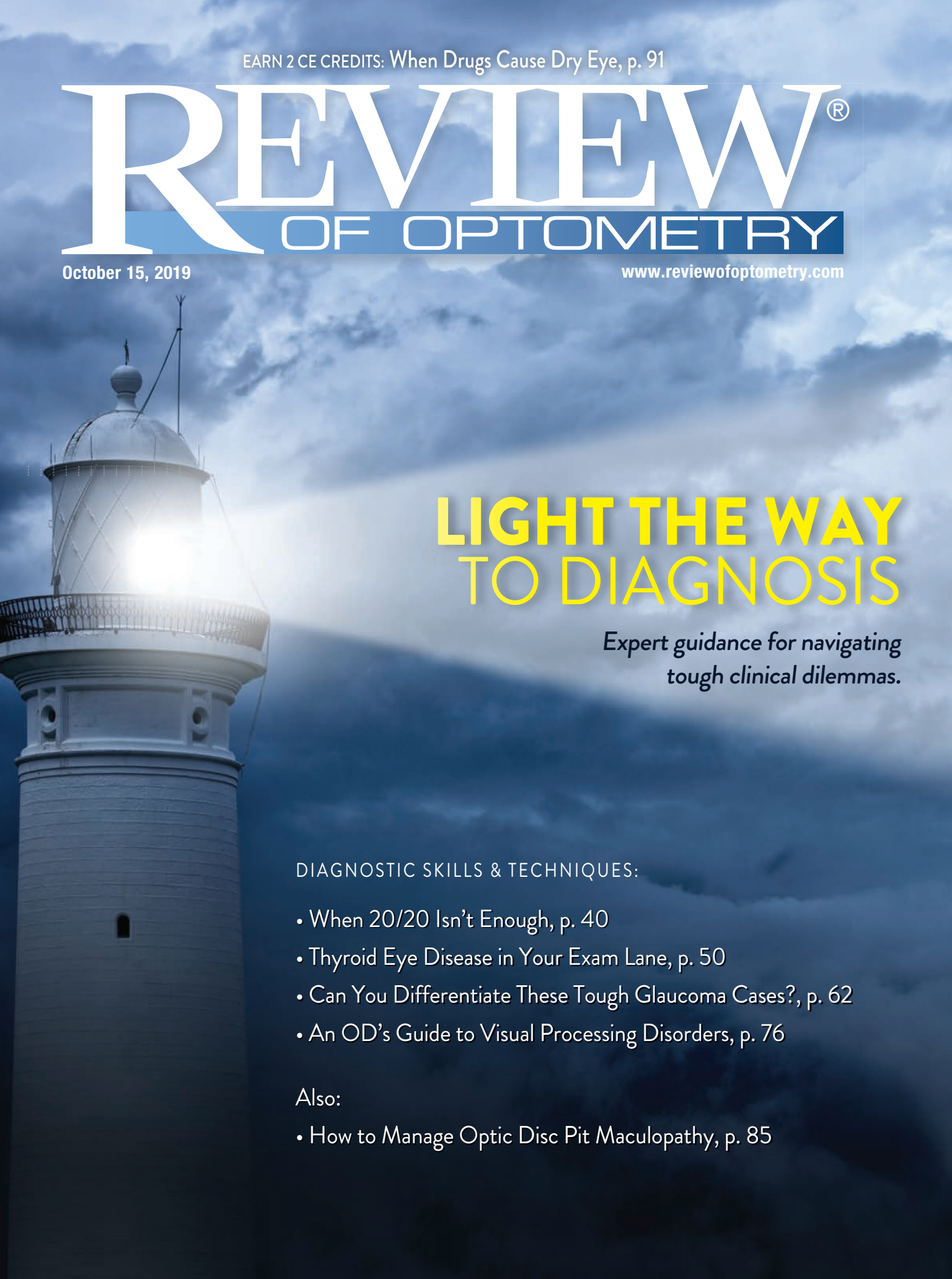

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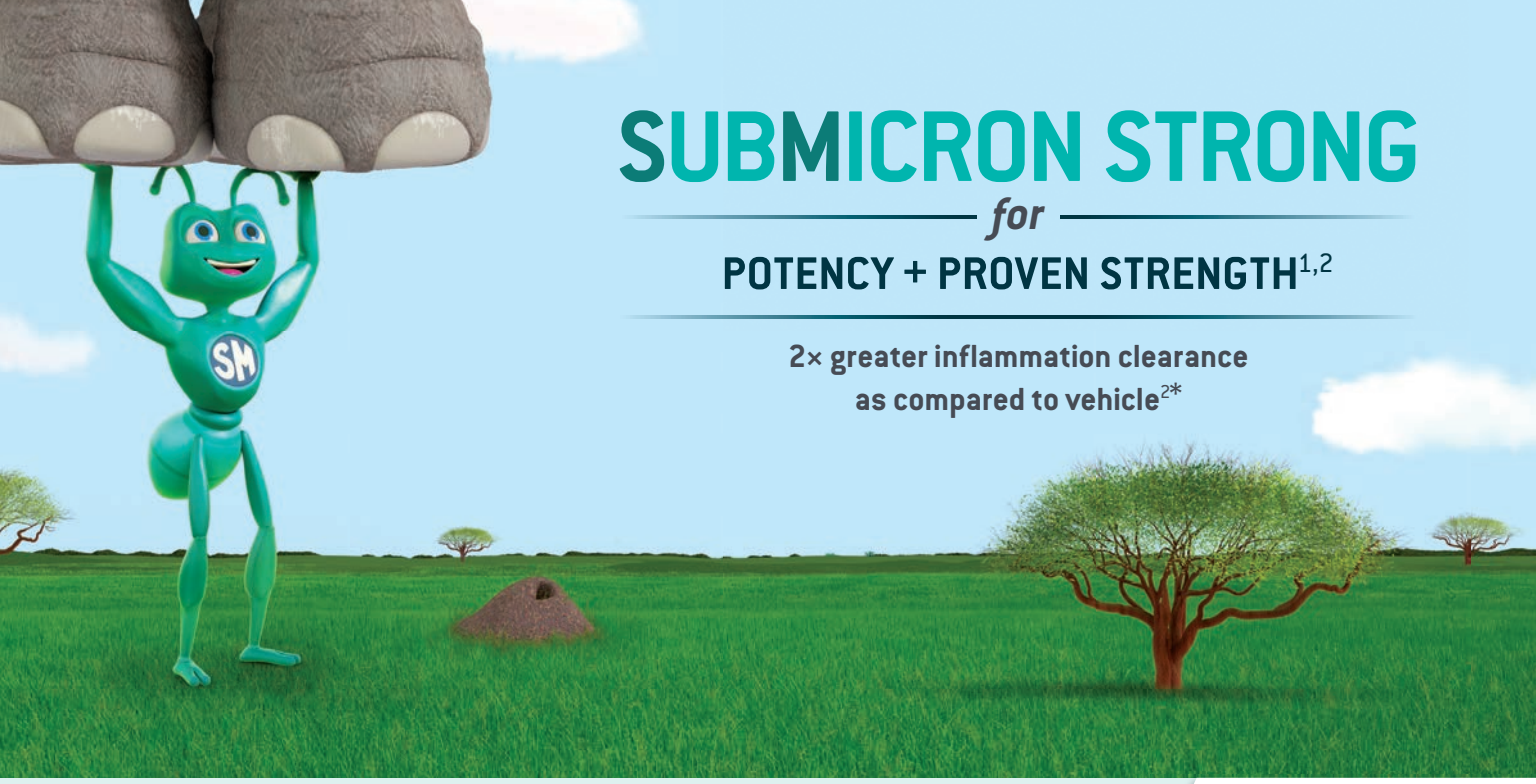

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for

POTENCY + PROVEN STRENGTH[1,2]

2× greater inflammation clearance as compared to vehicle[2]*

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- **Engineered with SM Technology™ for efficient penetration at a low BAK level (0.003%)[1,3]**
- **~2× greater penetration** to the aqueous humor than LOTEMAX® GEL (loteprednol etabonate ophthalmic gel) 0.5%[3]

Clinical significance of these preclinical data has not been established.

LOTEMAX® SM
(loteprednol etabonate ophthalmic gel) 0.38%

SMALL & MIGHTY
SUBMICRON PARTICLES

*PROVEN STRENGTH

- **30% of LOTEMAX® SM patients had complete ACC resolution** vs vehicle (15%) at Day 8 (N=371, *P*<0.0001)[1,2†]
- **74% of LOTEMAX® SM patients were completely pain-free** vs vehicle (49%) at Day 8 (N=371, *P*<0.0001)[1,2‡]

†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.

Indication

LOTEMAX® SM (loteprednol etabonate ophthalmic gel) 0.38% is a corticosteroid indicated for the treatment of post-operative inflammation and pain following ocular surgery.

Important Safety Information

- 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, and also in mycobacterial infection of the eye and fungal diseases of ocular structures.
- 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 LOTEMAX® SM is used for 10 days or longer, IOP should be monitored.
- Use of corticosteroids may result in posterior subcapsular cataract formation.

Important Safety Information (cont.)

- 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
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Based on 9669600-9669700 Revised: 02/2019

VOL. 156 NO. 10 ■ OCTOBER 15, 2019

IN THE NEWS

Comparing three visual field testing methods: standard (SS), Swedish interactive thresholding algorithm and faster (SFR), researchers in Australia found that, **although SFR can save significant time compared with SS, 29.3% of its results were unreliable. Using the SS method, only 7.7% were unreliable.** SFR also resulted in higher sensitivity values (on average, 0.5dB for glaucoma patients), which was greater in the case of field loss (<19.0dB).

Phu J, Khuu S, Agar A, Kalloniatis M. Clinical evaluation of SITA-Faster compared to SITA-Standard in normal subjects, glaucoma suspects and glaucoma patients. Am J Ophthalmol. August 27, 2019. [Epub ahead of print].

Researchers found both **swept-source anterior segment OCT and traditional gonioscopy provided different anterior chamber dimensions, as well as false positive angle-closure diagnoses based on the OCT readings**. The researchers noted a deeper anterior chamber depth and a lower lens vault were significantly associated with false positives on OCT.

Porporato N, Baskaran M, Tun TA, et al. Understanding diagnostic disagreement in angle closure assessment between anterior segment optical coherence tomography and gonioscopy. Br J Ophthalmol. September 6, 2019. [Epub ahead of print].

From 2,992 OCT images from glaucoma patients and glaucoma suspects, a Johns Hopkins–based research team found that **poor signal strength lowers the reliability of OCT scans**. Anything below three had a significant impact on reliability. **For patients with severe glaucoma, decreases in signal strength between 10 and three had a large impact on reliability compared with mild or moderate glaucoma patients.**

Yohannan J, Cheng M, Da J, et al. Evidence based criteria for determining peripapillary OCT reliability. Ophthalmology. August 29, 2019. [Epub ahead of print].

Pseudoexfoliation Can Alter Pupils

Research identifies metrics that can help anticipate glaucoma onset.

By Bill Kekevian, Senior Editor

The accumulation of pseudoexfoliative material can alter both static and dynamic pupillary characteristics, according to investigators. Additionally, their study found a reduced amplitude of pupil contraction is associated with progression from pseudoexfoliation syndrome (PES) to pseudoexfoliative glaucoma (PEG).

The team looked at 40 patients with PES, another 30 with PEG and 43 control subjects. They took static pupillometry measurements such as scotopic, mesopic, low photopic and high photopic pupil diameter, as well as dynamic measurements that included resting diameter and amplitude of pupil contraction. Other measurements included the latency, duration and velocity of pupil contraction and pupil dilation. These measurements, obtained using an automatic quantitative pupillometry system, were then compared between the groups.

The research shows that scotopic, mesopic and low photopic pupil diameter values are significantly lower in patients with PES and PEG compared with controls and that the parameters between patients with PES and those with PEG were similar. However, the amplitude of pupil contraction values of patients with PEG was lower than patients with PES and controls. The PES patients had significantly lower amplitude of pupil contraction values compared with controls, and the velocity of pupil contraction was higher in controls.

"Clinical implications of using automatic quantitative pupillometry is limited at this time, as the technology was unable to differentiate PES from PEG," notes Brian D. Fisher, OD, an attending optometrist at The Villages VA Outpatient Clinic in Florida. "Furthermore, most PEG patients will be on some form of topical hypotensive therapy, and these medications have been shown to alter pupillary size, further complicating the use of automatic quantitative pupillometry."

Currently, most clinicians use OCT and visual fields to assess PES and PEG patients, a practice that won't change until further research examines the comparability between these tools and automatic quantitative pupillometry, according to Dr. Fisher.

Tekin K, Kiziltoprak H, Sekeroglu M, et al. Static and dynamic pupil characteristics in pseudoexfoliation syndrome and glaucoma. Clin Exp Optom. July 30, 2019. [Epub ahead of print].

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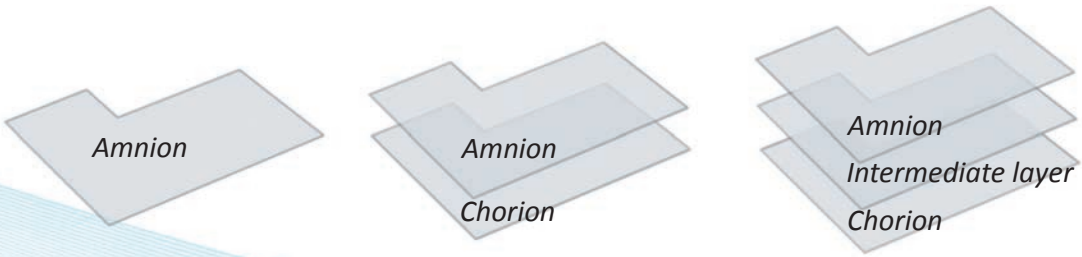

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Vaping Linked to Worse Ocular Surface Disease

E-cigarettes can make dry eye more painful and the tear film more unstable.

With a series of hospitalizations and even a few deaths linked to vaping, the use of e-cigarettes is under renewed scrutiny.[1] The Centers for Disease Control and Prevention point to approximately 530 cases of acute lung injury across 38 states and one US territory that often involved a life-threatening build up of fluid in the lungs.[1] The epidemic has even led to a federal criminal investigation and prompted the CEO of one of the major manufacturers of e-cigarettes—Juul—to step down.[2,3] But, new research is showing the harm these trendy nicotine delivery devices can inflict goes beyond the respiratory system. Vaping can aggravate the ocular surface as well and is directly connected with moderate-to-severe dry eye symptoms, according to a study published in *Optometry and Vision Science*.[4]

According to the researchers, hazardous byproducts from pyrolysis of the liquid constituents can both instigate and worsen dry eye symptoms. The team looked at 21 patients who used e-cigarettes and 21 healthy nonsmokers who were

E-cigarette use has been connected with moderate-to-severe dry eye symptoms.

all evaluated using noninvasive tear break-up time, fluorescein break-up time, ocular surface staining, tear meniscus height, Schirmer testing and the ocular surface disease index (OSDI) survey. As an additional measure, the researchers documented the effect of voltage degree used during vaping.

The OSDI scores show those who vape experience symptoms of moderate-to-severe eye dryness at a higher rate than their nonsmoking counterparts. Vapers also experienced significant reductions of noninvasive tear break-up time, fluorescein break-up time and tear meniscus height compared with nonsmokers. Schirmer tests showed higher results in those who also used vaping products.

As a secondary finding, the researchers found that, even amongst vapers, those who use a greater voltage aggravated their dry eye symptoms and tear instability more than those who used lower voltage—so Schirmer test results increased with voltage as well.[4]

"Investigation of the ocular surface health at cellular and molecular levels is warranted to gain a deeper understanding of the effect of e-cigarettes on the eyes," the team concluded.[4]

1. CDC. Outbreak of lung injury associated with e-cigarette use, or vaping. Smoking & Tobacco Use. www.cdc.gov/tobacco/basic_information/e-cigarettes/severe-lung-disease.html#latest-outbreak-information. September 19, 2019. Accessed September 25, 2019.
2. CDC. Transcript of CDC telebriefing: update on lung injury associated with e-cigarette product use, or vaping. CDC Newsroom. www.cdc.gov/media/releases/2019/t0919-lung-inury-vaping.html. September 19, 2019. Accessed September 25, 2019.
3. Juul Labs. Juul Labs names new leadership, outlines changes to police and marketing efforts. Company News. newsroom.juul.com/2019/09/25/juul-labs-names-new-leadership-outlines-changes-to-policy-and-marketing-efforts/. September 25, 2019. Accessed September 25, 2019.
4. Md Isa NA, Koh PY, Doraj P. The tear function in electronic cigarette smokers. Optom Vis Sci. 2019;96(9):678-85.

Exhaust Exposure Doubles AMD Risk

New findings show traffic-related air pollution can wreak havoc on the retina. Using insurance databases, researchers in Taiwan followed 39,819 subjects age 50 or older, all of whom had no signs of age-related macular degeneration (AMD) at baseline and lived in areas that had air-quality monitoring stations.

During 11 years of follow-up, 1,442 developed the condition. After dividing those patients by their level of exposure to traffic pollution, those with the most exposure were 84% more likely to develop AMD than those with the least exposure. And this was after adjusting for other potential factors, such as age, gender, household income and underlying illnesses.

While an intriguing finding, the researchers admit the observational nature of the study can only provide an association, not causation.

Chang K, Hsu P, Lin C, et al. Traffic-related air pollutants increase the risk for age-related macular degeneration. J Invest Med. 2019;67:1076-81.

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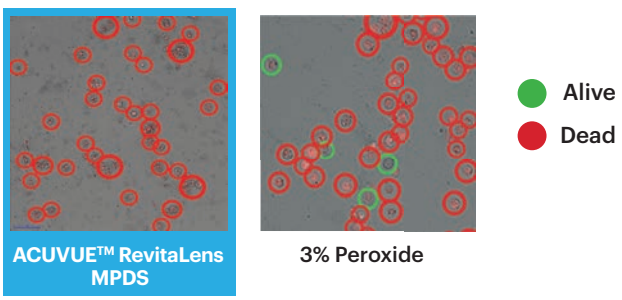
Several in-vitro studies suggest ACUVUE™ RevitaLens MPDS performs favorably vs. leading multi-purpose solutions. However, there is no standardized method for testing the efficacy of lens care solutions against Acanthamoeba.[1]

6 hours live-cell imaging of Acanthamoeba castellanii trophozoites.[1]*



ACUVUE™ RevitaLens MPDS

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* In an in-vitro study between competitive brands and ACUVUE™ RevitaLens MPDS, time-lapse measurements were taken to accurately document the time course for eradication of Acanthamoeba castellanii trophozoites. The live-cell methodology visually demonstrates the efficacy of each of the contact lens solutions in eradicating Acanthamoeba castellanii trophozoites.

[1]JJV Data on File 2018. ACUVUE RevitaLens Multipurpose Disinfecting Solution Packaging Claims

ACUVUE™[®] RevitaLens Multi-Purpose Disinfecting Solution is indicated for the care of soft (hydrophilic) contact lenses, including silicone hydrogel lenses. Use this product as directed in the product carton to disinfect, clean, rinse, store, remove protein and condition contact lenses. Do not use this product if allergic to any ingredient in ACUVUE™ RevitaLens MPDS. Problems with contact lenses and lens care products could result in corneal infection and/or ulcers and lead to loss of vision. It is essential that patients follow the directions and labeling instructions for proper use of lenses and lens care products, including the lens case.

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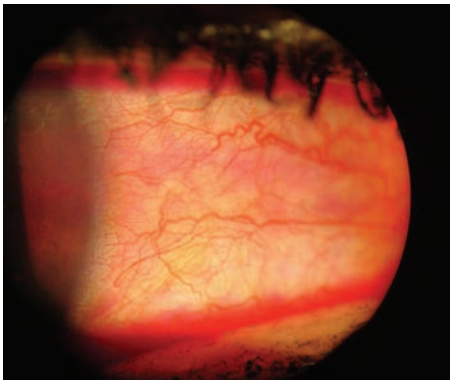
Rose Bengal Bests Infectious Keratitis

The popular stain is also effective against many microbial invaders.

Optometrists are familiar with rose bengal's clinical value in diagnosing ocular surface disease by staining dead corneal and conjunctival cells. Turns out, it also has a potential role to play in treating infections. The procedure, called rose bengal photodynamic antimicrobial therapy (RB-PDAT), can be considered an appropriate adjunct treatment for severe, progressive infectious keratitis, according to researchers from the University of Miami Miller School of Medicine.



Patients who underwent RB-PDAT were considered successful if they avoided therapeutic keratoplasty.

The investigation evaluated 18 patients—seven males and 11 females ranging from 17 to 83 years old—with active microbiologic infections. Those with a progressive infectious keratitis who did not respond to standard medical therapy underwent RB-PDAT at the Bascom Palmer Eye Institute from January 2016 through March 2018. Of the subjects, 10 had *Acanthamoeba*, four had *Fusarium spp*., two had *Pseudomonas aeruginosa* and one had *Curvularia spp*. The last patient had no confirmed microbiologic diagnosis.

The researchers noted a number of conclusions from the study. The most frequent clinical risk factor for keratitis was contact lens wear, which was present 79% of the time. The average area of epithelial defect prior to the first RB-PDAT treatment was 32mm2±27mm^2, and the average stromal depth hyper-reflectivity measured with anterior segment OCT was 269µm±75µm. Patients who underwent RB-PDAT were considered successful cases if they were able to avoid therapeutic keratoplasty. This was achieved in 72% of cases, with an average time to clinical resolution of 46.9±26.4 days.

Naranjo A, Arboleda A, Martinez J, et al. Rose bengal photodynamic antimicrobial therapy (RB-PDAT) for patients with progressive infectious keratitis: a pilot clinical study. Am J Ophthalmol. September 5, 2019. [Epub ahead of print].

Nepafenac Improves Dilation in Diabetics

Researchers recently found that topical nepafenac has an additive effect on pupil dilation, which can be particularly useful when evaluating diabetes patients before cataract surgery. Neuropathic complications of diabetes can impair a patient's pupillary response, giving clinicians a smaller pupil diameter to work with during dilation. The limited view can hinder the clinician's ability to effectively assess ocular health. A surgeon adding nepafenac, commonly used to control pain and inflammation, to their presurgical regimen could help achieve sufficient pupil dilation for appropriate management and uncomplicated surgery.

This prospective comparative study evaluated 43 diabetes patients and 39 controls. The team took baseline pupil diameter measurements at baseline and at one hour after instilling cyclopentolate 1.0% in both eyes and nepafenac 0.1% to the eye receiving surgery.

The investigators found that baseline pupil diameters of both eyes were similar in both groups. They note that the change in pupil size from baseline to mydriasis was statistically significantly greater in the study eyes (2.69±0.53) than in the fellow eyes (2.54±0.61) in the diabetes group but that there was no statistically significant difference in the control group (2.94±0.63 vs. 2.86±0.58). When the groups were compared, the diameter changes were similar in the study eyes of both groups, while the changes in the fellow eyes were lower in the diabetes group, indicating the enhancing effect of nepafenac on pupil dilation in diabetic patients.

Based on their findings, the researchers recommend routine use of topical nepafenac 0.1% for pupil dilation before an exam and presurgery to achieve better results.

Kiziltoprak H, Koc M, Yetkin E, et al. Additive effect of topical nepafenac on mydriasis in patients with diabetes mellitus. Eye Contact Lens. September 9, 2019. [Epub ahead of print].

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Investigators on the Fence About SLT

Selective laser trabeculoplasty (SLT) is already a first-line glaucoma therapy in Europe, yet it hasn't reach the same status in the United States. Still, many practitioners are beginning to recommend the procedure sooner, and the American Academy of Ophthalmology's Preferred Practice Patterns suggest clinicians consider SLT as a first-line therapy in select patients, such as those who are noncompliant or struggling with the cost or side effects of medication.[1]

Now, new research further muddies the waters, suggesting SLT does not provide any improvement in patients' glaucoma-specific quality of life (QoL) compared with topical mediation.[2]

An international team of researchers looked at 167 treatment-naïve, mild-to-moderate primary open-angle or exfoliation glaucoma patients who were randomized to either SLT or topical medication as a first-line therapy. They used the glaucoma outcomes assessment tool (GOAT) to evaluate the patients' glaucoma-specific QoL at 12 and 24 months. They also documented IOP reduction and ocular surface parameters.

They found both treatment approaches improved GOAT measures, although the SLT group recorded a significant improvement in "social well-being" compared with the medication group at the two-year mark. Medication seemed to better control IOP long-term, given successful pressure reduction was 18.6% higher in the medication group compared with the SLT group. However, that success comes with a trade-off, as more patients in the medication group presented with conjunctival hyperemia and eyelid erythema compared with the laser group.

"Overall, we did not find evidence that SLT was superior to medication in improving glaucoma-specific QoL," the researchers concluded.

For some practitioners, the study raises more questions than it answers, according to Nate Lighthizer, OD, an associate professor at the Oklahoma College of Optometry. The pre-treatment IOP and overall success rates are just one piece of the puzzle.

"SLT is better for quality of life or 'social well-being,' which is expected. It also has fewer side effects, which is also expected, but didn't lower IOP by 25% as much as medications did," he explains. "So while it did not show that SLT is superior to medications, I don't know that the study points towards one treatment being superior to the other. Each treatment has its advantages."

The key is knowing how to select the right candidates for laser treatment and then educating them appropriately.

1. Prum BE, Rosenberg LF, Gedde SJ, et al. Primary open-angle glaucoma preferred practice pattern guidelines. Ophthalmology. 2016;123(1):41-111.
2. Ang GS, Fenwick EK, Constantinou M, et al. Selective laser trabeculoplasty versus topical medication as initial glaucoma treatment: the glaucoma initial treatment study randomised clinical trial. Br J Ophthalmol. September 5, 2019. [Epub ahead of print].

All Screens, No Play Affects Teen Myopes

It seems intuitive that increased screen time—especially for young patients—can increase myopia development. After all, it increases their near-work and blue light exposure and, often, decreases sunlight exposure and physical activity. Now, thanks to a Danish study, optometrists have even more research to share with patients.

The Copenhagen Child Cohort 2000 Eye Study focused on gathering information about the ubiquitous screen devices. Their specific findings are in regards to myopia prevalence in 16- and 17-year-old children and its relation to both physical activity and screen time.

Data from 1,443 questionnaires shows that low physical activity and high screen time contribute significantly and can roughly double the risk of developing or worsening myopia. The researchers qualified less than three hours per week of physical activity as "low physical activity" and more than six hours a day as "high" screen time. Myopia was defined as a non-cycloplegic subjective spherical equivalent refraction ≤–0.50D.

"Our results support physical activity being a protective factor and near work a risk factor for myopia in adolescents," the researchers concluded.

Hansen M, Laigaard P, Olsen E, et al. Low physical activity and higher use of screen devices are associated with myopia at the age of 16-17 years in the CCC2000 Eye Study. Acta Ophthalmol. September 9, 2019. [Epub ahead of print].

A Billion Reasons to Improve Eye Care

New WHO report calls for immediate action against vast challenges in ocular health.

A third of the world's population suffers from some form of vision impairment, and at least one billion people do so without current or prior medical attention that could have made a difference in outcomes and lives, according to the World Health Organization (WHO).

The organization recently issued its first *World Report on Vision*, which gathers global information about the burden of eye conditions and vision impairment. Aging populations, changing lifestyles and limited access to eye care are the main drivers of the rising numbers.

To better serve their citizens, low- and middle-income countries in particular need investments in medical facilities and caregiver workforce totaling at least $14.3 billion. High-income countries like the United States also face structural problems that allow eye care delivery to lag behind need, particularly in rural and minority communities.

The WHO is hoping the report will give the organization and other stakeholders a platform from which to lobby for greater commitments to eye care worldwide.

Impairment by the Numbers

Globally, at least 2.2 billion people have vision impairment, the report states. Of the one billion that could have been prevented or has yet to be addressed, uncorrected presbyopia tops the list, affecting 826 million people. Next highest is unaddressed refractive error (123.7 million), followed by cataract (65.2 million), glaucoma (6.9 million), corneal opacities (4.2 million), diabetic retinopathy (three million) and trachoma (two million).

That one billion is likely an underestimation, the WHO report notes, as potentially preventable cases of age-related macular degeneration are unknown and data on childhood visual impairment is lacking.

Regarding the combined 11.9 million people with vision impairment or blindness due to glaucoma, diabetic retinopathy and trachoma that could have been prevented, the report estimates the costs of preventive measures would have been $5.8 billion. "This represents a significant opportunity missed in preventing the substantial personal and societal burden associated with vision impairment and blindness," according to the report.

Addressing the Challenges

The *World Report on Vision* sets out concrete proposals to address challenges in eye care. Its key proposal is to "make integrated people-centered eye care, embedded in health systems and based on strong primary health care, the care model of choice and scale it up widely."

"People who need eye care must be able to receive high-quality interventions without suffering financial hardship," the report states. "Including eye care in national health plans and essential packages of care is an important part of every country's journey towards universal health coverage."

Policymakers should also direct attention to elevating the role of optometry, according to the report, which notes that "acceptance of optometry as a profession remains an issue in many countries and is an important advocacy issue going forward." The report concludes that "in some countries, productivity may be diminished because a section of the health workforce, such as optometrists, are not accredited to carry out eye care services independently."

The WHO admits that major challenges lie ahead, particularly the fact that global eye care needs will rise sharply due to changing demographics and lifestyles—the prevalence of any near vision impairment is highest in regions with longer life expectancies, and environmental factors such as decreased time spent outdoors and increased near-work activities are largely driving projected increases in myopia. The WHO estimates that, by 2040, there will be a 50% increase in the number of people worldwide requiring access to routine (i.e. yearly or biennially, depending on setting) retinal examinations for diabetic retinopathy.

Given the stark estimates and the growing global need, the WHO says it is committed "to working with countries to improve the delivery of eye care, in particular through primary health care; to improving health information systems for eye care; and to strengthening the eye care workforce."

To Learn More

The World Health Organization's *World Report on Vision* is available at www.who.int/publications-detail/world-report-on-vision.

Those attending the American Academy of Optometry meeting in Orlando can attend the Plenary Session on Wednesday, October 23, to hear more on the report's findings. ■

The World Health Organization. World report on vision. www.who.int/publications-detail/world-report-on-vision. October 8, 2019.

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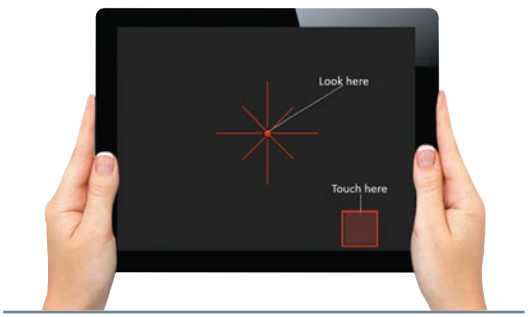

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Contents

Review of Optometry October 15, 2019

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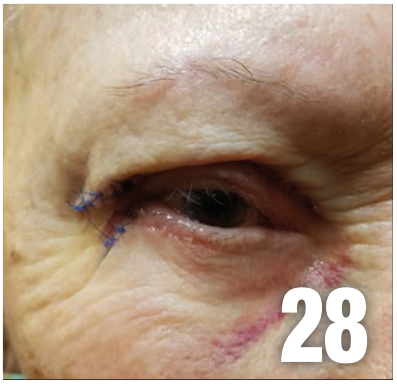
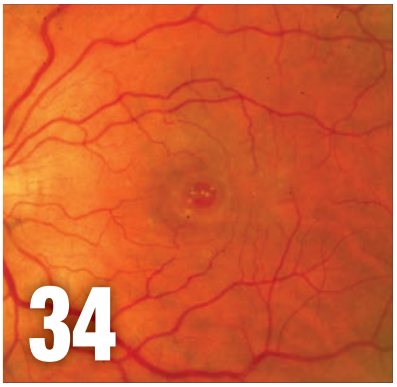
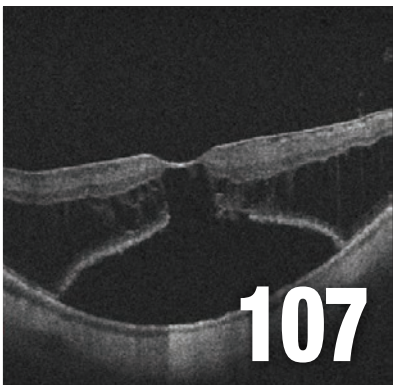
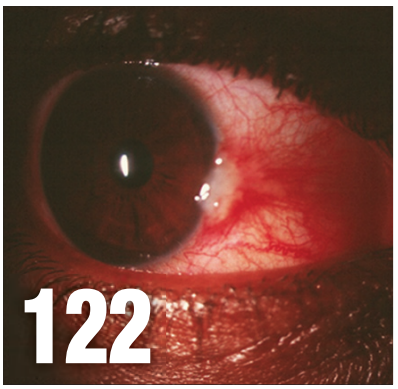
Departments

Review of Optometry October 15, 2019









REVIEW OF OPTOMETRY

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References:

1. Xiidra [Prescribing Information]. Lexington, MA: Shire US. **2.** TFOS DEWS II Research Subcommittee. Report of the Research Subcommittee of the Tear Film & Ocular Surface Society Dry Eye WorkShop II (2017). *Ocul Surf.* 2017;15(3):269-649. **3.** FDA approves new medication for dry eye disease. FDA News Release. July 2016. http://www.fda.gov/newsevents/newsroom/pressannouncements/ucm510720.htm. Accessed July 12, 2016. **4.** Food and Drug Administration. Electronic Orange Book. http://www.fda.gov/downloads/Drugs/DevelopmentApprovalProcess/UCM071436.pdf. Accessed June 26, 2018.

Indication

Xiidra® (lifitegrast ophthalmic solution) 5% is indicated for the treatment of signs and symptoms of dry eye disease (DED).

Important Safety Information

Xiidra is contraindicated in patients with known hypersensitivity to lifitegrast or to any of the other ingredients.

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To avoid the potential for eye injury or contamination of the solution, patients should not touch the tip of the single-use container to their eye or to any surface.

Contact lenses should be removed prior to the administration of Xiidra and may be reinserted 15 minutes following administration.

Safety and efficacy in pediatric patients below the age of 17 years have not been established.

For additional safety information, see accompanying Brief Summary of Safety Information on the adjacent page and Full Prescribing Information on Xiidra-ECP.com.

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Instill one drop of Xiidra twice daily (approximately 12 hours apart) into each eye using a single-use container. Discard the single-use container immediately after using in each eye. Contact lenses should be removed prior to the administration of Xiidra and may be reinserted 15 minutes following administration.

CONTRAINDICATIONS
Xiidra is contraindicated in patients with known hypersensitivity to lifitegrast or to any of the other ingredients in the formulation.

ADVERSE REACTIONS
Clinical Trials Experience
Because clinical studies are conducted under widely varying conditions, adverse reaction rates observed in clinical studies 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 five clinical studies of dry eye disease conducted with lifitegrast ophthalmic solution, 1401 patients received at least 1 dose of lifitegrast (1287 of which received lifitegrast 5%). The majority of patients (84%) had ≤3 months of treatment exposure. 170 patients were exposed to lifitegrast for approximately 12 months. The majority of the treated patients were female (77%). The most common adverse reactions reported in 5-25 % of patients were instillation site irritation, dysgeusia and reduced visual acuity. Other adverse reactions reported in 1% to 5% of the patients were blurred vision, conjunctival hyperemia, eye irritation, headache, increased lacrimation, eye discharge, eye discomfort, eye pruritus and sinusitis.

Postmarketing Experience
The following adverse reactions have been identified during postapproval use of Xiidra. Because these reactions are reported voluntarily from a population of uncertain size, it is not always possible to reliably estimate their frequency or establish a causal relationship to drug exposure.

Rare cases of hypersensitivity, including anaphylactic reaction, bronchospasm, respiratory distress, pharyngeal edema, swollen tongue, and urticaria have been reported. Eye swelling and rash have been reported.

USE IN SPECIFIC POPULATIONS
Pregnancy
There are no available data on Xiidra use in pregnant women to inform any drug associated risks. Intravenous (IV) administration of lifitegrast to pregnant rats, from pre-mating through gestation day 17, did not produce teratogenicity at clinically relevant systemic exposures. Intravenous administration of lifitegrast to pregnant rabbits during organogenesis produced an increased incidence of omphalocele at the lowest dose tested, 3 mg/kg/day (400-fold the human plasma exposure at the recommended human ophthalmic dose [RHOD], based on the area under the curve [AUC] level). Since human systemic exposure to lifitegrast following ocular administration of Xiidra at the RHOD is low, the applicability of animal findings to the risk of Xiidra use in humans during pregnancy is unclear.

Animal Data
Lifitegrast administered daily by intravenous (IV) injection to rats, from pre-mating through gestation day 17, caused an increase in mean preimplantation loss and an increased incidence of several minor skeletal anomalies at 30 mg /kg / day, representing 5,400-fold the human plasma exposure at the RHOD of Xiidra, based on AUC. No teratogenicity was observed in the rat at 10 mg /kg /day (460-fold the human plasma exposure at the RHOD, based on AUC). In the rabbit, an increased incidence of omphalocele was observed at the lowest dose tested, 3 mg /kg /day (400-fold the human plasma exposure at the RHOD, based on AUC), when administered by IV injection daily from gestation days 7 through 19. A fetal No Observed Adverse Effect Level (NOAEL) was not identified in the rabbit.

Lactation
There are no data on the presence of lifitegrast in human milk, the effects on the breastfed infant, or the effects on milk production. However, systemic exposure to lifitegrast from ocular administration is low. The developmental and health benefits of breastfeeding should be considered, along with the mother's clinical need for Xiidra and any potential adverse effects on the breastfed child from Xiidra.

Pediatric Use
Safety and efficacy in pediatric patients below the age of 17 years have not been established.

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

NONCLINICAL TOXICOLOGY
Carcinogenesis, Mutagenesis, Impairment of Fertility
Carcinogenesis: Animal studies have not been conducted to determine the carcinogenic potential of lifitegrast.
Mutagenesis: Lifitegrast was not mutagenic in the *in vitro* Ames assay. Lifitegrast was not clastogenic in the *in vivo* mouse micronucleus assay. In an *in vitro* chromosomal aberration assay using mammalian cells (Chinese hamster ovary cells), lifitegrast was positive at the highest concentration tested, without metabolic activation.
Impairment of fertility: Lifitegrast administered at intravenous (IV) doses of up to 30 mg/kg/day (5400-fold the human plasma exposure at the recommended human ophthalmic dose (RHOD) of lifitegrast ophthalmic solution, 5%) had no effect on fertility and reproductive performance in male and female treated rats.

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REVIEW OF OPTOMETRY

PRINTED IN USA

Outlook

By Jack Persico, Editor-in-Chief

Bad Optics

The easiest way to help almost a billion people is simply to give them a pair of glasses. But that's only the start.

It's commonplace to be jaded about the epidemiology figures for eye diseases. We routinely see huge numbers tossed about—e.g., two million Americans have AMD, three million have glaucoma—and gloss over the human impact of those words. It brings to mind a quote often attributed to Joseph Stalin: "One death is a tragedy. One million deaths is a statistic."

But there are real people behind these numbers, and it's worth remembering that when we see the staggering figures in a new report from the World Health Organization on the pervasiveness of vision loss.

An array of numbers in the *World Report on Vision* tells the story of vision's inherent fragility, beginning with top-line estimates of people affected by eye conditions capable of leading to impairment but not yet inflicting any. Figures for myopia (2.6 billion sufferers) and presbyopia (1.8 billion) highlight the silver lining: the most pervasive eye problems are at least the easiest ones to rectify. More intractable, of course, are those conditions brought on by disease processes like AMD, which affects 196 million people globally, diabetic retinopathy (146 million) and glaucoma (76 million).

The WHO puts the tally for those experiencing some kind of vision loss at 2.2 billion people, and says one billion could have been helped if they had gotten better—or, heck, any!—eye care. "As usual, this burden is not borne equally," the report says. "It weighs more heavily on low- and middle-income countries, on older people, and on rural communities."

Drilling down into that one billion figure, we see the lion's share is comprised of uncorrected presbyopia, affecting 826 million people. Consider that for a minute. Something as simple as a pair of drugstore readers could make a profound difference for a group of people 2.5 times the size of the entire US population. Next highest is unaddressed refractive error (123.7 million), followed by cataract (65.2 million), glaucoma (6.9 million), corneal opacities (4.2 million), diabetic retinopathy (three million) and trachoma (two million).

So, using the WHO's numbers, at least 950 million people just need glasses, by far the easiest thing in eye care to provide. It makes me think of the (frustrating, to us in optometry) use of the word *optics* in political parlance to mean *bad PR*. Clearly, the optics of it all doesn't look good. Add in the need for preventive care and medical intervention for active eye disease, and the global burden seems nearly insurmountable.

One WHO recommendation involves giving optometry a boost. The profession sorely needs it. The scope, and very definition, of optometry varies wildly among countries. In many, it's much more akin to opticianry. "In some countries," the report states, "productivity may be diminished because a section of the health workforce, such as optometrists, are not accredited to carry out eye care services independently."

The WHO report offers a sobering look at the work to be done. Building up optometrists globally—as both vision *and* eye health pros—could make a profound difference. ■

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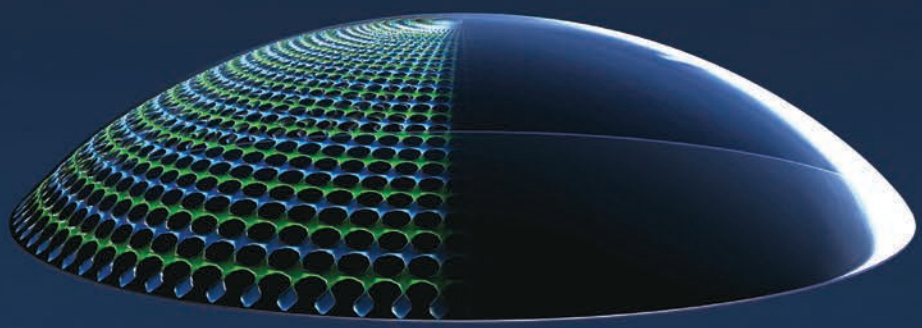

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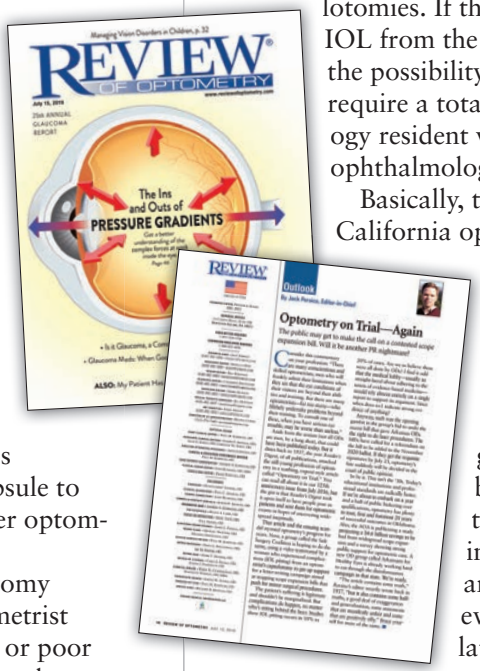
Letters to the Editor

The July 2019 Outlook column, "Optometry on Trial—Again," discussed the public relations case being made against optometric scope of practice expansion in Arkansas. Earlier this year, the state's ODs won legislative approval to perform minor laser surgical procedures and were quickly met with a concerted effort by the medical lobby to discredit optometry. One tactic used by the group is a video testimonial from a patient who suffered vision impairment from IOL pitting resulting from an optometrist's capsulotomy.



Don't Get Burned by Lasers

As an OD with considerable experience comanaging cataract surgery patients, I would like to offer my thoughts on what is a manufactured case against one optometrist concerning one patient that was then used to castigate the entire profession:

(1) Cheaper IOLs without laser ridges might require IOL pitting to get the capsule to open regardless of the operator (whether optometrist or ophthalmologist).

(2) IOL pitting on one YAG capsulotomy patient by an ophthalmologist or optometrist does not indicate either poor technique or poor equipment. Pitting IOLs on *all* YAG capsulotomies obviously does represent a problem, which I have seen with certain ophthalmologists.

(3) Even when I have seen consistent, extensive pitting of the IOL from an ophthalmologist in my area, I haven't had patients complain about their vision. I do question the symptoms the patient reported as caused by a pitted IOL. Thus, I have not seen a cause and effect between IOL pitting and reduced vision in any patient that I can remember in 35 years of post-op cataract surgery patient care.

(4) Though I have not done even one YAG capsulotomy, I have extensive experience with patients who have had the procedure. Equipment company technicians used to train ophthalmologists on how to perform the procedure, but I believe most YAG laser instruments have so many automatic features that this is no longer necessary. Initially, the lasers had problems with alignment and the instruments were hard to use correctly, but that was 30 years ago.

(5) In California, a lobbyist for organized ophthalmology testified falsely that he was in his ophthalmology residency program being trained how to use the YAG capsulotomy laser when he inadvertently pressed a wrong button, causing an IOL to dislodge into the back of the eye. Only after emergency surgery with a gifted university chorioretinal specialist was the eye saved.

IOLs don't fall back into the eye with YAG capsulotomies. If the patient had an iris-fixated IOL from the late 1970s, there would be the possibility of this, but this would also require a totally incompetent ophthalmology resident with a totally incompetent ophthalmology university professor.

Basically, this statement killed an old California optometry bill that included the ability to do YAG capsulotomies. The thought of IOLs 'falling back into eyes' and multiple patients going blind was too much for the legislative committee. So great was the lie by the lobbyist that the California optometrists who were testifying did not know what to say and did not know if this had ever happened. The bill was later substantially amended to exclude YAG capsulotomy privileges for California optometrists.

(6) Why would optometrists want to do YAG capsulotomies in the first place? If the result is anything but stellar, the patient could then return to the original ophthalmologist and give the MD ammunition. The ophthalmologist could blame the optometrist for almost anything, including floaters (which are common anyway), halos if the capsulotomy is not big enough and, of course, IOL pitting, which doesn't bother the patient but can be photographed and shown to stoke fears and resentment.

(7) The issue of the cost of using a laser must also be considered. Can an optometrist afford to do this procedure? Probably not. Refractive lasers might be something for optometry to consider; keeping that out of our hands is what this is all about. Sometimes there is no reality to the politics of medical procedures and patient care, and monetary factors influence what we do and why.

—Don Stover, OD, Porterville, CA

It's no longer just an exam.

The new exam room experience.

The exam room is an extremely important part of your optometric/ophthalmology practice. As a doctor, you spend the majority of your day interacting with patients, staff and equipment in this confined space. It is an intimate setting where you have the opportunity to diagnose a patient's wellness and change their lives with the ability to help them see better. As an eye care provider you are the most significant figure in your practice, you are your own brand. The way you operate and present your knowledge, while improving the lives of your patients, is reflected in the space you occupy.

This space is more than just an exam lane, it is also an opportunity for you to capture greater revenue and simplify the process of selling eyewear, lenses and elective procedures. Having the right equipment and tools in an organized space is a reflection of the attention to detail in the clinical care you give your patients. As you spend time bouncing from one room to another, patients are isolated and have time to scan their surroundings. They will be quick to take notice of old worn out chairs, clutter on your desk and the cleanliness of your space. Many patients may judge your office by how it looks, including how messy or organized it is.

Take the opportunity to self-evaluate your space from the patients' point of view:

- **When was the last time you sat in your examination chair?**
- **Is it still comfortable, clean and fully functioning?**
- **Is the room inviting?**
- **Does it have an accent wall to add a bit of style?**
- **Are there tripping hazards or uncon-cealed computer cords and other wires?**
- **How easy is it to dim lights or turn them on and off?**
- **Do you have to stop what you are doing to go across the room?**
- **Finally, how easy is it to grab what you need such as tissues, hand sanitizer, drops, trial lenses and other supplies?**

Have a staff member or partner doctor conduct an exam on you and see how efficient, or inefficient, your exam process is. Study how often you have to turn your back on your patient, while taking notes or typing on the computer.

Exam environment by ORVOS Exams, a division of the Eye Designs Group.

The design of an exam lane has changed. Computer vision testing systems, retina imaging, chairs and stand engineering has advanced, along with exam lane furniture. A simple angle in the desk can help achieve the necessary eye contact with your patients. Using advanced lighting technology like remote control switches will promote more efficient workflow with minimal interruptions. Removing clutter from your work surface, having the supplies you need at eye level and within arm's reach alleviates the daily search for script pads, drops and tools. Mounting your computer screen to an articulating arm above your desk will allow you to easily share information with your patients by conveniently being able to share your monitor.

Finally, don't forget your stool. You need to ensure a perfect fit. No matter if you're seeing one patient or an entire day of them, your exam room stool is a vital part of your daily routine. By making sure your stool addresses your particular needs, you will feel less fatigue, along with being more productive and comfortable throughout the day. Many companies offer a series of ergonomically designed and engineered stools and chairs that promote proper posture, allow proper adjustment and offer stability. Stools are an investment in your health, so choose wisely.

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Beating 20/Blurry

Acuity is just the tip of the visual health iceberg. Don't forget to test other aspects of a patient's visual perception. **By Paul M. Karpecki, OD, Chief Clinical Editor**

We all have patients who measure 20/20 but still have vision complaints. These situations can be frustrating, because the patient is usually right—something else is hindering their vision. These newer diagnostic tools can help you think beyond visual acuity to diagnose any number of conditions.

Black on White

Rarely do you see such extreme contrast as black letters on a white background in the real world, and yet that is what we use to test subtle vision changes. Our trusty Snellen chart, introduced back in 1862, still works, even though we know it may not be a truly sensitive test of vision.

But we now have access to better testing to augment our visual acuity testing. Contrast sensitivity testing (M&S Technology) can identify subtle vision changes due to everything from cataracts to retinal disease. Research shows contrast color testing (Konan Medical) is far superior to Ishihira plates, which can miss more than 50% of protanopes.[1] Point-spread-function (PSF) images with the VMax PSF phoropter (VMax Vision) helped a second-year optometry student provide more accurate refractions compared with three faculty members (with more than 50 years of refracting experience between them) using a standard manual phoropter.[2]

Lens Dysfunction

For many, the crystalline lens becomes dysfunctional long before insurance will cover a cataract procedure. Some may even argue that presbyopia is the beginning of notable lens dysfunction. As the lens continues to grow, it starts with presbyopic issues and then begins to scatter light. Thus, devices such as the HD analyzer (Visiometrics) can be valuable in detecting pathology based on light scatter from the lens or tear film.

Other helpful technologies include those with Scheimpflug imaging capabilities. Clinicians can capture Scheimpflug images with the camera perpendicular to the slit beam, imaging about 25,000 data points to create a 3-D model of the anterior segment. The Pentacam (Oculus), Galilei (Ziemer) and now the VX130 (Visionix) all include Scheimpflug imaging, which can reveal pathology ranging from subtle keratoconus to early refractive lens changes, even in patients seeing 20/20 vision.

Ocular Issues Front to Back

Although dry eye disease (DED) typically manifests with fluctuating vision, many of these patients measure 20/20 and complain of blurry vision. Diagnostic tools such as the Keratograph 5M (Oculus), LipiView (Johnson & Johnson Vision) and osmolarity testing (TearLab) can help reveal an underlying DED etiology.

In retinal disease, patients with early epiretinal membranes and mild diabetic macular edema may still test 20/20. Here, OCT imaging is key. Most early macular degeneration remains undiagnosed, even though patients with early signs will have night vision issues while still measuring 20/20 on high contrast testing. More sensitive tests such as dark adaptometry (AdaptDx, Maculogix) can uncover the diagnosis in about six minutes.

When things just don't add up, consider neurological diseases affecting the eye and use pupil testing, color vision and visual field testing. While the swinging flashlight test can be difficult and doesn't identify a subtle RAPD without filters, a new technology, Eyekinetix (Konan Medical), provides an RAPD assessment in less than 40 seconds and can identify subtle asymmetry indicating neurological disease or neuropathies such as glaucoma.

The 20/blurry patient is both a challenge and an opportunity. Most of these patients have seen multiple doctors and are still searching for answers. Our ability to listen carefully, measure more effectively and use advanced diagnostics and clinical acumen can provide some much-needed insight into these cases.

This is what separates us from remote refractions; we complete an entire ocular health assessment, benefiting both the patient and our practice. It's well worth the effort. ■

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

1. Birch J. Efficiency of the Ishihara test for identifying red-green colour deficiency. Ophthalmic Physiol Opt. 1997;17(5):403-8.
2. Newman C, Lievens C, Kabat A, Weber J. Repeatability of the Vmax voice activ subjective refractor (VASR) and traditional refractive methods in a healthy population. Poster presented at 2018 ARVO Annual Meeting, April 29, 2018, Honolulu, Hawaii.

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Should I Stay or Should I Go?

Sometimes it's worth hauling your phoropter over to a new building, if only for a little exercise. **By Montgomery Vickers, OD**

It's harder to hit a moving target. This is a lesson all optometrists—and Navy SEALs—know well. Becoming stagnant only leads to more mosquitos, at least here in Texas. Therefore, move or die, right?

Of course, this could mean all sorts of things in practice. In some offices, it means get off your rear and take a walk—the literal translation of the "die" part, I guess. Getting out for a daily stroll can make you healthier and, of course, give you a chance to vape in peace. (Disclaimer: unlike the typical serious advice in this column, I do not advise vaping. I would never advise unhealthy activities… as I type this column while driving to work.)

Moving can also mean to getting off your rear when it comes to new tech. I know it's hard. I'm 66, and I still remember Prentice's Rule. I never actually *used* Prentice's Rule, but since I missed that question on the Boards in 1979, I'll never forget.

Actually, new technology can keep you interested in your career. Not because it intrigues you, teaches you and allows you to better care for patients. It does that, but you also have to stay in the game just to pay for the darn stuff. It's expensive.

Movin' on Up

What about moving your office? Is that a good idea? And, also, have you lost your mind?

It's hard to move from your current location. For one thing, even if you send a driver to every patient's home to drive them to their appointment—after a stop at Starbucks for a pumpkin spice latte—some patients will still insist the new office is not convenient. Also, a certain percentage of patients will automatically assume that you are raising your fees because of the upgrade. That's assuming you are moving up, not down, in relocating.

Some patients will be unhappy because "it's just not the same." That's true, the toilet actually works at the new office.

So, why move at all? There are some good reasons. You already know the old office's toilet doesn't work. Plumbers are expensive; save that $200 fee by spending a million dollars to move.

But sometimes the old office just peters out. The roof is falling in, and the area has deteriorated into a place where kids hang out and do dangerous and foolish things like text one another. Time to go.

Maybe you need more space. Growth in your practice is a great problem indeed and, here in Texas, many areas are just exploding with increased population. That's why we (and by "we" I mean my brilliant young colleagues… I am just the junior associate) have just increased our office footprint by 6,000 square feet.

Sadly, moving doesn't solve all the world's problems. Mo' footage, mo' problems. Mo' staff, mo' problems. Mo' technology, mo' problems. Three bathrooms have solved one problem, at least.

You have to know yourself and your area before any move. When I moved my office in dear ol' St. Albans, WV, I went from four exam rooms and eight employees to two exam rooms and two and a half employees. I downsized at just the right time to get lean and mean, increasing efficiency and the bottom line in a small but lovely little space filled with all the technology I could afford. My patients and I both benefitted, and that's always the goal with any move.

Move to do better and be better. That's when it works. ■

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Eyes Wide Open

Eyelid surgery requires a conservative approach to avoid exposure keratitis.

Edited by Paul C. Ajamian, OD

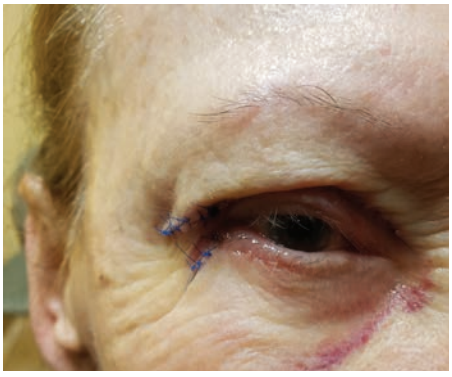
Q A patient came in as an emergency due to severe pain in both eyes after a recent blepharoplasty at a plastic surgery center. I asked her to close her eyes, and she couldn't. Her corneas revealed exposure keratitis as expected. What is the predicted course of her post-op recovery?

A "Manage and optimize risk factors such as dry eye and ocular surface disease pre-op," James P. Milite, MD, an oculoplastic surgeon of Omni Eye Services in Iselin, NJ, says. "A conservative approach is mandated intraoperatively, with careful skin resection and attention to eyelid closure."

When a patient asks you about lid surgery, take into consideration complaints of visual obstruction and brow fatigue. Cosmetic concerns, such as hooding or dermatochalasis, might be accentuated by physical discomfort that abrasion of the upper lid fold creates as it overrides the cilia. "Brow ptosis can cause a false appearance of upper eyelid hooding, leading to possible unexpected skin over-resection," says Dr. Milite.

Surgical Considerations

Margin reflex distance distinguishes true ptosis with low position of the upper eyelid margin from hooding. Have the patient focus on a focal light source with both eyes and measure the distance from the pupillary light reflex to the upper and lower eyelid margins. Cases with good levator function (greater than 10mm) can employ levator advancement. Poor levator function—more commonly seen in ptosis of congenital, muscular dystrophic or neurologic etiology—requires frontalis suspension.



Post-op overcorrection of ptosis could cause upper eyelid retraction and exposure keratitis.

To prevent post-op upper eyelid retraction and exposure keratitis, Dr. Milite emphasizes that careful preoperative incision marking can help avoid excessive skin excision. He believes a conservative approach is always appropriate. "Residual skin excess can always be secondarily resected, but excess skin removal cannot be easily corrected," Dr. Milite says.

In cases where true ptosis exists and the levator position has advanced onto the tarsus, the surgeon will check eyelid position by asking the patient to open and close their eyes intraoperatively. They will assess symmetry as well as check for over- or under-correction. If the main source of visual obstruction is excessive skin fold overhang, patients should still open and close their eyes intraoperatively. An intraoperative levator dehiscence induced by resecting skin could be missed and lead to an under-correction.

Post-op Checks

Dr. Milite advises to check for eyelid symmetry and ensure that eyelid closure is complete. "Symmetry is usually not exact at initial follow-up due to edema, so schedule a three- or four-week post-op visit when the edema has resolved," he says. The patient should expect two to three weeks of post-op swelling and bruising.

If exposure keratitis persists, there are many possible causes. Primarily, there might be over-advancement of the levator muscle or there is a skin shortage from excessive skin resection or both. If it's the levator muscle, a re-operation and levator position adjustment usually corrects it.

If the skin has been excessively removed, Dr. Milite says that is much more difficult to manage. He recommends maximizing topical lubrication, nocturnally applying ointment, placing punctual plugs, using moisture chamber goggles and massaging the upper eyelid skin downward once incisions have healed. If the patient's problems persist, refer them back to the surgeon.

Maintaining ocular health takes priority over patient expectations of cosmetic and visual outcomes overall, so temper their expectations when counseling them before surgery, Dr. Milite adds. In the end, adequate eyelid closure and maintenance of a healthy ocular surface always trumps cosmesis. ■

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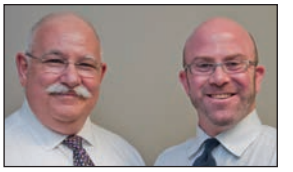


Putting Add Power to the Test

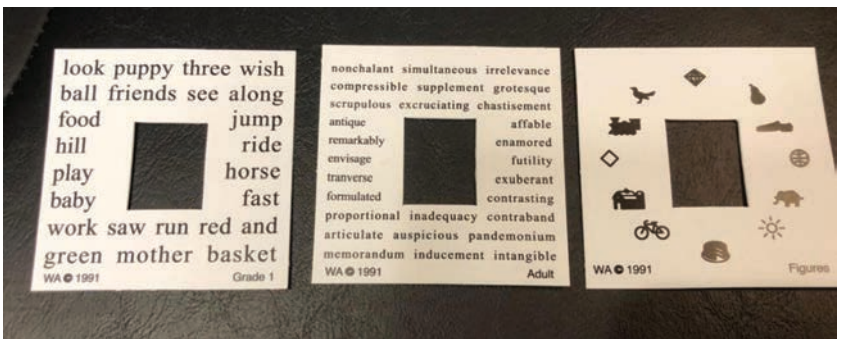
Learn more about the monocular estimation method and the fused cross-cylinder test to help manage your binocular vision patients. **By Marc B. Taub, OD, MS, and Paul Harris, OD**

One of the greatest challenges of treating younger patients with binocular vision and accommodative issues is finding an appropriate add power. One size does not fit all. Let's dig a little deeper into two ways we can do this: the monocular estimation method (MEM) and the fused cross-cylinder (FCC) test. Some practitioners have been taught that the two methods are interchangeable and you can do one or the other to measure the lag or lead of accommodation. In reality, the two could not be more different.

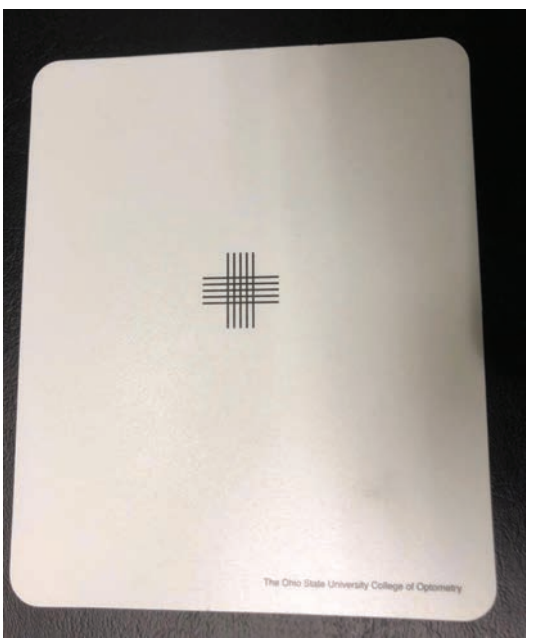
The FCC Test

We perform the FCC test in dim illumination. With the phoropter in place, set the cross-cylinder lenses in front of the patient. Have the patient close their eyes, place the horizontal/vertical grid at 40cm and add +0.50D of sphere. When the patient opens their eyes, ask them which lines appear darker and clearer. If they report that the lines are equal or horizontal, add plus lenses until the vertical lines are now the blackest or clearest. This is the desired endpoint. If the patient reports that the lines are vertical, on the other hand, add minus lenses until the lines are equal or horizontal. This is the desired endpoint in this situation.

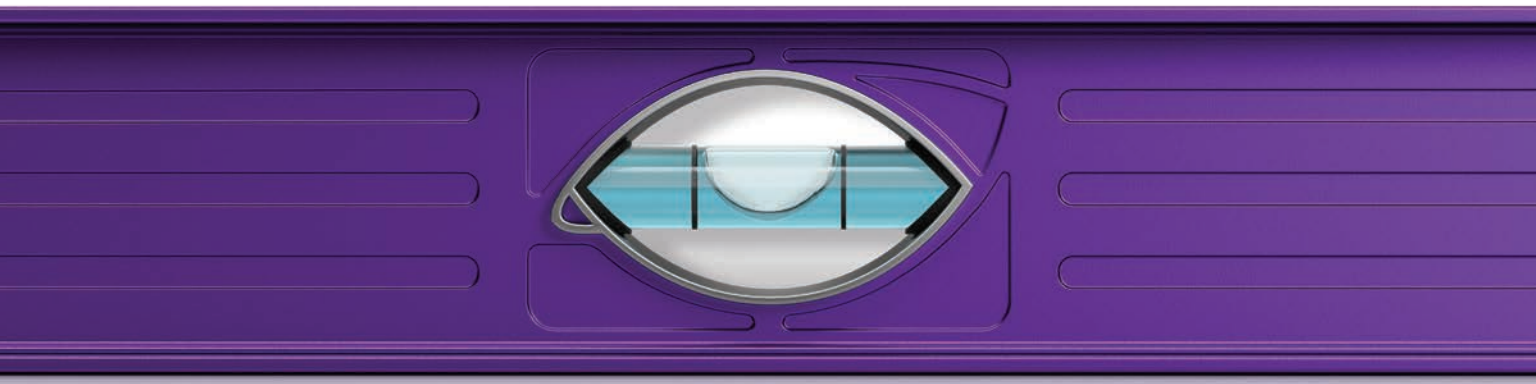
The expected values of the FCC test are +0.50D +/-0.50, which is why we start with +0.50D of sphere. One caveat to keep in mind is that if minus is found, it is best not to use that same amount of minus during the remainder of the near testing sequence. If a higher amount of plus is found, it is considered a lag of accommodation or under-accommodation. By contrast, if a lower amount of plus, or a minus endpoint, is found, it is considered a lead of accommodation or over-accommodation.



MEM cards (above) and FCC test cards (below) are both useful tools to help identify appropriate add powers in patients with binocular vision and accomodative issues.



Though we are trained to look for "the number," we should also be using the patient's responses to assess how they interpret their visual space in this situation. There are numerous variations to the procedure in regard to the starting lens. While I (Dr. Taub) use the previously discussed sequence, Dr. Harris starts with no added lenses, just the distance refraction, to determine if the patient sees what is expected and then adds plus that equates to the stress-point retinoscopy number he obtained in his chair tests. Others start with +1.50D and take it away in increments until the first vertical is reported.



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IMPORTANT SAFETY INFORMATION

CONTRAINDICATIONS
Contraindicated in acute superficial herpes simplex keratitis, vaccinia, varicella, and most other viral diseases of the cornea and conjunctiva; mycobacterial infection of the eye; fungal diseases; acute purulent untreated infections, which like other diseases caused by microorganisms, may be masked or enhanced by the presence of the steroid; and in those persons who have known hypersensitivity to any component of this preparation.

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

[a]**STUDY DESIGN:** The efficacy and safety of FLAREX (n=41) vs FML* (n=37) were evaluated in a randomized, double-blind clinical trial in 78 patients with ocular surface inflammation (eg, conjunctivitis, episcleritis, scleritis) in one or both eyes. In a separate randomized, double-blind clinical trial in 82 patients with ocular surface inflammation in one or both eyes, the efficacy and safety of FLAREX (n=37) vs prednisolone acetate 1.0% (n=45) were evaluated. In these studies, patients administered either FLAREX or FML*/prednisolone acetate 1.0% every 2 hours for the first 2 days and then every 4 hours thereafter, with signs and symptoms of inflammation assessed at Days 1, 3, 8, and 13. At each visit, investigators determined if symptoms in the involved eye were resolved (cured), improved, unchanged, or worsened. If a patient was rated as cured before the end of the study, steroid drops were discontinued and the patient was considered to have completed the trial.[2]

[b]Cost information based on Wholesale Acquisition Cost (WAC), 2019 data.

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FLAREX (fluorometholone acetate ophthalmic suspension) is indicated for use in the treatment of steroid-responsive inflammatory conditions of the palpebral and bulbar conjunctiva, cornea, and anterior segment of the eye.

DOSAGE AND ADMINISTRATION
Shake Well Before Using. One to two drops instilled into the conjunctival sac(s) four times daily. During the initial 24 to 48 hours, the dosage may be safely increased to two drops every two hours. If no improvement after two weeks, consult physician. Care should be taken not to discontinue therapy prematurely.

CONTRAINDICATIONS
Contraindicated in acute superficial herpes simplex keratitis, vaccinia, varicella, and most other viral diseases of the cornea and conjunctiva; mycobacterial infection of the eye; fungal diseases; acute purulent untreated infections, which like other diseases caused by microorganisms, may be masked or enhanced by the presence of the steroid; and in those persons who have known hypersensitivity to any component of this preparation.

WARNINGS AND PRECAUTIONS
Topical Ophthalmic Use Only
For topical ophthalmic use only. Not for injection.

Intraocular Pressure Increase
Prolonged use may result in glaucoma, damage to the optic nerve, and defects in visual acuity and visual field. It is advisable that the intraocular pressure be checked frequently.

Cataracts
Use of corticosteroids may result in cataract formation.

Delayed Healing
Topical ophthalmic corticosteroids may slow corneal wound healing. In those diseases causing thinning of the cornea or sclera, perforation has been known to occur with chronic use of topical steroids.

Viral Infections
Use in the treatment of herpes simplex infection requires great caution.

Bacterial Infections
Use of corticosteroids may suppress the host response and thus aid in the establishment of secondary ocular infections. Acute purulent infections of the eye may be masked or exacerbated by the presence of steroid medication.

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.

Contamination
Do not touch dropper tip to any surface, as this may contaminate the suspension.

Contact Lens Wear
Contact lenses should be removed during instillation of FLAREX but may be reinserted after 15 minutes.

Temporarily Blurred Vision
Vision may be temporarily blurred following dosing with FLAREX. Care should be exercised in operating machinery or driving a motor vehicle.

ADVERSE REACTIONS
Clinical Trials Experience
Glaucoma with optic nerve damage, visual acuity and field defects, cataract formation, secondary ocular infection following suppression of host response, and perforation of the globe may occur.

Postmarketing Experience
The following reaction has been identified during postmarketing use of FLAREX in clinical practice. Because reactions are reported voluntarily from a population of unknown size, estimates of frequency cannot be made. The reaction, which has been chosen for inclusion due to either its seriousness, frequency of reporting, possible causal connection to FLAREX, or a combination of these factors, includes dysgeusia.

USE IN SPECIFIC POPULATIONS
Pregnancy
Fluorometholone has been shown to be embryocidal and teratogenic in rabbits when administered at low multiples of the human ocular dose. Fluorometholone was applied ocularly to rabbits daily on days 6-18 of gestation, and dose-related fetal loss and fetal abnormalities including cleft palate, deformed rib cage, anomalous limbs, and neural abnormalities, such as encephalocele, craniorachischisis, and spina bifida, were observed. There are no adequate and well-controlled studies of fluorometholone in pregnant women, and it is not known whether fluorometholone can cause fetal harm when administered to a pregnant woman. Fluorometholone should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Lactation
Systemically administered corticosteroids appear in human milk and could suppress growth, interfere with endogenous corticosteroid production, or cause other untoward effects. It is not known whether topical administration of corticosteroids could result in sufficient systemic absorption to produce detectable quantities in human milk. Because many drugs are excreted in human milk, caution should be exercised when FLAREX is administered to a nursing woman.

Pediatric Use
Safety and effectiveness in pediatric patients have not been established.

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

NONCLINICAL TOXICOLOGY
Carcinogenesis, Mutagenesis, Impairment of Fertility
No studies have been conducted in animals or in humans to evaluate the possibility of these effects with fluorometholone.

PATIENT COUNSELING INFORMATION
Risk of Contamination
Do not touch dropper tip to any surface, as this may contaminate the suspension.

Use with Contact Lenses
The preservative in FLAREX, benzalkonium chloride, may be absorbed by soft contact lenses. Contact lenses should be removed during instillation of FLAREX but may be reinserted 15 minutes after instillation.

Temporarily Blurred Vision
Patients should be advised that their vision may be temporarily blurred following dosing with FLAREX. Care should be exercised in operating machinery or driving a motor vehicle.

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References: 1. FLAREX [package insert]. Fort Worth, TX: Alcon Laboratories, Inc; 2017. **2.** Leibowitz HM, Hyndiuk RA, Lindsey C, et al. Fluorometholone acetate: clinical evaluation in the treatment of external ocular inflammation. *Ann Ophthalmol*. 1984;16(12):1110-1115. **3.** Data on file. Fort Worth, TX: Eyevance Pharmaceuticals LLC. **4.** US Department of Health and Human Services, Food and Drug Administration. *Approved drug products with therapeutic equivalence evaluations*. (Orange Book). 38th ed. Washington, DC: US Department of Health and Human Services, Food and Drug Administration; 2018.

FLA-09-19-AD-42

The MEM

This is one of many options for near-point retinoscopy. The purpose, like the FCC test, is to assess the accommodative response. You can complete the MEM at any point in the examination, either with the habitual glasses or the potential new prescription. Attach an MEM card of the appropriate cognitive level (pictures and basic or complex words) to the retinoscope and hold it at the patient's Harmon Distance—the distance from the patient's elbow to their middle knuckle, or the closest distance at which the person should be performing activities. Any closer than this, regardless of the lenses prescribed, the amount of near visual stress increases significantly.

Use loose lenses or skiascopy bars to neutralize the patient's reflex while they name the pictures and read the words out loud. The trick is to insert the lens quickly and not leave it in place too long to avoid impacting the accommodative response. The lens and light from the retinoscope should not move simultaneously; the lens should be in place before the retinoscope begins sweeping.

The result is based on the assumption that the accommodative stimulus at distance is zero, so the MEM can also determine if the patient is over- or under-minused. The expected result is +0.25D to +0.50D +/-0.50. As with the FCC test, greater amounts of plus indicate a lag of accommodation, while plano to minus indicates a lead of accommodation.

Spot the Differences

In looking at these two tests, there are obvious differences. The FCC test is part of the near-point sequence. While it seems that every doctor has their own near-point sequence, I complete the FCC test after the binocular balance, which is then followed by the negative and positive relative accommodation tests. This provides the clinician with a potential near-point lens, through which we can conduct further binocular and accommodative testing. The MEM, on the other hand, is essentially a stand-alone test that is not part of the near-point sequence. Dr. Harris adds the base-out and base-in duction results in this sequence.

We perform FCC in the phoropter and the MEM in free space. Once you put a patient behind the phoropter, you can never be too sure where their attention is focused. The phoropter is a foreign object that is placed close to the face, and that in and of itself, in our experience, can cause near-point changes.

Target-wise, the FCC test uses vertical and horizontal lines, while the MEM uses pictures and words of varying sizes. The accommodative response is different based on the targets and cognitive components. It is one level of accommodation to identify a picture and read a word out loud and another to look at black lines and determine which are clearer or darker. Both require concentration, but the tasks are quite different.

The MEM is an objective test, while the FCC test is subjective. I prefer the MEM for this reason, as I have learned to trust what I see as the accommodative response as opposed to what the patient says they appreciate. The patient's interpretation of the black lines can sometimes be problematic, leading to results that do not make sense or seem antithetical to the rest of the examination data. This is one of the reasons some clinicians drop the FCC test from their examination sequence. Eliminating a test that prompts more questions than it answers is the most logical response in this case.

Even though the FCC test is subject to interpretation, its results should not be discounted. Think of the FCC test as a measure of how efficient and effective a patient is at using their visual system. If there isn't an obvious endpoint and the patient is wavering between several different but equal responses, maybe this is actually telling us how we should prescribe and treat moving forward.

Without a distinct endpoint, near-point lenses are less of a treatment choice. Vision therapy is a more appropriate option to provide the patient meaningful experiences that they can use to build their visual library. When using the results of either test to prescribe a near-point lens, you must leave the patient with their +0.50D buffer (the expected result of each test) in place. For example, if the patient presents with an FCC test or MEM result of +1.00D, you would not want to prescribe a +1.00D add. The most you should consider is +0.50D. Prescribing away the buffer can lead to further near-point issues and have an impact on the distance refraction.

The MEM and the FCC test cannot be used interchangeably. The presence of the phoropter, differing targets and the objective vs. subjective nature of the tests make them completely different. It is up to you which you prefer for your patients, but make sure to choose one. ■


Facedown Showdown

Everyone agrees timely macular hole intervention is crucial—but post-op positioning is up for debate. **By Diana Shechtman, OD, Jay M. Haynie, OD, and Brendan Girschek, MD**

Macular holes are a well-known problem for older patients. While their incidence is relatively low, affecting just 0.02% of patients aged 40 or older, full-thickness defects can lead to central vision loss if not treated properly.[1] Women are significantly more likely to present with a macular hole, with a 64% increased risk compared with men, and Asian-Americans have a whopping 177% increased risk compared with Caucasians.[1]

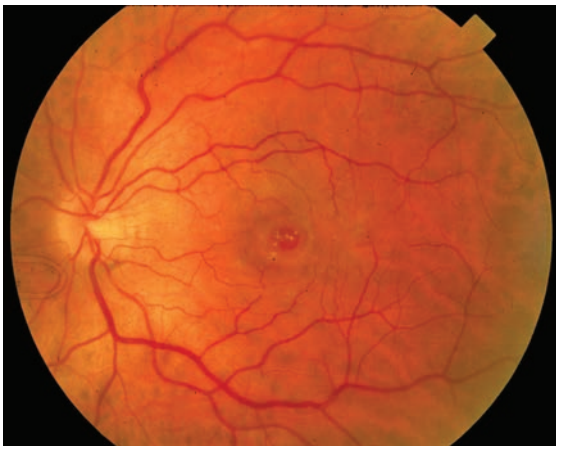

Fig. 1. This patient's three-month history of vision loss was due to a full-thickness macular hole.

Tractional forces—such as vitreomacular traction (VMT) and adhesion (VMA)—are contributing factors to the formation of an idiopathic macular hole. Although the typical presentation is unilateral, a patient with a macular hole has about a 10% chance of developing a macular hole in the contralateral eye in the presence of VMA/VMT.

As is often the case with non-urgent retinal problems, most optometrists will encounter such patients at some point in their careers and will have to make the call: monitor or refer?

Set the Stage

Careful evaluation with OCT imaging can help you identify at-risk patients and stage the macular hole when present.

A stage 0 to 1 macular hole is described as a partial posterior vitreous detachment (PVD) with insertion of the posterior hyaloid into the fovea and no disruption of outer retinal layers. Partial-thickness (or lamellar) macular hole formation is a lateral or tangential tractional disorder with intact outer retinal layers. Many of these subtle contour changes are asymptomatic but should be observable, and clinicians can initially watch most lamellar holes closely.

Stage 0 to 1 cases that warrant heightened attention include those seen with Amsler grid changes, subjective vision changes and (often hyper-) autofluorescent changes at the foveal base, disruption to the contiguous line of photoreceptor segments overlying the retinal pigment epithelium and progression.

Stage 2 to 4 macular holes are described as full-thickness and typically require surgical intervention. Unlike lamellar macular holes, full-thickness macular hole formation is an anterior-to-posterior tractional disorder.

Finding Closure

Most patients—about 90%—with later-stage macular holes can achieve closure with pars plana vitrectomy (PPV) and internal limiting membrane (ILM) peeling; still, 10% experience a failed procedure and remain at risk for vision loss.[2]

The goals of macular hole surgery include closure of the hole, improving visual function and minimizing future visual deterioration. It is imperative to refer and consider surgical invention promptly, considering delays in surgical repair once vision loss has occurred lead to sub-optimal visual improvement.

Since macula hole formation is caused by abnormal adhesion to the foveal margins associated with impending PVD, the most critical step of surgical repair is complete PVD to remove all anterior-to-posterior traction at the site of the macular hole.

Much like repairing a bone fracture, whereby a cast or splint stabilizes the reset break, a gas bubble added at the end of surgery helps close the macular hole via two mechanisms: (1) surface tension (encouraging the edges of the macular hole to re-approximate) and (2) dehydrational force (assisting in maintaining hole closure).

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Although most patients are advised to keep a facedown position for a week, many macular holes close within one to two days post-op, negating the need for facedown head positioning for a full a week. This is assuming the gas fill of the vitreous cavity is significant enough to contact the macular hole at that time.

By being as efficient as possible and limiting frequent re-entries into the eye via the vitrectomy ports, the surgeon can preserve the anterior hyaloid, thereby limiting the contact of gas with the posterior lens capsule. In Dr. Girschek's experience, an extremely small percentage of patients undergoing fluid-gas exchange (and not being restricted to facedown head positioning) require cataract surgery during the first three months post-op. Additional facedown positioning is not required for full closure of the hole, in his estimation. However, clinicians should still advise patients to avoid a supine head position to prevent a buoyant gas bubble from shallowing the anterior chamber and potentially causing acute angle-closure glaucoma.

Macular hole surgery has an extremely high success rate, and the sooner surgical repair is performed after macular hole formation, the better the visual outcome. Although historically necessary, facedown head positioning may not be required to achieve successful surgical repair, assuming careful attention is paid intraoperatively to complete vitreous removal and fluid drainage. Efficient and expedient surgical repair may also reduce the risk for post-op cataract formation due to gas contact.

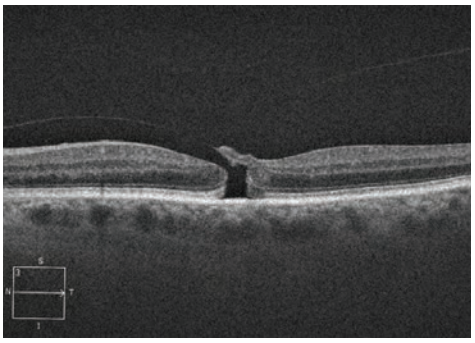

Fig. 2. This stage 2 macular hole has an attached posterior hyaloid.

Patient in a Better Position

Case by Drs. Girschek and Shechtman

A 62-year-old white female was referred by an optometrist for macular hole evaluation in her left eye following vision loss for the past three months (*Figure 1*). Her best-corrected visual acuity measured 20/400 OS, and OCT revealed a full-thickness macular hole. Due to the clinical presentation, surgical intervention was recommended with PPV, ILM peel, indocyanine green (ICG) dye and fluid-gas exchange. Facedown positioning was not encouraged. When the patient returned a week later, her clinical exam findings and OCT images showed full closure of the hole. She was asked to return to the clinic in one month for follow up.

Intervene Sooner

Comments by Dr. Haynie

Since its introduction in the 1990s, PPV has been a go-to management option for full-thickness macular holes (stages 3 and 4) in which the posterior hyaloid has separated from the retinal surface.[3] An important yet often overlooked step in the surgery is ICG-guided peeling of the ILM. This not only aids in successful closure of the macular hole, but it also reduces potential failures.

We follow a management regimen in our practice similar to that of Drs. Girschek and Shechtman, with one key difference. While their group does not require facedown positioning for all patients, the surgeons in my group continue to comply with the recommended facedown positioning for 72 hours post-op.

The case presented by Drs. Girschek and Shechtman highlights the classic clinical appearance and OCT images of a stage 4 macular hole pre- and post-surgical repair—something we are all familiar with. Less known is the management strategy necessary for a stage 2, or impending, macular hole, defined as a full-thickness defect whereby the posterior hyaloid is still attached (*Figure 2*). Here's how we handle these cases in my practice:

A 66-year-old Caucasian female was referred for an evaluation with a complaint of blurred vision, a central scotoma and metamorphopsia for two months. Her best-corrected visual acuity measured 20/40-2 OD, and she missed letters during her acuity assessment. She was diagnosed with an impending macular hole (*Figure 3*). In the case of a stage 2 macular hole, the current treatment options can include close observation, intravitreal use of Jetrea (ocriplasmin, Thrombogenics), pneumatic vitreolysis or more traditional PPV with fluid-gas exchange.

Jetrea was approved by the FDA in 2012 for the treatment of symptomatic vitreomacular adhesion. The objective is to induce a PVD, and the medication has a reported success rate for all macular hole types of approximately 27%.[4] Success with Jetrea for stage 2 macular holes specifically is approximately 40%.[4]

However, after Jetrea became more popular among retina specialists, an increasing number of complications were reported, including retinal detachment and permanent loss of central vision or scotoma development. Thus, its use has diminished, and our practice no longer

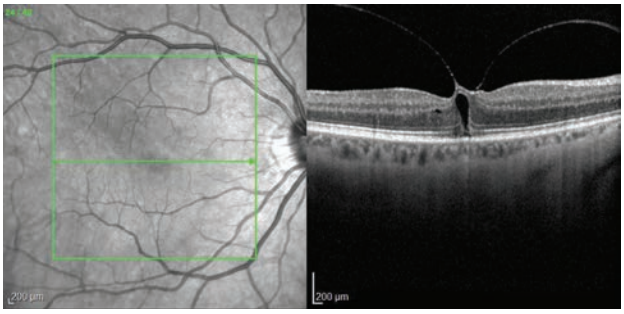

Fig. 3. OCT imaging revealed an impending macular hole as the cause of this patient's visual complaints.

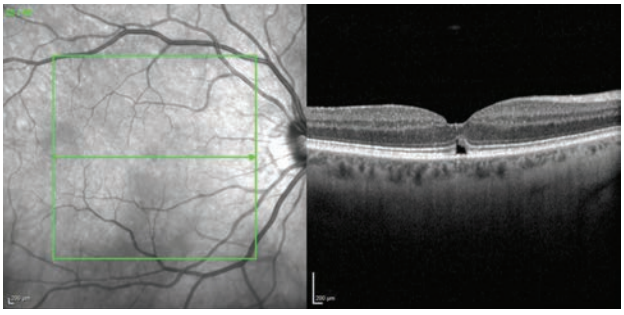

Fig. 4. Pneumatic vitreolysis helped release the posterior hyaloid and close the macular hole.

offers this as a treatment option.

Our patient elected to have a pneumatic vitreolysis. Thirteen days post-treatment, OCT imaging confirmed release of the posterior hyaloid with macular hole closure, except for a small subretinal cleft—a finding that typically resolves over time (*Figure 4*).

Pneumatic vitreolysis is an in-office outpatient procedure. The surgeon injects C_3F_8 or SF_6 gas into the vitreous cavity, followed by a therapeutic anterior chamber parancentesis. The patient is instructed to comply with facedown positioning for five days. This procedure comes with several advantages, including: its outpatient nature, lower cost compared with PPV and a reported success rate of 60% to 100%.[5]

Clinicians should consider pneumatic vitreolysis for selective cases with a stage 2 impending macular hole. ■

Dr. Girschek is a retina surgeon at Retina Macula Specialists of Miami.

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Sensory Motor Testing Defined

Know when you need more than a basic exam—and how to code for it.

By John Rumpakis, OD, MBA, Clinical Coding Editor

Primary eye care practices often encounter issues relating to the neurological system. One of the first signs of a serious neurological issue may, in fact, be extraocular muscle (EOM) abnormalities that manifest during diagnostic testing. While basic sensory motor testing is part of any comprehensive ophthalmic examination, performing extended testing is not—but that doesn't mean it doesn't have a place in optometric practice.

The sensorimotor examination is a critical diagnostic test in some instances when you suspect a neurological issue. However, many individuals either misconstrue its use or simply don't understand its definition and the coding requirements associated with it. Here's what you need to know.

Part of the Routine

Basic sensory motor testing evaluates and assesses the ocular range of motion to determine if the EOMs move together in the various cardinal positions of gaze. This is typically indicated in the medical record of a comprehensive exam (920X4) as ocular motility. Documentation of ocular alignment is also required in the basic examination and is often noted as ortho(phoric) when normal and eso(phoric or tropic) or exo(phoric or tropic) when abnormal. In pediatric patients, the typical notation of fix and follow (F&F) is generally used to describe both visual acuity and gross motility.

As a required element of a 920X4 examination, the basic sensory motor exam represents incidental testing and is not separately reimbursed. Additionally, it is also included as an element of the evaluation and management (E&M) codes when indicated and is listed as EOM.

Beyond the Basics

When you need a quantitative sensorimotor examination, using prisms to measure ocular deviation, and the accompanying sensory function test (e.g., stereo rings, stereo fly, Worth 4-dot, Maddox rod), you should use 92060 - sensorimotor examination with multiple measurements of ocular deviation (e.g., restrictive or paretic muscle with diplopia) with interpretation and report (separate procedure).

This 92060 code exists to define a more extensive test and may be billed separately and in addition to the 920X4 or 992XX examination.

Keep in mind that, as with HCPCS/CPT codes, this one includes a parenthetical statement that the examination represents a "separate procedure." The inclusion of this statement indicates that the procedure can be performed separately but should not be reported when a related service is performed.

As part of the documentation of a 92060, you must make written order in the patient record that the basic sensory motor test as described above is abnormal and that more extensive testing is indicated. This is what constitutes the medical necessity required to order and perform the extended sensory motor testing. In accordance with CPT rules, an interpretation and report of the test results, effect on the patient's condition and course of treatment are required as they would be for any special ophthalmic test performed. The notations for the test should be clearly identifiable and distinct from any office visit notes. The interpretation and report should also be clearly distinct from the assessment and plan of the office visit notes.

Repeat testing is indicated with a clear medical necessity based on new symptoms, disease progression, new findings, unreliable results from earlier tests or a change in treatment. In most cases, more extensive testing is required when the basic information gathered from the 920X4 or 992XX examination is insufficient to properly assess the patient's status. Repeat testing is not expected for a patient who is stable, presents with no complaints or has a condition that is properly controlled.

Neurological assessments are part of every basic examination, but an extended test that both qualifies and quantifies the sensory and motor aspects of the extraocular muscles is another valuable diagnostic tool in every OD's toolbox. Don't be afraid to code and bill for this separately, if the need is there and you perform the test properly. ■

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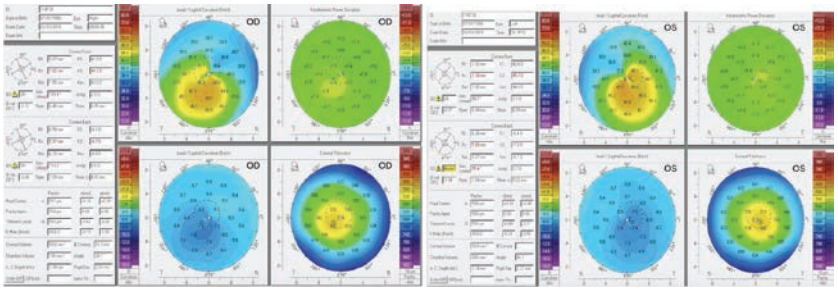
When 20/20 Isn't Enough

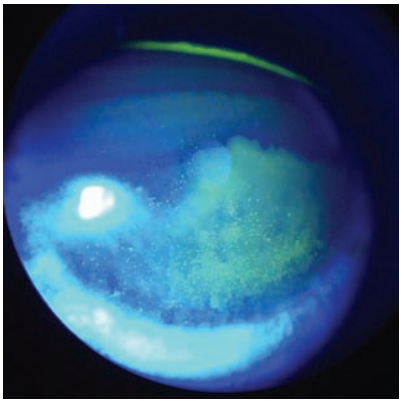
Patients may be correctable to excellent acuity, but underlying pathology could still distort, interrupt or disturb their vision. By Julie Tyler, OD, and Thuy-Lan Nguyen, OD

The foundation of optometry is evaluating a patient's visual status by asking, "Which is better, 1 or 2?" The goal is usually to achieve 20/20, or close, as their best-corrected visual acuity (BCVA). When a patient has reduced vision, it is our mission to determine the cause. Sometimes we discover significant pathology even when patients are correctable to 20/20. It is not unusual for these patients to have symptoms of visual distortion, glare, halos, fluctuating vision, ocular discomfort and headaches.

Here, we review conditions where a patient may be correctable to 20/20, but could be ultimately diagnosed with ocular pathology. If these conditions are not detected, there could be significant, long-term visual or systemic consequences.

Visual acuity measured with a simple pinhole occluder will help determine if a refractive change is likely to improve vision. We will discuss supportive ancillary testing such as corneal topography, color vision testing and optical coherence tomography (OCT), as well as indications of when to use technology to diagnose ocular pathology when the primary visual symptom or findings is not decreased BCVA.





Figs. 1 and 2. Above, these corneal topography scans show a patient with subclinical keratoconus. The patient here was refractable to 20/20 but complained of excessive glare and halos. Note the inferior steepening and the thin central corneal thickness.

Fig. 3. At left, this patient demonstrates diffuse corneal staining with sodium fluorescein due to toxicity from multipurpose contact lens solution.

Cornea

Keratoconus is a progressive corneal ectasia characterized by a weakening and thinning cornea.[1] While the vast majority of keratoconus presents bilaterally, doctors find a high prevalence of asymmetry between the eyes. Research from 2010 defines forme fruste keratoconus as the contralateral eye in unilateral keratoconus

without any ectatic changes.[2]

In mild cases, such as subclinical keratoconus or forme fruste keratoconus, no obvious biomicroscopy signs—such as apical scarring, Vogt's striae, Fleisher's ring or Munson's sign—may present (*Figures 1 and 2*). These patients may be refractable to 20/20, but still report symptoms of debilitating glare, ghost image, pseudodiplopia and a history of active eye rubbing. For these patients, central keratometry readings measured by standard autorefractors are often misleading.

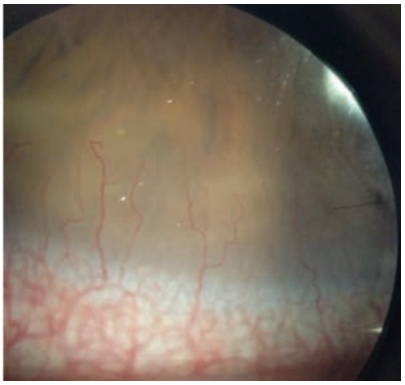


Fig. 4. This asymptomatic patient's corneal neovascularization is due to contact lens overwear.

To help ensure early and accurate detection, corneal topography is critical. Specifically, a corneal thickness map and the difference in keratometry readings between the thinnest and thickest areas may be required. Patients with forme fruste keratoconus will have a lower corneal thickness and a higher keratometric differences compared with normal eyes.[3]

Glare testing and contrast sensitivity can be important diagnostic testing for early forme fruste patients. A 2016 study found that contrast sensitivity was more affected and is a more sensitive indicator of visual function in keratoconus compared with BCVA.[4] Fortunately, form fruste keratoconus tends to progress slowly, if at all. But the chronic symptoms may still affect a patient's quality of life. Therefore, proper diagnosis and management can have a significant effect on a patient's functional vision.

Contact Lenses

Optometrists are well aware that bacterial corneal ulcers from contact lens overwear can be devastating to a patient's vision. In cases of acute bacterial keratitis, we immediately advise a patient to discontinue contact lens wear. These conversations must be initiated when patients have signs of contact lens overwear that have not yet affected vision, even if they are asymptomatic.

A variety of ocular findings are associated with contact lens overwear that may present prior to decreased visual acuity but may affect the quality of a patient's vision, as well as increase that patient's risk for more serious complications. For example, even when a patient is correctable to 20/20 and asymptomatic, we should evert the upper eyelid to evaluate the superior palpebral conjunctiva and use sodium fluorescein to check for corneal integrity due to associated risks of new ocular symptoms and complications (*Table 1*).

Often, patients will continue to experience 20/20 visual acuity but have visual complaints regarding the quality of their vision when they undergo punctate epithelial keratopathy (PEK) related to contact lens wear or dry eye. Any contact lens wear can potentially affect the integrity of the corneal surface and cause damage to corneal epithelial cells.

The most common and convenient clinical method for visualizing and diagnosing corneal damage is with the use of sodium fluorescein.[5] The extent of corneal staining from contact lens wear can vary depending on the degree of insult. Diffuse superficial staining covering the entire cornea can result from toxicity to preservatives in multipurpose solutions (*Figure 3*). Patients with this low-grade staining may not complain of any blurred vision, ocular discomfort or dryness.[5]

Contact lens wear can also compromise optical clarity by causing corneal edema, which is a hypoxia-related corneal complication. Patients with microcystic edema from contact lens wear often only complain of halos and glare around lights (Sattler's veil). At times, subtle edema may not be easily visualized on slit lamp examination.

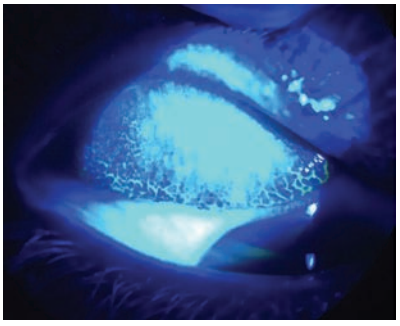


Fig. 5. By everting this patient's upper lid and using sodium fluorescein dye with a cobalt blue light, you can see their giant papillary conjunctivitis from contact lens overwear.

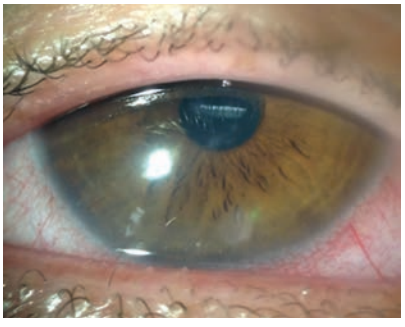


Fig. 6. This patient's anterior segment evaluation shows a case of CLARE.

Measuring central corneal thickness with pachymetry or measuring corneal density with instruments such as Scheimpflug imaging via Pentacam (Oculus) and anterior segment OCT (AS-OCT) may also be useful to assess the full impact of contact lens wear on the cornea.[6]

Corneal neovascularization (CNV) can result from contact lens wear, corneal infections or corneal inflammatory conditions (*Figure 4*). When hypoxia stimulates vessel growth from the pericorneal vascular plexus into the cornea, the result is new vascularization in an otherwise clear structure.[7]

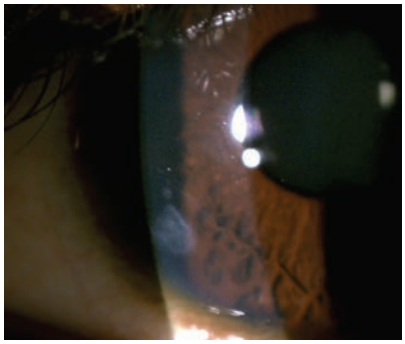


Fig. 7. You can see this patient's residual peripheral corneal scar from a resolved contact lens–associated corneal infiltrative event.

A Japanese research team classified CNV based on grades of severity. Grade 1, the least severe, involves only the peripheral cornea. Grade 2 indicates peripheral and mid-peripheral involvement. Grade 3 signifies modest vascularization involving the entire cornea. Grade 4 is the most severe and indicates massive vascularization covering the entire cornea.[8]

Many patients with mild CNV are asymptomatic with 20/20 visual acuity. More severe, visually significant CNV associated with contact lens wear is less common with high oxygen permeable silicone hydrogel materials, especially daily disposable modalities. However, it can still occur and progress, especially if the contact lenses are abused and overworn.

Giant papillary conjunctivitis (GPC)—or contact lens–induced papillary conjunctivitis (CLPC)—is a potential complication of contact lens wear. Patients may present with 20/20 vision but often complain of redness, itching, contact lens intolerance, excessive lens movement and mucous discharge.[9] GPC prevalence was significantly higher with conventional hydrogel contact lenses.[9]

We see less severe GPC with frequent replacement and daily disposable lenses, especially silicone hydrogel materials, which attract more lipid than protein deposits. Therefore, many practitioners may not look for GPC when a patient is 20/20. The definitive way to diagnose GPC is by everting the eyelids to visualize hyperemia and a papillary reaction in the palpebral conjunctiva. Instilling sodium fluorescein and viewing the upper tarsal conjunctiva with cobalt blue light can enhance the view of the papillary reaction (*Figure 6*). Other concurrent allergic and atopic conditions can increase the risk of developing GPC.

Contact lens–induced acute red eye (CLARE) is an inflammatory response to contact lens wear more often seen in patients who overwear their lenses. Patients may complain of end-of-day redness, discomfort or contact lens intolerance, glare and photophobia without any blurred vision. Biomicroscopy generally reveals circumlimbal or diffuse conjunctival injection.[10] CLARE often resolves without topical medications by discontinuing contact lens wear temporarily, or decreasing the hours of contact lens wear. Additionally, switching to higher oxygen permeable silicone hydrogel materials can also reduce symptoms of CLARE. However, if patients continue to overwear their lenses, mild CLARE may progress to more sight-threatening conditions such as infectious keratitis.

Contact lens–associated corneal infiltrative events (CIEs) are self-limiting inflammatory responses that indicate a host's immune response to infection or an immune response to antigens (*Figure 7*).[11] CIEs are often used to describe a sterile keratitis. However, investigators found that when cultured, sterile infiltrates are often positive for bacteria.[12]

Exotoxins released by gram-positive bacteria such as *Staphylococcus aureus* can cause migration of inflammatory cells, including polymorphonuclear leukocytes, which leads to infiltrate formation.[12] Young male patients may be more likely to develop contact-lens–related complications due to poor compliance

Table 1. Coding for CL Complications

Possible complications of contact lens wear in patients with 20/20 acuity	ICD-10 code (x indicates OD, OS, OU)
Punctate epithelial keratopathy	H16.14x
Corneal edema due to contact lens	H18.21x
Corneal neovascularization	H16.40
Giant papillary conjunctivitis	H10.41x
Red eye associated with contact lens	H57.89
Infiltrate of cornea associated with contact lens	H18.21x
Marginal corneal ulcer	H16.04x

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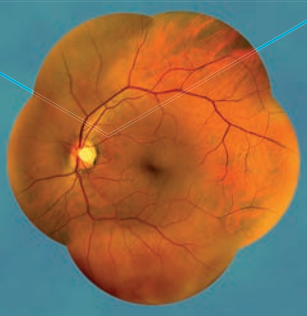

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Table 2. Pharmaceutical Side Effects

Medication	Visual Symptoms	Associated Conditions
Accutaine	Blurriness, double vision, tunnel vision, decreased night vision, CL intolerance	Dry eye
Amiodarone	Green haloes, glare, color vision changes	Light sensitivity due to corneal verticillata, vortex keratopathy, crystalline lens changes, IIH, optic neuropathy
Antihistamines	Fluctuating vision, CL intolerance	Dry eye
Chloroquine, Hydroxychloroquine	Ring of disrupted paracentral vision	Corneal verticillata, vortex keratopathy, bull's-eye maculopathy, anterior subcapsular lens change
Corticosteroids	Glare, metamorphopsia	Cataracts, delayed wound healing, increased IOP, IIH, serous chorioretinopathy
Digoxin	Color vision changes, visual sensations, flickering vision, blur, photophobia	Reduction in IOP, optic neuritis
Doxycycline	Photosensitivity, color vision defects, diplopia	IIH
Alendronate sodium	Associated inflammatory eye disease during the first 30-90 days	Uveitis, scleritis
Indocin	Corneal verticillata (vortex keratopathy), light sensitivity	RPE changes, IIH
Moxifloxacin	Redness, photophobia	Distorted pupils due to bilateral acute iris transillumination
Oral contraceptives	Corneal curvature steepening, reduced tears	Dry eye, contact lens intolerance, IIH, retinal vascular occlusions, TIA
Tamoxifen	Ant. segment: corneal verticillata, vortex keratopathy	IIH, macular deposition (white crystalline)
Topiramate	Large myopic shift	Secondary "angle closure" associated with choroidal infusion
Sildenafil	Color perception changes ("bluish" tinge), light sensitivity, photopsia	Serous maculopathy, optic neuropathy
Vigabatrin	Binasal visual field defects	Optic nerve pallor, retinal nerve fiber layer defects, ERG abnormalities, decreased Arden index on EOG

and greater risk-taking behaviors, according to some research.[13] The incidence of CIEs in daily soft contact lens wearers is approximately 3%.[11] When reviewing extended wear patients, the incidence of symptomatic CIEs ranges between 2.5% to 6% and, when including asymptomatic CIEs for extended wear, the incidence increases dramatically to 20% to 25%.[11]

Unlike CIEs, contact lens peripheral ulcers (CLPU) are focal excavations of the epithelium, which can cause infiltration and necrosis of the anterior stroma near the corneal margins. Patients with CLPU may be asymptomatic with 20/20 visual acuity or may complain of redness, pain or foreign body sensation. Microbial keratitis (MK) can be visually significant and involves actively proliferating bacteria, which requires treatment.[11]

Dry Eye Disease

Perhaps one of the more common types of patients with 20/20 vision and concurrent complaints are those with dry eye disease (DED). In the Tear Film and Ocular Surface Society's Dry Eye Workshop report from 2017, dry eye is defined as "a multifactorial disease of the ocular surface characterized by a loss of homeostasis of the tear film, and accompanied by ocular symptoms, in which tear film instability and hyperosmolarity, ocular surface inflammation and damage, and neurosensory abnormalities play etiologic roles."[14]

This thorough and complex definition reflects the variety of challenges we may have managing these patients. Individuals suffering from DED may vary from only having mild symptoms and signs to severe ocular surface complications.

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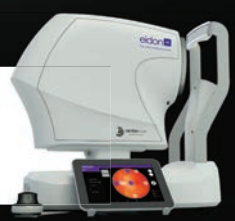

To identify patients at risk for DED and associated ocular symptoms such as fluctuating vision, light sensitivity and unstable refractions, start by asking triaging questions and identifying risk factors such as smoking, contact lens wear or at-risk medications. A dry eye survey such as the Ocular Surface Disease Index (OSDI) or the Dry Eye Questionnaire-5 (DEQ-5), due to its short length and discriminative ability, can be given prior to the examination.[14,16] Additionally, for soft contact lens (SCL) patients, a preferred survey would be the eight-item Contact Lens Dry Eye questionnaire (CLDEQ-8), which has repeatable results and comes closest to identifying SCL wearers who would likely benefit from clinical management of dry eye.[17,18]

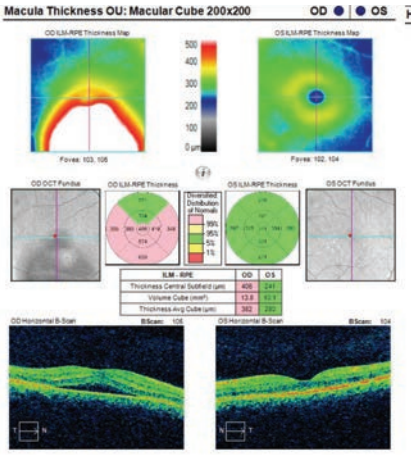

Fig. 8. This macular cube image demonstrates a patient with RPE disruption with CSCR.

When patients are identified early with DED, additional attention may be warranted during refraction to assess for consistency of responses, as the tear film is the first refractive component of the eye. When asked to blink during refraction and visualize the changes in visual quality, patients often recognize the contribution of DED to their symptoms and may be more apt to follow through on therapy.

Systemic Medications

Many systemic medications can impact vision—some of which may not directly affect acuity but can cause changes in color vision, quality, transient visual obscurations or other atypical symptoms (*Table 2*). When patients have complaints that are new or different—even if they have 20/20 BCVA—consider whether any of the patient's medications is likely to play a role.

One ocular condition associated with many medications is idiopathic intracranial hypertension (IIH), historically referred to as pseudotumor cerebri. It may be associated with tetracycline, doxycycline, minocycline, corticosteroids, oral contraceptives, amiodarone, tamoxifen, levothyroxine and high-dose vitamin A therapy. These patients often present early in the condition with good acuity but with headaches, possible double vision and pulsatile tinnitus.

Due to the finding of bilateral optic nerve head elevation customarily observed with IIH and the high—risk differential of true papilledema, patients with this finding warrant immediate referral for brain and orbit imaging followed by a lumbar puncture to rule out increased intracranial pressure.

Patients may complain of a blurred or distorted area in their vision and still have 20/20 BCVA and have a concurrent scotoma that affects the paracentral field. In this case, visual field testing may be especially helpful.

Visual field assessments ranging from threshold automated visual fields to Amsler grids may be beneficial in identifying and targeting defects. Additionally, in cases where blur or distortion is reported, it is important for the doctor to personally assess the individual's demographic status, review systemic medications, personality type and general observations. For example, an elderly patient with a history of smoking and a complaint of distortion is more likely to have complications associated with age-related macular degeneration to explain the scotoma whereas a younger, highly stressed individual may be more likely to have a diagnosis of central serous chorioretinopathy (CSCR).

Central Serous Chorioretinopathy

These patients nearly always report a spot in their vision but, depending on the location of the scotoma, may have decreased BCVA or may retain 20/20 visual acuity.

Though the exact etiology for CSCR is unknown, suspected associations include type-A personalities, corticosteroid use, pregnancy and a variation associated with optic nerve pits.[19-23] For patients using corticosteroids and developing CSCR, it appears that both the choroid and retinal pigment epithelium (RPE) are involved (*Figures 8 and 9*). One proposed hypothesis is that some patients, due to idiosyncratic susceptibility of their individual mineralocorticoid pathway, may be more susceptible to CSCR while using steroids.

A detailed review of medications and history by the eye care practitioner is important for these patients as the types of steroids associated may vary widely—anywhere from oral steroids used to treat facial palsy, systemic lupus erythematosus or ulcerative colitis to steroid inhalers, epidural steroid injections and even dermatological/topical steroid use.[19-22]

When CSCR is suspected, an OCT of the area of elevation and associated tissues may be beneficial for

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a variety of reasons. Not only can an OCT help confirm the diagnosis of CSCR but it may also rule out other high-risk differentials and comorbidities (e.g., choroidal neovascular membrane).

Additional assistance from OCT measurements can help in understanding possible underlying associations with a diagnosis of CSCR.[23] In 2017, investigators described differences in OCT measurements of the choroid between eyes with CSCR associated with steroids vs. acute idiopathic CSCR during the acute phase.[23] The researchers measuremed between the outer border of the RPE and the chorioscleral border using SD-OCT and found that patients suffering with CSCR associated with steroids have significantly thinner OCT choroidal measurements than those with acute idiopathic CSCR.[23] In those with a steroid association, communication with both the patient and any managing physicians is key in reducing the patient's systemic "steroid" load and allowing the CSCR to resolve.

Initial management of patients with CSCR may include reassurance and observation or stress relief management, modifications of medications (steroids) for patients with associations and regular dilated examinations (three to six weeks) until resolution. However, for patients with chronic (persistent subretinal fluid for three to six months) or recurrent CSCR, various therapeutic options exist—from short-duration, multi-session subthreshold micropulse yellow laser to photodynamic therapy with vertoporfin to oral aldosterone antagonists.[24-26]

Even patients with 20/20 BCVA may have visual symptoms such as glare, halos, ocular pain, distorted vision, headaches and may also show signs of pathology while being asymptomatic—all concerns that warrant additional care. Patients with good vision may ultimately have other conditions such as keratopathies, maculopathies and optic neuropathies which require management. As eye care specialists, we can use technology to assist our patient's well-being. For example, corneal topography and OCT are now common equipment in many practices. Clearly, the integration of technology can change the way we diagnose and manage our patients. Assessing visual acuity is only a small part of what we are capable of. These examples show that refracting a patient to 20/20 is not always enough. ■

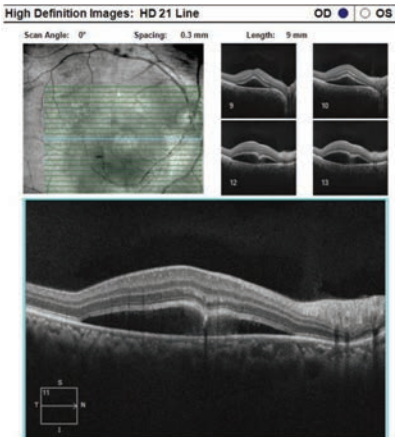

Fig. 9. This is a high-definition 21-line raster of the RPE disruption with CSCR from the same patient from page 46.

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Thyroid Eye Disease In Your Exam Lane

Whether managing associated side effects or referring and comanaging, you are integral to these patients' care teams.

By Bobby Saenz, OD, MS, Brett Mueller, DO, PhD, Anthony Vanrachack, OD, and Brett Davies, MD, MS

Thyroid-associated ophthalmopathy (TAO), also known as thyroid eye disease or Graves' ophthalmopathy, is the most common autoimmune inflammatory disorder of the orbit and periorbital tissue, with approximately three million Americans affected.[1,2] This prevalence is similar to that of glaucoma in the United States. Historically, TAO was limited to patients with Graves' disease and the clinical triad of orbital signs, hyperthyroidism and pretibial myxedema.[3] Now, research shows only 80% of patients with TAO have Graves' (hyperthyroidism); the other 20% consists of patients who are hypothyroid (10%) and euthyroid (5% to 10%).[4] Because TAO can precede, coincide with or succeed the diagnosis of thyroid dysfunction, optometrists need to be capable of making an early diagnosis, as TAO can be vision-threatening, impact a patient's appearance and result in loss of quality of life.[5,6]

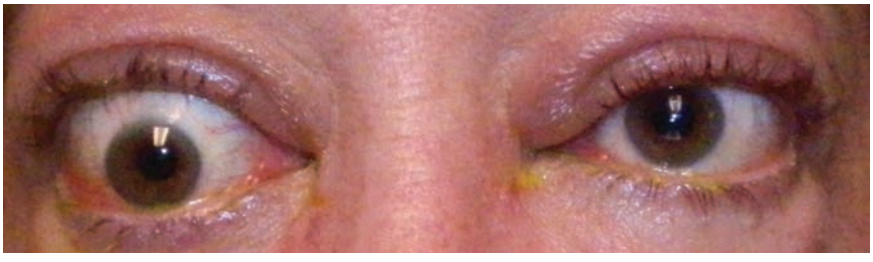

Fig. 1. Hypotropia of the right eye in a patient with TAO.

Where Exposure Starts

Three broad categories summarize the pathogenesis for TAO: (1) inflammation of the periorbital soft tissue; (2) activation of a subpopulation of orbital fibroblasts that are capable of undergoing adipocyte differentiation, leading to hyperplasia of adipose tissue; and (3) overproduction of glycosaminoglycans by orbital fibroblasts.[7] Orbital fibroblasts produce collagen and glycosaminoglycans in the extracellular matrix and create a strong polyanionic charge, which causes an extremely high osmotic pressure, leading to extraocular muscle swelling and possible optic nerve compression.[8]

TAO presents in two distinct phases: the active inflammatory phase and the inactive, or stable, phase. Typically, the stronger and more aggressive the active phase, the higher the likelihood of more severe sequelae.[9] The active inflammatory period typically lasts between six and 24 months and is followed by a quiet, stable, chronic fibrotic period.[10] During the early active phase, immunomodulators and radiotherapy may limit the progression of TAO. Once the disease is in the inactive state, surgery may help to improve cosmesis, comfort and function.

Early Clues

The clinical signs for TAO are vast, with most patients developing eyelid retraction, proptosis and other features of ocular exposure such as epiphora, photophobia, pain, grittiness, diplopia and decreased vision.[11] If the inflammation extends to the extraocular muscles, patients can develop conjunctival erythema, chemosis, restrictive extraocular myopathy, hypotropia, double vision and compressive optic neuropathy (*Figure 1*).

You can make the diagnosis of TAO based on presenting ocular signs and symptoms. Changes in appearance and exposure symptoms are the most common early findings in TAO.[9] Other symptoms are vague and often attributed to normal aging, especially in middle-aged women with eyelid swelling that is worse in the morning.[12]

The most common sign in TAO is lid retraction, which occurs in 82% of patients.[13] This may be due to increased sympathetic tone, overaction of the levator and superior rectus muscles to compensate for inferior rectus restriction, or inflammation and scarring of the levator complex (*Figure 2*).

The second most common sign is proptosis (62%), which is caused by expansion of the orbital fat, muscles or both (*Figure 3*). Proptosis can be measured with an exophthalmometer. Values greater than 18mm to 20mm for Caucasians, 16mm to 18mm for Asians and 20mm to 22mm for African-Americans suggest proptosis. Asymmetry of 2mm or greater also suggests proptosis.[14,15] If a clinician does not have an exophthalmometer, orbital CT scans can evaluate the amount of proptosis as well.

Proptosis and eyelid retraction can lead to increased corneal exposure and ocular surface disease—a common reason why patients make an eye appointment. If the disease state is aggressive, the proptosis and lid retraction can lead to lagophthalmos, exposure keratopathy, microbial keratitis and perforation. Patients at risk for perforation typically have significant lagophthalmos and an absence of Bell's phenomenon due to fibrosis of the inferior rectus muscle.

Restrictive extraocular myopathy (42%) and optic nerve dysfunction (6%) are also likely signs.[13] Dysthyroid optic neuropathy, sight-threatening but potentially reversible, should be suspected with desaturation of colors, afferent pupillary defects or decreased vision.[9] Most cases of dysthyroid optic neuropathy present with muscle enlargement but not necessarily severe proptosis.[9] Some believe patients with tight eyelids have limited anterior movement of the globe, putting them at greater risk for compression of the optic nerve.

Other signs include temporal flare, conjunctival injection and chemosis, Von Graefe's sign (delayed eyelid lag during downgaze) and Dalrymple's sign (widening of the palpebral fissures). Clinical signs are typically bilateral and symmetric but can be asymmetric or unilateral. In these cases, asking about symptoms

Table 1. TAO Evaluation

Clinical Exam	• Visual acuity • Color vision • Pupillary examination • Ocular motility • Hertel exophthalmometry • Intraocular pressure (primary gaze and upgaze) • Adnexal examination • Slit-lamp examination • Dilated fundus examination
Laboratory Tests	• Thyroid-stimulating hormone (TSH) • T3 • Free T4 • Thyroid peroxidase (TPO) antibodies • Thyrotropin-binding inhibitory immunoglobin
Imaging	• Orbital ultrasound to assess extraocular muscle size • Orbital CT without contrast or MRI to assess extraocular muscle size, orbital fat and proptosis

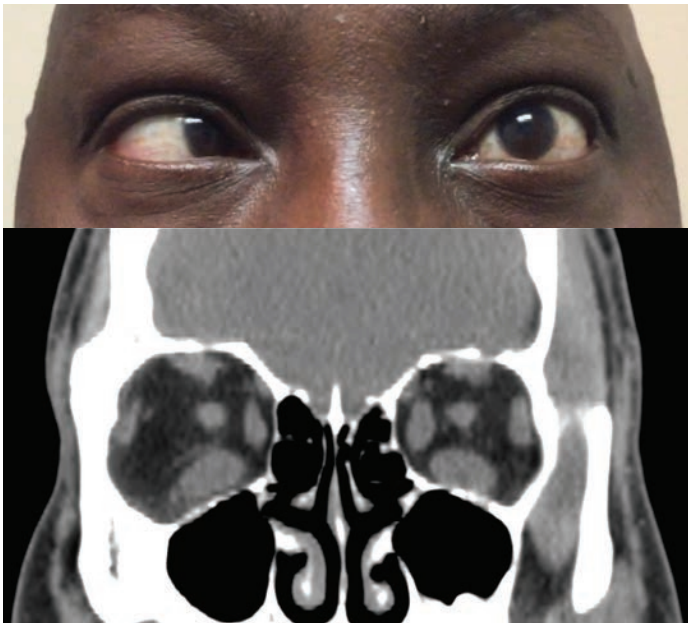

Fig. 2. Above, restricted movement of the left eye in temporal gaze. Below, coronal CT scan of the orbits shows an increased thickness of the left medial rectus and both inferior recti.

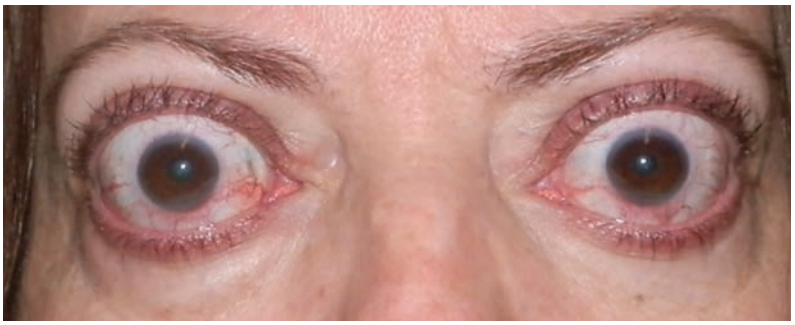

Fig. 3. Upper eyelid retraction and proptosis in a patient with orbital congestion (conjunctival injection) in the active phase.

of thyroid dysfunction—such as hair loss, heat or cold intolerance, weight changes, skin changes, memory problems and changes in their mood—can help make the diagnosis (*Table 1*).

Asymmetric Presentations

When you suspect a unilateral presentation and the patient reports little or no systemic symptoms, consider orbital imaging and thyroid serum testing. If you discover normal or mildly abnormal thyroid functions, consider alternate etiologies (*Figure 4*).[16]

The most common differential of TAO is orbital pseudotumor or non-specific orbital inflammation.[17] Non-specific orbital inflammation can present with TAO signs such as proptosis, extraocular muscle restriction, red eye and chemosis. Non-specific orbital inflammation is usually acute and painful, but if the clinical picture is unclear, orbital imaging can assist in distinguishing between these different disease processes.[16] For example, on CT scan TAO demonstrates the pathognomonic pattern of extraocular muscle enlargement while sparing the muscle tendon. Orbital pseudotumor, however, demonstrates enlargement of both the extraocular muscle and tendon.[9]

Keeping Score

Several clinical scoring systems exist for assessing the severity of TAO, including the mnemonics NO SPECS and VISA. These systems can help guide your evaluation and treatment of patients with TAO, but currently no system prevails as the gold standard.

Most clinicians know the classic NO SPECS classification: ***N***o physical signs or symptoms, ***O***nly signs, ***S***oft tissue involvement, ***P***roptosis, ***E***xtraocular muscle involvement, ***C***orneal involvement and ***S***ight loss (due to optic nerve compression).[18] While helpful, it does not assess clinical activity, nor does it provide sufficient information to document the disease between visits as a means for guiding management.[9,19]

The VISA classification—***V***ision, ***I***nflammation, ***S***trabismus and ***A***ppearance—was developed to permit grading of both clinical severity and activity based on both subjective and objective inputs (*Figure 5*).[20] This system helps direct appropriate management in a logical sequence by targeting the most relevant aspect of the disease affecting the patient. For example, vision dysfunction from the optic nerve is the first priority. VISA is also beneficial in assessing TAO and grading changes and can act as a guide for therapy.[20] The International Thyroid Disease Society has adopted it, and it is used in recent clinical trials.

Another popular method grades TAO from mild to

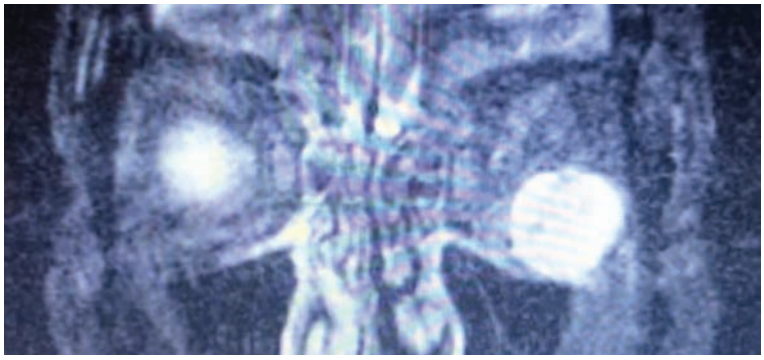

Fig. 4. This patient presented with unilateral proptosis. Exam findings suggested TAO; however, further testing, including this MRI, revealed an inferolateral postseptal orbital mass (hyperintense here) abutting the inferior and lateral rectus muscles. These features were highly suggestive of an orbital cavernous venous malformation (hemangioma).

Table 2. Clinical Activity Score

Clinical Findings	Score
Retrobulbar pain	0-1
Pain on extraocular muscle movement	0-1
Eyelid erythema	0-1
Conjunctival injection	0-1
Chemosis	0-1
Inflammation of caruncle	0-1
Eyelid edema	0-1
Total:	0-2: inactive TAO 3-7: active TAO

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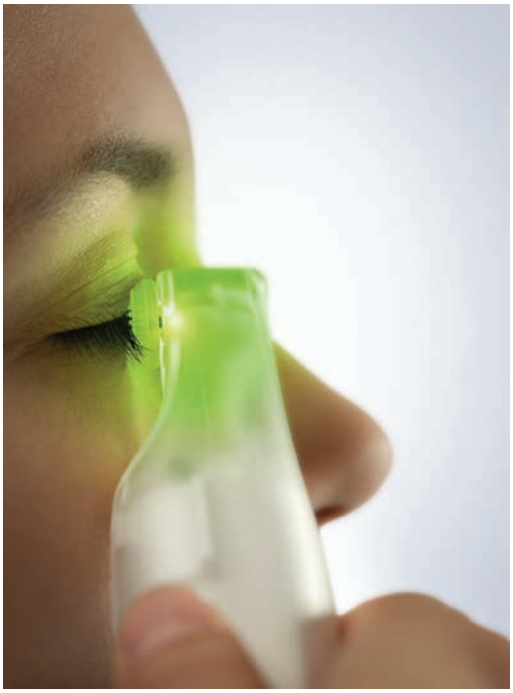

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severe.[21] The European Group on Graves' Orbitopathy (EUGOGO) defines mild disease as minimal eyelid swelling, lid retraction or proptosis with little or no extraocular muscle dysfunction. Moderate to severe TAO consists of some form of active disease with or without ocular motility dysfunction with diplopia and inflammatory features interfering with the ability to function. It may also include significant proptosis. Serious disease refers to sight-threatening conditions such as dysthyroid optic neuropathy and corneal ulceration.[9]

Timing is Everything

Evaluating the severity of orbital changes when diagnosing TAO is important, but this provides only a snapshot of the condition. Equally important is a temporal assessment of the course of the disease state and where it lies on Rundle's curve.

The key to determining if a patient with TAO is in a sight-threatening situation is assessing if the patient is in the active inflammatory phase or the stable, latent chronic fibrotic phase.

Active. Most patients who suffer vision loss from TAO due to corneal exposure or compressive optic neuropathy do so during the active inflammatory phase. You can use the clinical activity score (CAS) to identify active disease (*Table 2*).[18,22] Periorbital erythema and edema, conjunctival injection, chemosis, orbital inflammation and congestion, eyelid retraction, proptosis and diplopia typically characterize this phase. The active phase is typically a self-limiting disease process that lasts one year in nonsmokers and two to three years in smokers.[10]

When using CAS, note that severe disease complications, such as dysthyroid optic neuropathy, are still possible with low CAS scores, and patients with high CAS scores may have long-standing congestive changes that are unresponsive to any immunotherapy, requiring mechanical surgical decompression.

Latent. After the active phase plateaus, the patient enters the quiescent burn-out phase. This latent, chronic fibrotic phase presents with similar clinical findings as the active inflammatory phase (i.e., eyelid retraction, proptosis and diplopia) but does not have many of the inflammatory signs seen during the active phase (i.e., conjunctival injection and chemosis) (*Figure 6*).[23] These patients require frequent follow-up, as reactivation of inflammation can occur in 5% to 10% of patients over their lifetime.[24]

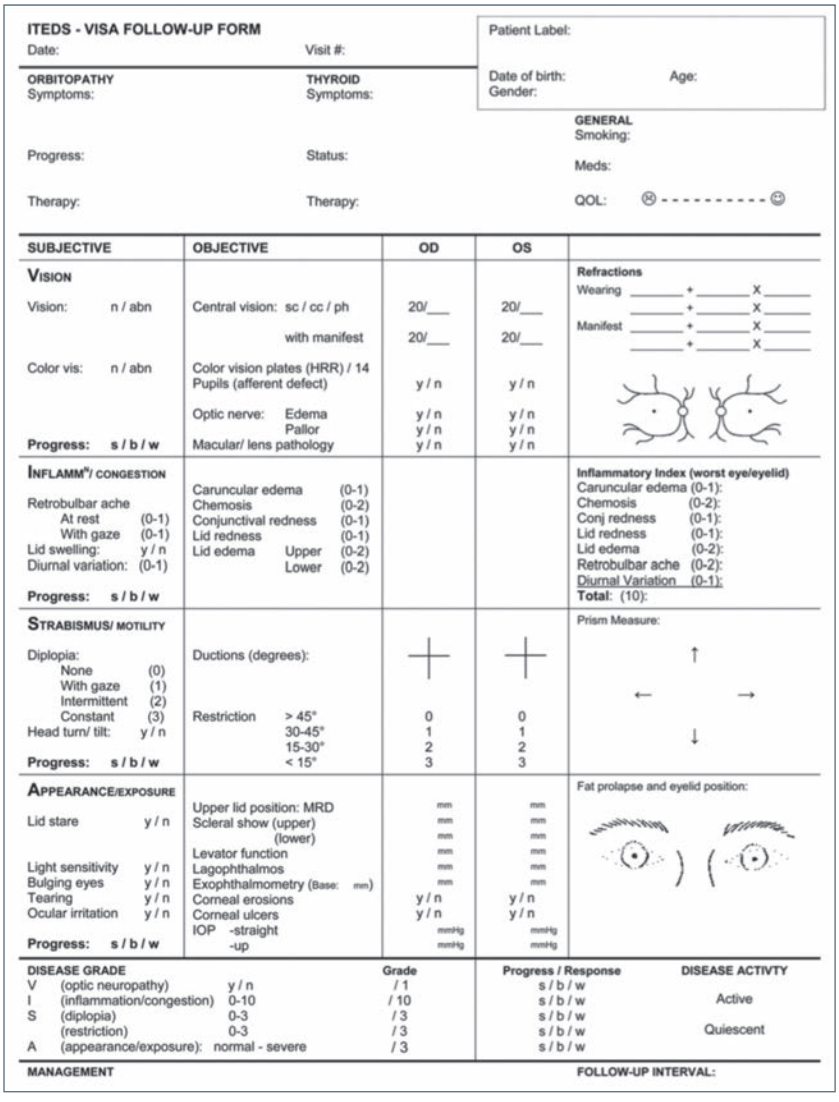

Fig. 5. The VISA classification form allows clinicians to monitor the severity and the activity of TAO.

Treatment Plan

Managing TAO includes restoring euthyroidism and smoking cessation.[6] Although euthyroidism is a treatment target, achieving well-controlled thyroid hormone levels will not improve TAO; however,

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Table 3. Management of Thyroid Eye Disease

Disease Stage	Recommendation
Mild	• Observation • Patient education and lifestyle changes such as: smoking cessation, salt restriction, elevation of head of bed, use of sunglasses, a gluten-free diet • Ocular surface treatment • Establish euthyroid state • Oral selenium
Moderate	• Topical cyclosporine • Eyelid taping at night • Moisture goggles or chambers • Prism glasses or selective ocular patching • Moderate-dose oral steroid therapy
Severe	• High-dose intravenous steroids • Surgical orbital decompression (followed by strabismus surgery, eyelid surgery or both) • Periocular radiotherapy
Refractory	• Steroid-sparing immunomodulators (e.g., rituximab)

poorly controlled thyroid hormone levels can make it worse. Thus, treatment of the thyroid gland must be simultaneous with but independent of treatment for the ophthalmopathy. Treatment includes a stepwise approach based on patient-reported symptoms, clinical examination and ancillary testing (*Table 3*).

Fortunately, the active inflammatory phase is usually mild and self-limited, often requiring only supportive intervention (e.g., artificial tears, topical anti-inflammatories). For most mild cases of TAO, simple treatment measures such as lubricants for lid retraction, nocturnal ointments for incomplete eye closure, prisms for diplopia and botulinum toxin injections for upper-lid retraction can be useful.

If the active phase is aggressive, it is essential to administer treatments that can decrease the severity and duration of the active disease process. Glucocorticoids, orbital radiotherapy and decompression/rehabilitative surgery are generally indicated for moderate-to-severe TAO and for sight-threatening optic neuropathy, cornea exposure and spontaneous globe subluxation. Other steroid-sparing immunomodulators, including rituximab, aim to treat the underlying molecular and immunological factors.[25]

Approximately 5% of TAO patients require some surgical procedure such as elective orbital decompression, eye muscle surgery or eyelid surgery.[26] Surgical intervention is best done once the active phase has

On the Horizon

Without any known biomarkers for the disease, thyroid dysfunction is currently detected with serum testing. However, a recent study investigating the tears of patients with TAO identified three new possible biomarkers, suggesting tears could be a source to diagnose TAO early and potentially even identify the amount of inflammation present.[25]

As for treatment options, researchers recently linked a newly discovered signaling pathway that involves immunoglobulin activation of insulin-like growth factor I (IGF-I) receptor (IGF-IR) in patients with Graves' disease.[31-33] This IGF-I signaling pathway can act synergistically with thyrotropin, enhancing its mechanism of action.[33] Studies show that an inhibitory antibody targeting IGF-IR can attenuate the actions of IGF-I, thyrotropin, thyroid-stimulating immunoglobins and immunoglobulins associated with patients with Graves' disease.[34,35] These observations led to the development of a new pharmacological compound called teprotumumab.[36] This compound is a fully human IGF-IR-inhibitor monoclonal antibody that can halt the signaling pathway of IGF-I and decrease the body's sensitivity to thyrotropin, thus lessening the signs and symptoms associated with Graves' disease.[36]

This led to the first double-masked, randomized, multicenter, placebo-controlled trial for patients with moderate and severe TAO.[36] When compared with placebo, teprotumumab demonstrated improvements in proptosis and other signs and symptoms associated with TAO.[36] Though the long-term benefits and safety profile are still under investigation, the results are promising. The FDA has granted this agent the fast-track designation, orphan drug designation and breakthrough therapy designation for Graves' orbitopathy. These could assist in fast-tracking this medication's review process through the FDA once the manufacturer submits a biologics license application. You should familiarize yourself with this new potential medication, as it will likely be the standard of care in treating patients with TAO in the near future.

resolved and the patient has been in the stable chronic fibrotic period for at least six months.[10] Complications associated with the use of blepharoplasty in active TAO can result in massive inflammation, orbit muscle involvement and overall unpredictable procedure results.[27] Thus, clinicians should rule out early and active TAO before recommending patients for lid surgery.[27]

It is important to note that medical therapy options for patients with moderate-to-severe TAO are lacking, with no current FDA-approved therapy. High-dose glucocorticoids, orbital radiotherapy and immunomodulators can reduce the amount of ocular and orbital inflammation associated with this disease, but their effect on proptosis is minimal, and their systemic

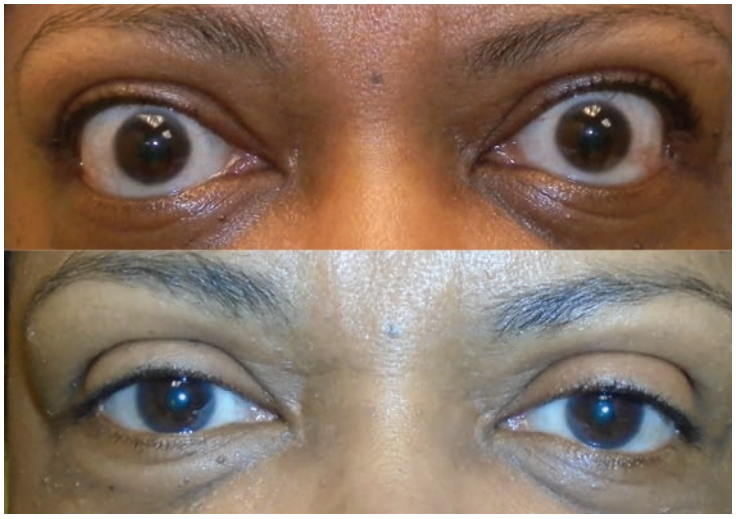

Fig. 6. Before (above) and after (below) orbital decompression of a patient in the stable, inactive phase.

TAO and Smoking

Patients with Graves' disease who smoke have a five-fold higher risk of developing TAO than those who do not smoke.[37] When orbital fibroblasts are exposed to cigarette extract, there is a dose-dependent statistically significant increase in glycosaminoglycan production and adipogenesis.[38] Evidence also suggests that smoking cessation reduces the severity of TAO and increases the chance of these patients having a favorable response to treatment.[39] It is crucial that ODs have a discussion about smoking cessation with all patients with Graves' and TAO.

side-effect profile can be high.[28-30] Many patients with TAO, despite high-dose glucocorticoid treatment and radiotherapy, do not improve and go on to develop compressive optic neuropathy, corneal exposure or orbital congestion.[31,32]

Clinicians are still on the hunt for alternative treatment modalities that will overcome the current treatment limitations. ■

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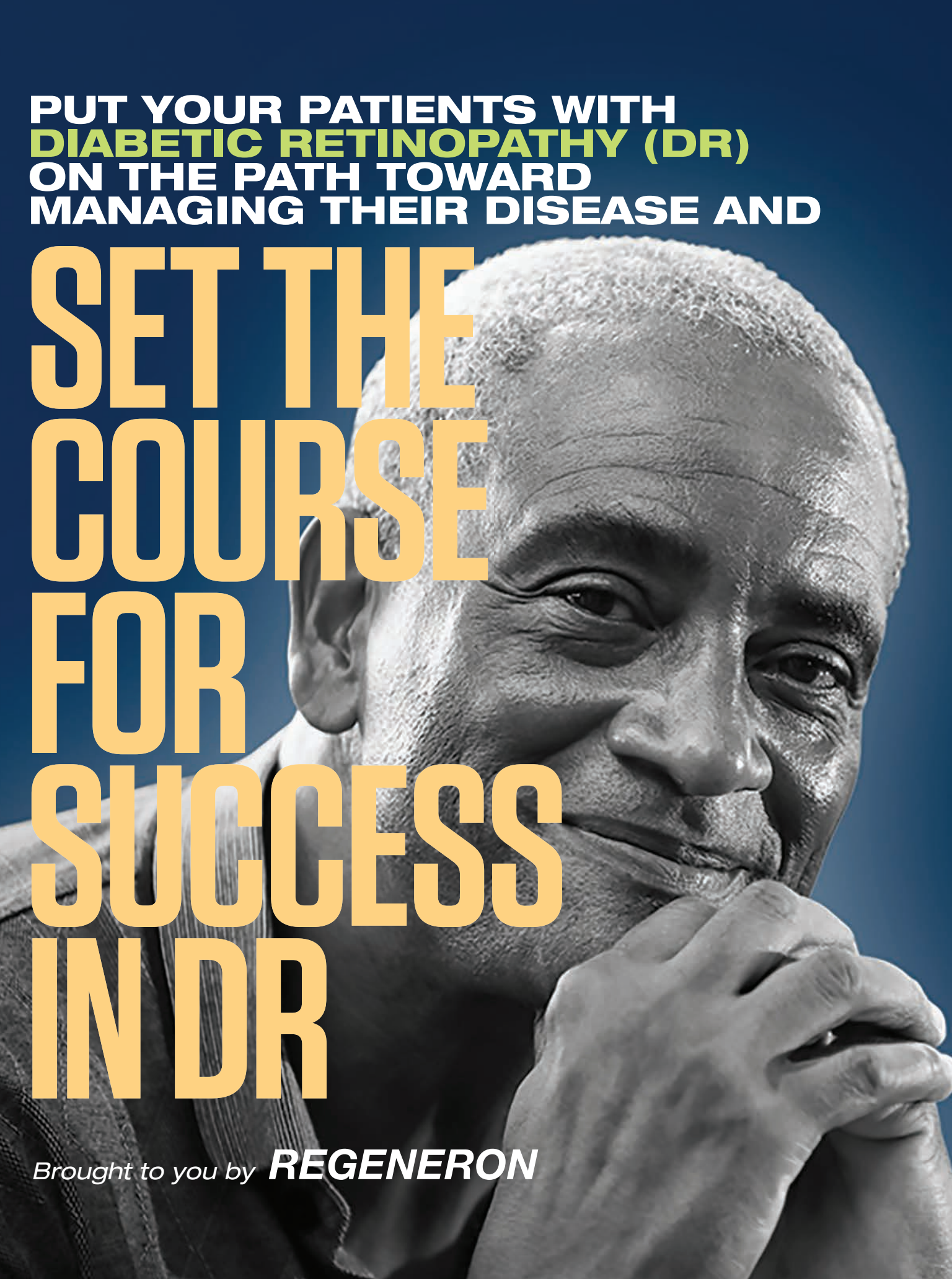

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Can You Differentiate These Tough Glaucoma Cases?

Just because it looks like POAG doesn't mean it is. Here's how you can get to the bottom of even the most difficult diagnoses.

By Brian D. Fisher, OD, David W. Johnson, OD, Austin R. Lifferth, OD, and April J. Fisher, OD

In 2010, 2.72 million Americans had glaucoma—1.9% of the US population older than 40 at the time.[1] This number is projected to increase to four million by 2030 and 6.3 million by 2050.[1] However, open-angle glaucoma may be over-diagnosed in as much as 50% to 60% of our patients.[2-5] While studies show that patients with a family history of glaucoma, are older in age or who are African American are more likely to be over-diagnosed, other clinical features often lead to over-diagnosis as well, such as larger nerves (and resultant larger cupping) and intraocular pressure (IOP) above 21mm Hg.[2-5] In addition, glaucoma may also be misdiagnosed when the patient's history is not taken into proper context or the neuroretinal rim is not properly evaluated.

These cases highlight the challenges of differentiating glaucoma from ocular hypertension or other conditions—and the importance of a thorough medical history and clinical exam.

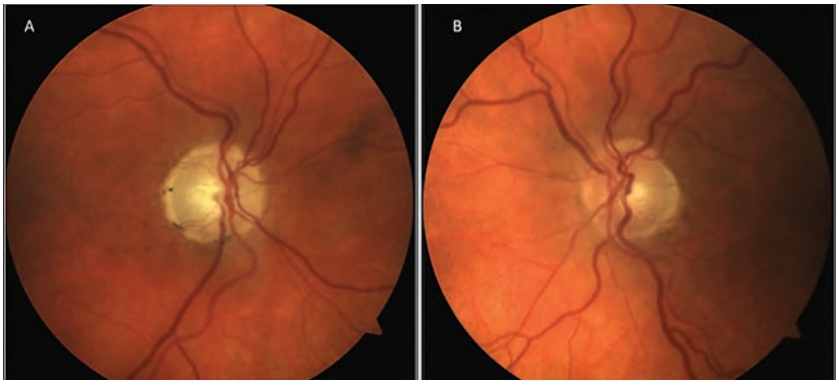

Fig. 1. The patient's optic nerve fundus photo shows evident neuroretinal rim pallor OD (A) and early glaucomatous cupping OS (B).

Case 1: Trauma Drama

A 58-year-old male was referred for a glaucoma evaluation due to enlarged cupping OU (*Figure 1*). His review of systems and personal ocular history was unremarkable other than a history of significant trauma to the right side of his head years ago. His best-corrected visual acuity (BCVA) was 20/50+2 OD and 20/20 OS with grade 1 relative afferent pupillary defect OD. His IOPs were in the mid to upper teens with average thickness pachymetry measurements. Gonioscopy showed that the ciliary body was visible in all four quadrants with lightly pigmented trabecular meshwork (TM)

OU, flat iris approach and no obvious angle recession OD.

Given his history of trauma and preliminary test results, we diagnosed him with non-glaucomatous traumatic optic neuropathy OD and early glaucomatous optic neuropathy (GON) OS.

Discussion. Histologically, GON is an ascending or anterograde neuropathy (damage occurring from retina to brain), which is characterized by retinal ganglion cell loss that extends upwards along the axons, resulting in neuroretinal rim loss. Non-glaucomatous optic neuropathy, however, is a descending or retrograde neuropathy (damage occurring from brain to retina) resulting from more than a dozen retinal, optic nerve and neurological disorders that ultimately lead to a loss of capillary perfusion and neuroretinal rim atrophy—with pallor that classically extends beyond any optic nerve cupping (*Table 1*).[6]

Clinically, GON is usually associated with older age, elevated IOPs, thinner pachymetry, visual acuity better than 20/40, greater cup-to-disc ratio with vertical elongation (especially focal neuroretinal rim loss), minimal, if any, neuroretinal rim pallor, greater frequency of parapapillary atrophy (especially beta zone) and optic disc hemorrhages in the inferior temporal and superior temporal sectors that are associated with retinal nerve fiber layer defects.[6,7] GON is also more likely to be associated with a confirmed family history of glaucoma and with visual field defects that tend to respect the horizontal meridian and have a relatively strong correlation with the optic nerve appearance.[6]

Non-glaucomatous optic neuropathy is usually not associated with neuroretinal rim loss—just pallor—and complete rim loss is never present.[6] Furthermore, non-GON does not commonly have significant parapapillary atrophy and, if it does, it rarely progresses. Additionally, and unlike GON, non-GON is usually associated with younger age (younger than 50), visual acuity worse than 20/40 and visual field defects that respect more the vertical meridian and are less likely to

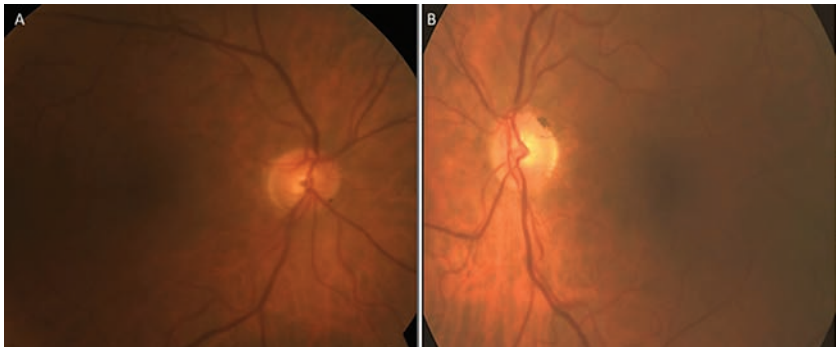

Fig. 2. This optic nerve fundus photo reveals significant focal thinning at 7 o'clock OD (A) and moderate generalized cupping without focal rim thinning OS (B).

Table 1. Etiologies and Examples of Non-glaucomatous Optic Neuropathy

Etiology	Example
Hereditary atrophy	• Recessive or dominant infantile optic atrophy • Behr atrophy • Leber's atrophy
Consecutive atrophy (follows diseases of the choroid or retina)	• Chorioretinitis • Pigmentary retinal dystrophy • Retinitis pigmentosa
Circulatory atrophy	• Central retinal artery occlusion • Branch retinal artery occlusion • Carotid artery occlusion
Metabolic atrophy	• Thyroid • Juvenile diabetes mellitus • Nutritional, toxic amblyopia • Tobacco, methyl alcohol and drug use
Demyelinating atrophy	• Multiple sclerosis • Devic disease
Pressure/traction atrophy	• Glaucoma • Papilledema
Post-inflammatory atrophy	• Optic neuritis • Meningitis • Sinusitis
Traumatic atrophy	• Blunt ocular/head trauma
Radiation atrophy	• Radiation exposure

Gandhi R, Amula GM. Optic atrophy. Medscape. https://emedicine.medscape.com/article/1217760-overview#a5. Accessed April 2019.

correlate with the optic nerve appearance.[6,8-10] Of particular clinical significance, non-GON GCC loss occurs prior to RNFL loss.

Despite these distinctions, some similarities between GON and non-GON can potentially lead to misdiagnosis. Both conditions can sometimes share the following morphological features: decreased retinal arteriole diameter with focal arteriole narrowing, reduced retinal nerve fiber layer (RNFL) visibility, localized RNFL defects and enlargement/deepening of the optic cup with correlating decreased neuroretinal rim thickness.[7-10]

Aside from the ever-important patient history, the most valuable qualitative variables for differentiating GON from non-GON is the neuroretinal rim thickness/perfusion appearance, the strength/appearance of the correlating visual field defects and the presence of associated glaucomatous disc hemorrhages. An accurate diagnosis is more likely to lead to a proper work-up (if indicated) and more appropriate treatment options.

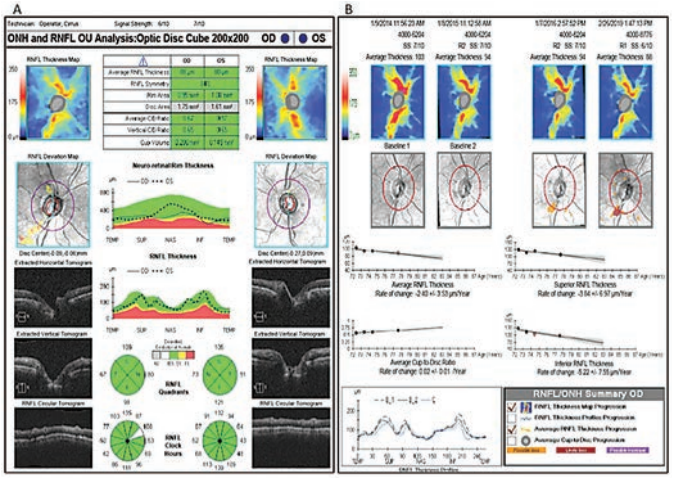

Fig. 3. OCT RNFL analysis shows focal inferotemporal RNFL loss OD (A), while RNFL guided progression analysis shows steep negative slopes of the inferior RNFL thickness OD, more so than the average and superior thicknesses (B).

Case 2: Tensions Running Low

A 77-year-old white male returned to the clinic 32 months after being lost to follow-up for glaucoma suspicion due to asymmetric cupping OS>OD. His ocular history was otherwise unremarkable, and he had no new complaints. His medical history included chronic lymphocytic leukemia, hypertension, hyperlipidemia, benign prostatic hyperplasia and hypothyroidism for which he was taking lisinopril, simvastatin, tamsulosin and levothyroxine. BCVAs were 20/25 OD and 20/20 OS with hyperopic and astigmatic correction. Pupil, extraocular muscle and confrontation field tests were all normal.

His anterior segments were normal with deep chambers. There was moderate nuclear sclerosis OD>OS. IOPs were 20mm Hg OU. On prior exams, his untreated IOP ranged from 17mm Hg to 21mm Hg OD and 16mm Hg to 20mm Hg OS with an average of 19mm Hg OU. His central corneal thicknesses were 553µm OD and 541µm OS. Dilated fundus exam was concerning for vertical cupping and focal inferotemporal neuroretinal rim thinning OS. Cup-to-disc ratios were 0.6x0.4 OD and 0.55x0.55 OS (*Figure 2*).

OCT RNFL analysis demonstrated normal average, quadrant and sector thicknesses (*Figure 3A*). However, on closer inspection, the RNFL TSNIT graph showed significant focal thinning inferotemporal OD. Furthermore, the color-coded RNFL deviation map and the qualitative review of the RNFL circular tomogram both showed thinning.

Research shows thinning of the RNFL at the 7 o'clock sector OD and 5 o'clock sector OS is the most useful discriminant between normal and mild glaucoma among all optic nerve head (ONH) and RNFL parameters included in a single OCT 200x200 optic disc cube.[11] Although this case shows 84% symmetry as normal (95% of normals in the normative database range from 76% to 95% for a 69-year-old, for example), we did note the inferior quadrant and inferotemporal 7 o'clock wedge sector was

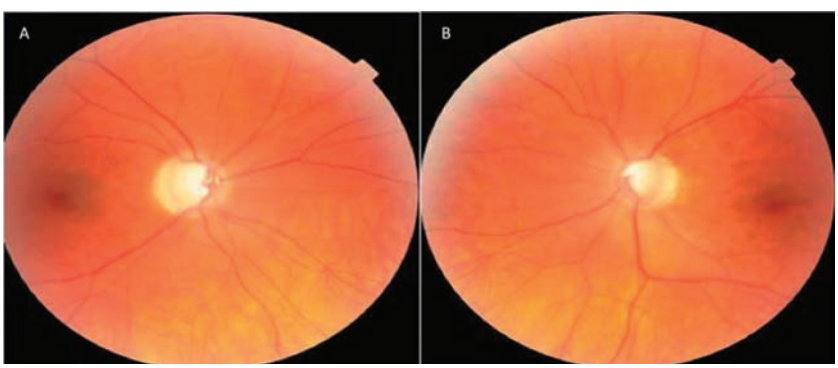

Fig. 4. This patient's optic nerve fundus photo indicates evidence of glaucomatous thinning and cupping, and papapapillary atrophy OD (A) and OS (B).

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23µm thinner OD than OS.[12]

The absence of red or yellow color flag on a single OCT RNFL analysis may lessen the concern for GON, or worse, raise doubt about the ONH assessment on exam. Glaucoma in the context of a normal or "all green" OCT RNFL analysis constitutes a false negative, often referred to as "green disease."[14] In this case, prior scans allowed for guided progression analysis (*Figure 3B*). The report showed new, statistically significant reduction in the average RNFL thickness and continued thinning of the inferior thickness OD. In one study, repeatable loss of ≥4µm was considered significant when comparing scans of adequate signal strength, ≥6µm or 7µm.[11,13]

Given these findings, the patient was diagnosed with low-tension, open-angle glaucoma of indeterminate stage OD, and he remained a suspect OS.

Discussion. While most consider increased IOP a hallmark of glaucoma, large population-based studies show as many as 32% of open-angle glaucoma patients have IOP less than 21mm Hg, considered low-tension glaucoma.[14] OCT structural evaluation of the ONH is a valuable tool to help assess low-tension glaucoma and can be more sensitive than standard automated perimetry in the early disease state.[13,15] Thus, this technology is now a standard component of the evaluation to aid in the early detection of all types of GON.[13,15]

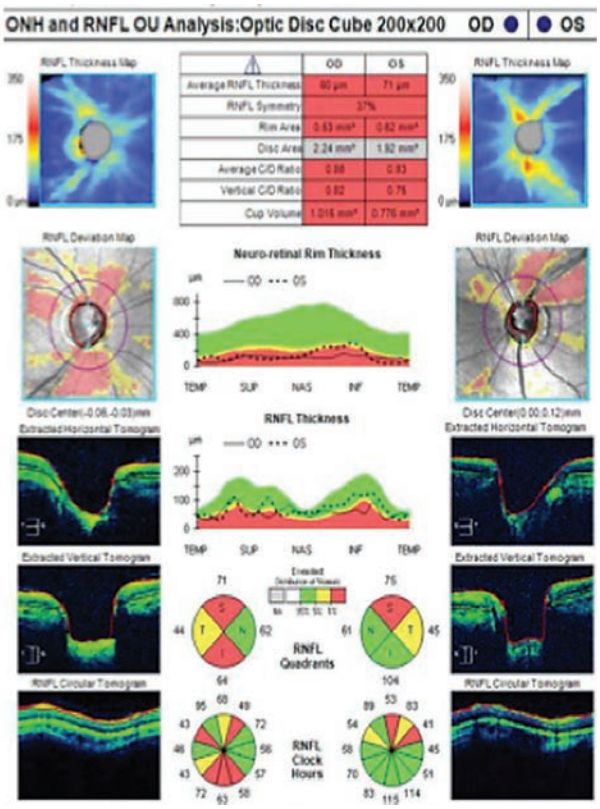

Fig. 5. OCT imaging shows advanced thinning on the thickness and deviation maps, which correlates with the circular RNFL tomogram.

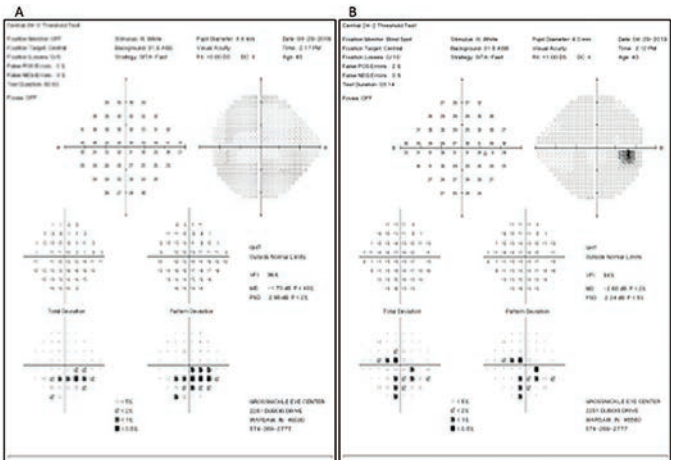

Fig. 6. The patient's SAP (24-2) reveals moderate inferior arcuate to fixation OS (A) and moderate inferior arcuate to fixation and superior central and paracentral defects OD (B).

Although the OCT normative database is a useful benchmark, it does have limitations. Clinicians should carefully interpret scans beyond the red, yellow and green designations. Clinicians can recognize green disease by scrutinizing the TSNIT graphs, qualitatively reviewing the peripapillary RNFL B-scans (while being mindful for segmentation errors) and using serial scans with progression analysis. In addition, inferotemporal RNFL thinning on OCT imaging is a key element in the detection of early or pre-perimetric glaucoma. Despite OCT's firm foothold in the standard of care for diagnosing and monitoring GON, careful scrutiny of the neuroretinal rim on funduscopy is vital.

Case 3: Pigments on the Loose

A patient in his late 30s was referred for a glaucoma evaluation with treatment-naïve elevated IOPs in the mid to low 30s and glaucomatous optic nerve appearance OU (*Figure 4*). His review of systems and ocular history was unremarkable other than a history of moderate- to high myopia (-7.50D OD, -8.50D OS) with a history of LASIK OU more than a decade ago. His BCVA was 20/20 OD and 20/20 OS with no relative afferent pupillary defect OU. Gonioscopy showed that the ciliary body was visible in all four quadrants

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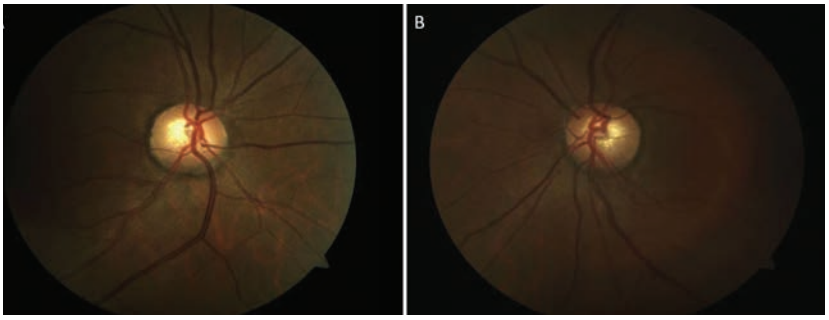

Fig. 7. The patient's optic nerve fundus photo shows a healthy rim and moderate cup OD (A) and OS (B).

with 3 to 4+ TM pigmentation, and slightly concave iris approach OU. The exam also showed multiple midperipheral, distinct transillumination defects and associated bilateral endothelial Krukenberg spindles OU. His preoperative pachymetry measurements were 574µm OD and 595µm OS.

Given the patient's age, advanced neuroretinal rim thinning, OCT thinning and correlating visual field defects, he was diagnosed with pigmentary dispersion glaucoma (*Figures 5 and 6*).

Discussion. More than a dozen secondary forms of glaucoma exist, and research shows they account for about 22% of all cases of confirmed glaucoma or glaucoma suspect patients; 57% of these patients have IOPs above 30mm Hg (*Table 2*).[16,17] These patients often have a positive history and ocular findings of trauma, surgery, neovascularization, inflammation or other abnormal findings that might explain the increased IOP.[17] They require active monitoring and aggressive treatment due to their commonly advanced stage and younger age at presentation.[17]

Table 2. Secondary Glaucomas[17]

Angle	Secondary Classification
Open	• Pigmentary glaucoma • Pseudoexfoliation glaucoma • Increased episcleral venous pressure (e.g., thyroid, Sturge-Weber) • Steroid-induced • Lens induction • Intraocular hemorrhage • Posner-Schlossman syndrome • Intraocular tumor debris • Blunt ocular trauma • Iatrogenic (laser/surgery or retinal detachment complications)
Closed	• Intraocular tumor • Chronic uveitis • Neovascular glaucoma • Iridocorneal endothelial syndrome • Aniridia • Aqueous misdirection dyndrome • Uveal effusion • Post-argon laser trabeculoplasty • Phakic intraocular lens

This case focuses on one of the more common forms, pigmentary glaucoma, which is a potential endpoint for those with pigmentary dispersion syndrome. The syndrome is a clinical triad first classified as mid-peripheral transillumination iris defects, pigment deposits on the corneal endothelium (Krukenberg spindle) and heavy TM pigmentation.[18,19] Reseaerch shows that the latter of these findings is what potentially leads to elevated IOPs and, if not properly treated, irreversible glaucomatous damage.[20] Conversion to glaucoma is possible in up to 50% of those with pigmentary dispersion syndrome.[21] Furthermore, one study reported the risk for developing pigmentary glaucoma from pigment dispersion syndrome was 10% at five years and 15% at 15 years. Those with an IOP greater than 21mm Hg at the initial examination also had an increased risk of conversion.[22]

Younger, white, myopic males are most at risk for pigmentary glaucoma, and they are usually diagnosed in their 3rd or 4th decades of life.[21] Fortunately, with a timely diagnosis, these patients usually respond well to topical treatment and selective laser trabeculoplasty.

One of the most important tools for differentiating between primary and secondary glaucoma is gonioscopy. This technique, along with a thorough patient history, leads to a more accurate diagnosis, a proper work-up (if indicated) and appropriate treatment options.

Case 4: Debris Holding Up the Works

A 78-year-old Caucasian male reported to our clinic for a routine eye exam. He reported an ocular history of "atypical" cataracts, but did not elaborate. He was not taking any ocular medications, and his BCVA was 20/20 OD and OS. Pupils, extraocular muscles and confrontation field tests were all normal. His IOPs measured 28mm Hg OD and 30mm Hg OS.

Notable anterior segment findings included a white, fluffy material around the pupillary margin OU, no transillumation iris defects

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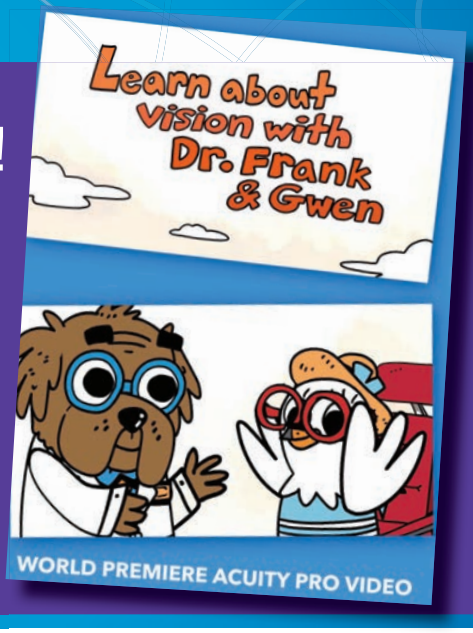

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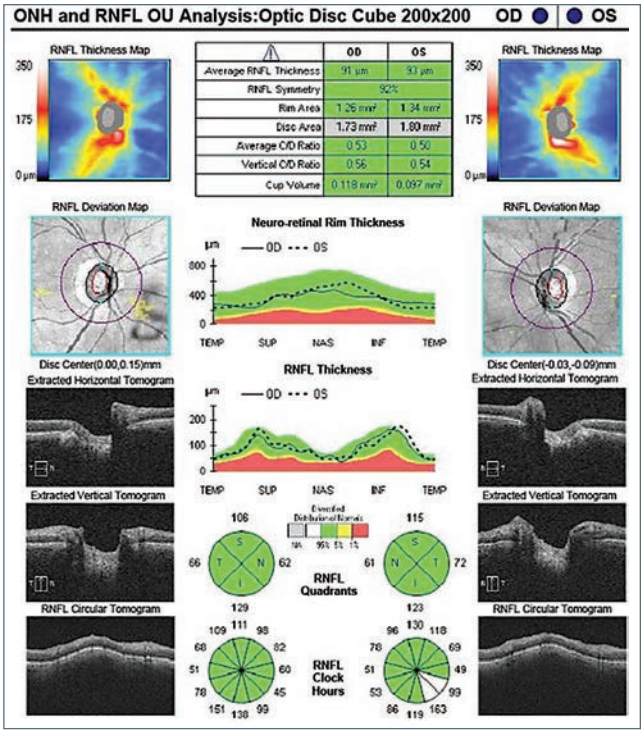

Fig. 8. This retinal nerve fiber layer OCT scan shows a normal TSNIT graph, normal quadrant and sectoral tomogram, and robust thickness maps OU.

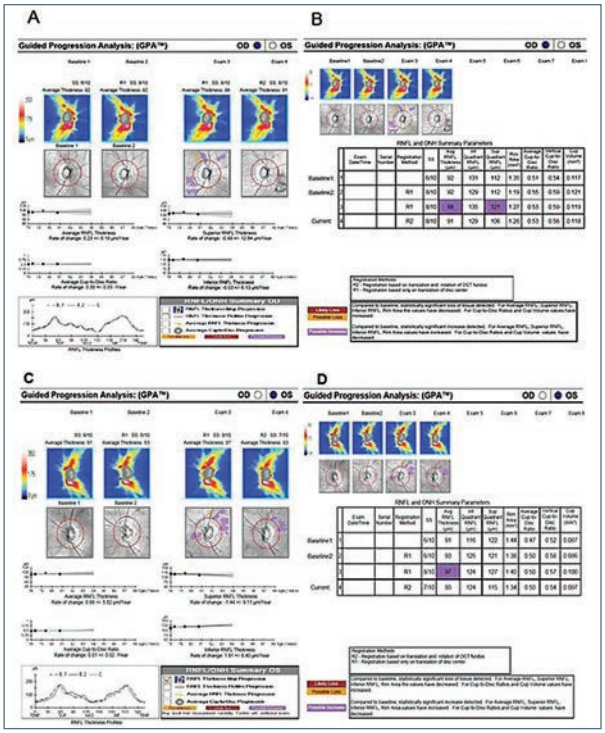

Fig. 9. RNFL guided progression analysis OD (A) and OS (C) shows stable RNFL thickness slopes. Furthermore, the RNFL and ONH parameters remain stable OD (B) and OS (D).

OU, and no corneal endothelial pigment OU. Gonioscopy showed an open angle 360°, increased TM pigmentation and Sampaolesi's line inferiorly. This increased pigmentation in the TM resembled a "brown sugar" appearance. All other anterior segment findings were within normal limits.

His dilated fundus exam showed a "ground glass" appearance of the anterior lens capsule, and retroillumination of the lens showed a "bull's-eye" appearance. There was a clear vitreous, a macula that was flat with a mild epiretinal membrane, an arterial-venous ratio of 2/3 and a peripheral retina that was flat and intact with no pathology noted OU.

The optic nerves had a cup-to-disc ratio of 0.55x0.55 OD and OS, and were of average size and normal shape (*Figure 7*). There was no Drance hemorrhage, pallor, loss of perfusion or RNFL wedge defect OU. An RNFL OCT was within normal limits OD and OS (*Figure 8*). GCC was deferred due to epiretinal membrane artifacts. Pachymetry readings measured by OCT were 550µm OD and 548µm OS.

Based on these findings we diagnosed the patient with ocular hypertension and pseudoexfoliation (PXF) syndrome in both eyes. However, we did not initiate any topical hypotensive treatment at this visit, as we wanted to acquire additional IOP measurements first. Over a series of four office visits (a few months), the patient had an untreated IOP range of 26mm Hg to 32mm Hg OD and 28mm Hg to 33mm Hg OS. We then initiated topical hypotensive treatment with latanoprost QHS OU due to the aggressive nature of PXF and the increased risk PXF possess when IOP is above threshold. He returned six weeks later and his IOP measured 16mm Hg OU, and his SAP 24-2 visual fields were within normal limits (*Figure 9*).

We followed this patient with serial visual fields and OCT exams. The RNFL OCT and visual fields remained stable over several years as shown in the GPA analyses (*Figure 10*). He required no further intervention.

Discussion. PXF is a systemic disorder characterized by progressive accumulation and granular deposition of abnormal extracellular pseudoexfoliative material in ocular tissues and many organs throughout the body. Deposits are composed of amyloid, laminin, collagen, elastic fibers and basement membrane.[23]

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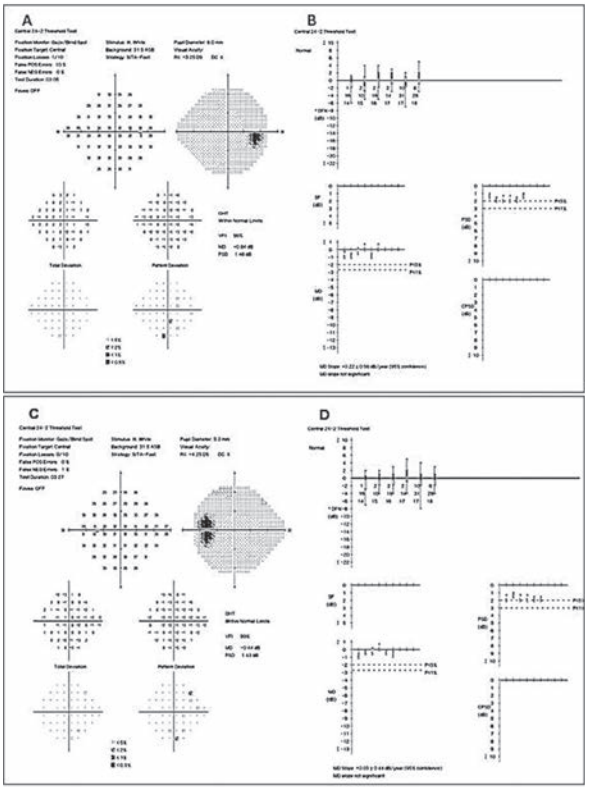

Fig. 10. The patient's most current SAP (24-2) visual fields show OD (A) and OS (C) being within normal limits and without glaucomatous defects. GPA slope OD (B) and OS (D) are stable to baseline, and the MD slope is "not significant."

PXF is predominantly associated with those of northern European and Scandinavian descent, but can be found in all populations and races.[24] Patients with PXF and IOPs above the normal threshold have double to triple the risk of developing glaucoma compared with those without PXF and IOP above the normal threshold.[25,26]

IOP elevation in PXF is caused by increased outflow resistance in the TM due to the blockage caused by pseudoexfoliative material. Exfoliation debris inherently occur and accumulate in the TM, which subsequently alters and damages the juxtacanalicular tissue beneath the inner wall of Schlemm's canal, causing increased IOP.[27,28] Pseudoexfoliation glaucoma (PXG) has a worse prognosis than primary open-angle glaucoma due to higher IOP levels, greater diurnal fluctuations and IOP spikes.[27,29] Topical hypotensive therapies are generally effective in lowering IOP in PXG cases initially, but due to the condition's refractory and recalcitrant nature, surgical options are usually needed.[27]

A patient's IOP is only a small part of the overall ocular picture. High IOP doesn't necessarily indicate glaucoma, and low IOP doesn't rule it out, either. Only with a thorough medical history and clinical examination, augmented by diagnostic tools such as fundus photography, OCT and visual fields, can clinicians see the whole picture and provide an accurate diagnosis. ■

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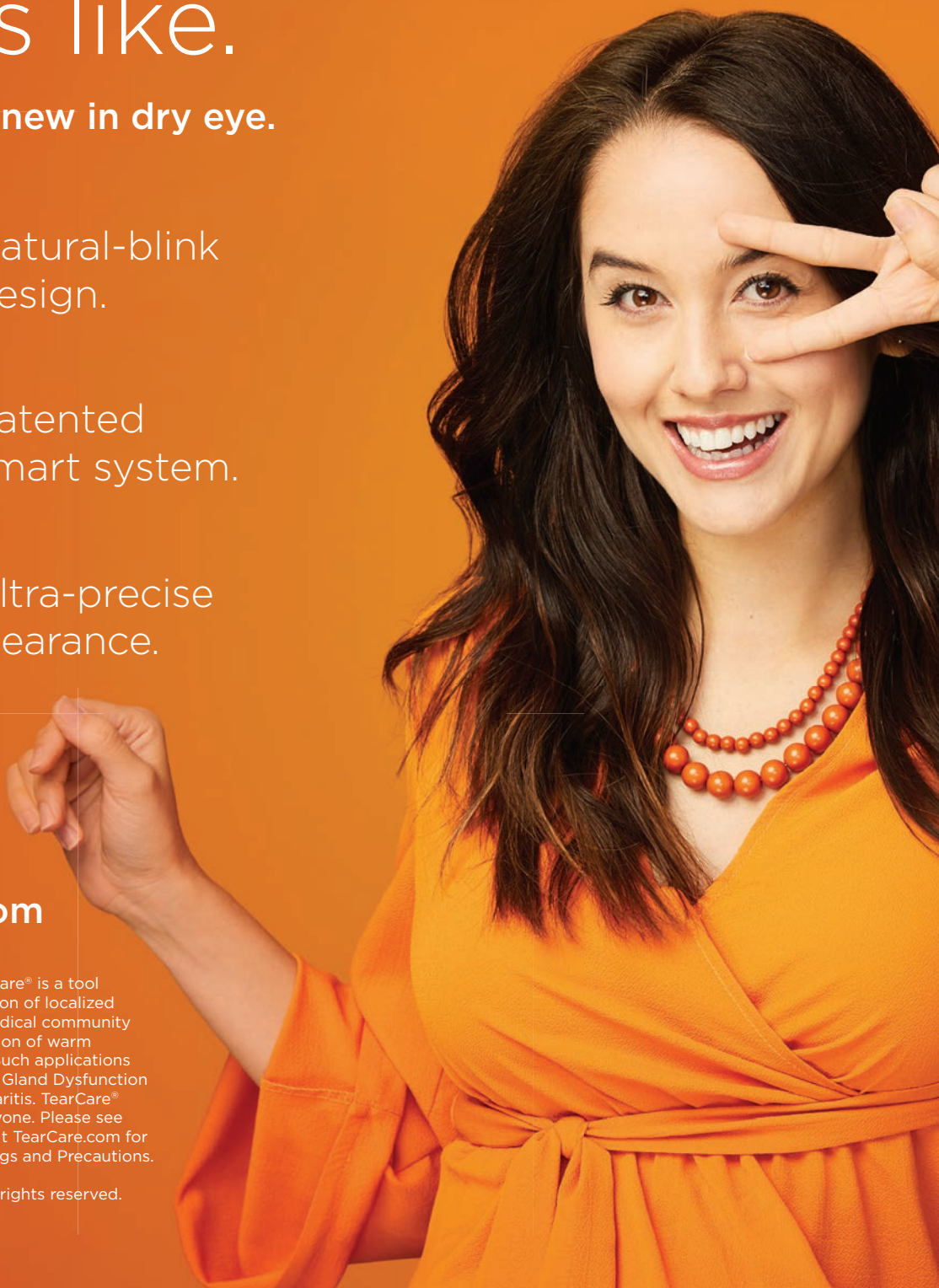

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PEER-REVIEWED PRA ARTICLE

An OD's Guide to Visual Processing Disorders

When you suspect your patient's sight is affected by a neurological issue, be sure to understand the differential diagnosis. By Nicholas Karbach, OD, and Andrew S. Gurwood, OD

Neurological insults can affect visual processing without creating loss of acuity. In other words, patients test 20/20, but something else is keeping them from seeing properly. These patients report to the eye clinic with puzzling symptoms. Their insults develop following stroke (ischemic or hemorrhagic), neurodegenerative diseases, trauma, infection, carbon monoxide exposure, toxic exposure to other chemicals, demyelinating disease, intracranial mass or psychiatric illness.

The patient may be unable to articulate the problems they are experiencing or, worse yet, they may be unaware of any issue. Newly detected abnormalities include visual field loss (which can sometimes manifest as neglect of one side), or difficulty navigating, following directions, identifying location where one is, naming objects or people, telling time, recognizing faces or one's self in the mirror, reading, writing, seeing patterns or doing simple arithmetic.

All of these issues can be produced by neurologic disease. Yet, optometrists may misunderstand the complaint and misdiagnose the underlying issue.

Sharing with patients this vegetable man image is a method by which clinicians can determine if patients have a particular issue with their visual processing. Patients with simultagnosia, for example, can describe the fruits and vegetables but won't recognize that they form the shape of a man.

Here, we review how strokes and other neurological conditions are likely to appear in your exam chair and how you can properly comanage these patients' systemic health while taking a lead role in safeguarding their visual health.

The Clock-Drawing Test

Take this case, for example. A 65-year-old female was referred to our retinal service by an outside practitioner who could not remedy a reading symptom she described as "distortion of the page." The practitioner documented no ocular etiologies. They included records of testing that demonstrated 20/25 visual acuity (VA) at distance and near with a +0.50/+2.50 sphere refraction OU.

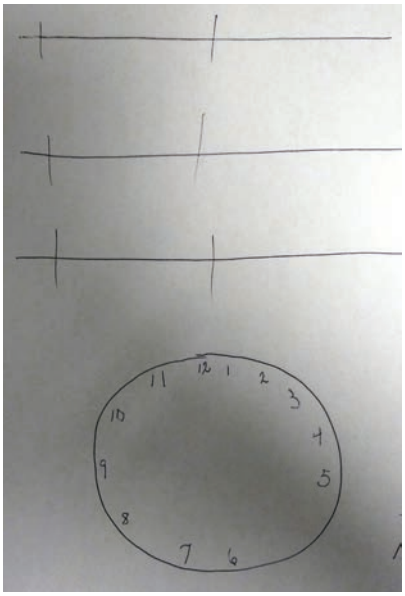
The data of the exam revealed no change in distance or near refraction, no appreciable change to her crystalline lenses, normal topography (no evidence of keratoconus), normal Amsler grid, no afferent pupillary defect (APD), no color or brightness abnormality, normal fundus appearance, normal central screening visual field and normal optical coherence tomography (OCT) (5-line raster, macula).

The retina specialist completed a dilated exam that confirmed these findings. A call was placed to her general practitioner, who explained that she had a history of right-sided parietal lobe cerebrovascular accident (CVA) from three years earlier, and her symptoms began after that. With this information, we decided to perform a "clock-drawing test," in which the patient is presented with a blank circle and asked to fill in the clock hours. The filled-in clock face demonstrated initial asymmetric distribution of the clock hours skewed to the right with subsequent correction. This is indicative of mild left hemi-spatial neglect. Visual field testing was not conclusive for hemianopia but demonstrated global loss of sensitivity. It was then we realized that the medical team and ophthalmic teams were independently managing her symptoms without ever associating them to the location of the CVA.

Her condition was re-explained to her and she was referred to low vision services, where she received vision rehabilitation care. She was provided with reading aids including a typoscope, electronic magnifiers and an illuminated hand-held magnifier to help with her work (jewelry crafting).

For Each Behavior, An Anatomy

Stroke symptoms—even visual symptoms—can be missed by any doctor, and that includes visual symptoms. When a primary vascular pathology produces changes in personality or mentation in the absence of a motor deficit (cranial nerve III, IV, VI, VII palsy, hemiparesis or hemiplegia) the possibility of illness or disease may be ignored or rationalized as "a bad mood or day" by the patient, friends, family and co-workers. This leads to a dissociation of the signs from symptoms, leading to a lack of testing.



Tasks like this clock-drawing test can help determine the extent to which an injury has impacted the patient's brain.

Every behavior has an anatomy. Architecturally, the brain is compartmentalized. If the area handling a task is affected, the system becomes dysfunctional.

The retina is designed to convert the electromagnetic energy of photons (the particles of light) into electric impulses and distribute it into the brain via the visual pathway.

The visual pathway carries electric impulses to three places: the Edinger-Westphal nucleus in the midbrain (pupillary constriction pathway—cholinergic), the occipital lobe (visual development center) and the pulvinar/sympathetic pathway (pupillary dilation pathway—adrenergic).[1,2]

Defects in any pathway can produce a problem specific to that pathway; a problem in the pupillary constriction pathway will produce constrictive abnormalities (a pupil that fails to constrict such as an APD, Adie's tonic pupil and Argyll Robertson pupils). Problems in the three neuron arc of the pupillary dilation pathway will produce abnormalities of dilation (e.g., Horner's syndrome), and issues with the traditional pathway carrying impulses to the occipital lobe will produce visual field defects and variable visual loss.[1]

Once the occipital lobe has created an image, the information enters the ventral stream. This is where understanding and context begins. When associative centers of the brain are either damaged or denied their neural input, seeing continues in the absence of understanding. This is called an agnosia.

The term "visual agnosia" refers to difficulty naming or identifying visual targets. The pathology occurs in the context of an intact visual pathway, intact memory and intact ability to think. Visual agnosia can be further divided into apperceptive agnosia (AA), or the impairment of processing patterns, and associative agnosia (AsA), or the impairment of visual memories and their meanings.[3]

Apperceptive Agnosia

This visual impairment results in a patient's inability to identify patterns; as such, they have difficulty identifying objects because they cannot see the way components form a single image. When shown a simple object, such as a mug or cup, these patients cannot draw it. An AA patient does not suffer from visual scotoma or lost acuity.

AA is the result of pathology that interrupts the ventral stream (connections between the occipital lobe and temporal lobes) in the occipital and temporal association areas in the distribution of the middle cerebral artery (the most common location of CVA.[1,4]

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AA patients are able to name the attributes of an object but are not able to identify the whole object. They maintain their ability to describe objects in detail and then recognize the object (name and function) when the object is placed in their hand.[3,5] As an example, an AA patient would not be able to draw or identify a set of car keys. They would be able to look at the set of car keys and describe what it is made of or describe the jagged edges of the "teeth" of the keys. However, when the set of keys were placed into their hand they are able to both name the object (a set of car keys) and describe what they do. These individuals also fail the Gollin form test.[3,6] When presented with Gollin forms, the individual sees a conglomeration of shapes but not the pattern they create. The AA patient looking at the toothpick-like illustration of the word "THIS" would report seeing the number "7415."[7]

Associative Agnosia

This is often "category-specific" to the portion of the brain that has been affected. Here, the damaged area of the brain is the occipital-temporal region.[1,3]

Damage here causes patients to lose the ability to identify category-specific visual information. For example, the patient may be unable to identify a dog by seeing it but can recognize a dog by its bark.[8] Unlike AA patients, they can draw a reasonable likeness of these objects when they are shown them but cannot explain what they are. However, when asked what a "lion" or "car" or "chair" is, they can articulate the correct answer.

Global agnosia occurs in the worst cases. Here, diffuse hypoxic injuries such as carbon monoxide poisoning create widespread damage over large cerebral expanses involving visual memories and their associations.[3,5]

A suspect for appreceptive agnosia can be offered this Gollin form test. Those with the condition see the shapes, but not the pattern they create. For instance, above, they would report seeing the number 7415, but not the word "THIS."[3]

Prosopagnosia

This is an inability to recognize faces and is associated with focal temporal-occipital junction lesions.[3,5,9,10]

In the apperceptive form of prosopagnosia (PA), patients experience difficulty discriminating specific features of a face. In the associative form, the patient can identify, compare and match features of a face but can't match it to the person they know.[3] An extreme example is "mirror agnosia," a form of prosopagnosia where the subject is unable to recognize themselves in the mirror.[11,12]

Clinical tests used to uncover PA include the "famous faces test" and the "Benton facial recognition test."[3,9]

It is likely that many PA patients have broader object agnosias not limited to faces, but the inability to recognize a face stands out due to social ramifications.[9]

Interestingly, people can be born with prosopagnosia (2.5% of the general population).[3] These people appear to have difficulty remembering if they previously met someone and also appear to have trouble with people's names.[3]

Simultagnosia

Lesions in the dorsal stream and the inability to process multiple details of an object at the same time—a sort of narrowed window of attention—are indicative of simultagnosia (SA). Unlike those with other agnosias, these patients are often symptomatic to others; they appear as inattentive or aloof.[3]

The "cookie theft picture" (shown on p. 80) is a classic test for SA.[1] Here, the SA patient verbalizes that the dishes are being cleaned but does not notice the water overflowing from the sink or the child stealing cookies. These patients are often labeled as clumsy because they have difficulty navigating around obstacles in a room. They also fail the "vegetable man test" (shown on p. 75), where they're asked to view the picture of a man whose image is constructed of fruits and vegetables. The SA patient will describe individual fruits and vegetables but will fail to see the person.[13] Other tests include the "alphabet test" (see p. 82), where a letter of the alphabet is created by other, smaller letters.[14]

SA is common in occipital lobe stroke, although it can also result from neurodegenerative diseases such as posterior cortical atrophy.[3] It may also occur in the context of Bálint syndrome (acquired triad of simultagnosia, optic ataxia with spatial distortion and ocular motor apraxia) secondary to parietal lobe stroke.[15] SA is a diagnosis that can be predicted based upon neuroimaging; however, cognitive dysfunction, hemineglect and visual field defects should be ruled out before administering testing.

Topographic Agnosia

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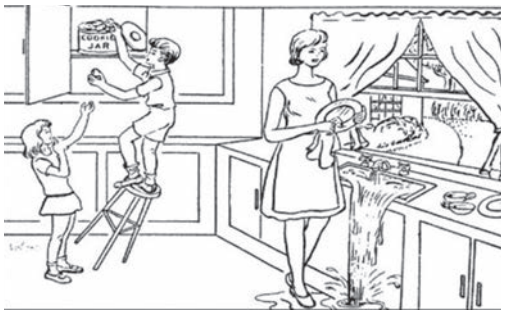

The "cookie theft" test for SA uses this picture. Patients with the condition will verbalizes that the dishes are being cleaned but will not notice the water overflowing from the sink or the falling child stealing cookies.

These patients can remember and verbally repeat directions and can explain how to get from one place to the other but are unable to execute the task.[3] Difficulty with navigation is reported in up to 30% of post-stroke patients and has a strong correlation with subjective quality of life.[16] Again, this occurs in the context of intact cognition and memory.[3] There are two possible types. The first, "landmark agnosia," occurs when the patient has difficulty recognizing significant landmarks that they encounter in their environment.[3] It can affect both novel and familiar landmarks.[17] Researchers believe this occurs from damage to the lingual and fusiform gyri.[17] The second type is the inability to construct a mental spatial map and arises from damage to the hippocampus or retrosplenial cortex.[3]

Reading, Writing, Arithmetic

The inability to read, with and without agraphia (the inability to write) is an acquired "form-agnosia" preventing reading in previously literate patients and is called alexia.[3,18-21] The issue is secondary to damage in the area of the brain that completes cortical processing of language.[20] The lesion is in the left occipital lobe and the splenium of the corpus callosum. These patients experience an absence of perceptual or language deficits.

Pure agraphia is rare. It is more often accompanied by alexia, or aphasia (inability to articulate). Individuals with apraxic agraphia can spell normally when asked to complete the task orally, in the setting of otherwise intact visual/language processing.[21] The cortical area responsible for creating apraxic agraphia is unclear as lesions in the parietal, frontal or thalamic areas can all cause agraphia.[21]

Acalculia is the acquired impairment of arithmetic skills despite normal reasoning and language skills.[22,23] These patients understand what a number is and can see numbers on a page but are unable to perform the reasoning skills used in basic arithmetic. It is associated with left posterior parietal lobe damage.[22]

Visual Orientation

Allochiria is when a patient responds to stimuli presented to one side of their body as if the stimuli had been presented on the opposite side.[24,25] Allochiria typically occurs following left hemispheric stroke.[26] The condition results in mental reversal of hemispheric orientation. In the clock-drawing test, a patient with allochiria can correctly copy the 9 o'clock number onto a blank clock face but when asked to draw a clock face from memory, they will transpose it to the wrong side (the 3 will wind up in the 9 position).

This is in contrast to a patient with hemispatial neglect, who will draw most clock hours in the preserved hemifield.

Peripheral visual awareness plays a role in seeing and spatial awareness. It permits focus of attention and aids in directing accurate motor commands toward objects. Loss of the visual field creates a lack of awareness and ability to interact with objects in space.[27] Negative scotomata (neural field loss) are missing areas in the visual field filled in by the brain with no perception of missing vision. Patients with this issue often complain of vertigo (dizziness), balance difficulty and reading difficulty.[27]

Hemifield visual field loss is a common sequela of stroke, space-occupying lesion or traumatic brain injury.[28] Estimates of its prevalence in stroke range from 10% to 57% of patients.[28,29] Unfortunately, some patients either do not realize their field loss exists or cannot articulate the experience secondary to cognitive decline or inability to communicate.[30,31] Awareness often arises when patients fail to perform tasks that require visual feedback, such as navigating an unfamiliar space, driving or reading.[28,30]

Automated visual field testing can help discover hemifield deficits, but are inconsistent.[28] Confrontation visual fields are less sensitive and can miss up to 50% of severe defects.[28,30] Clinicians must be good observers. Tell-tale exam room behaviors—such as scuffs and scrapes on one side of the patient's clothing or shoes, exaggerated head turning toward the affected side or persistently reading one side of the line of letters on the visual acuity chart or reading material—may all serve as clues to missing visual fields.

Visual neglect is often associated with hemifield visual field loss

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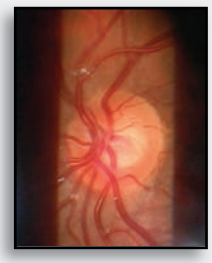

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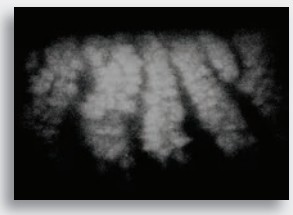

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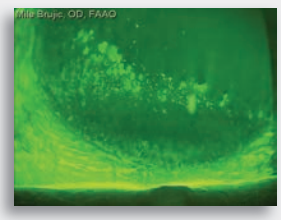

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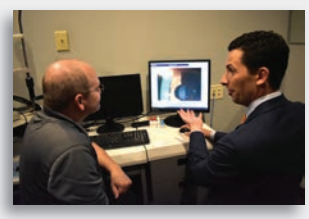

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and occurs when the patient lacks awareness of the contralesional visual space. It most commonly occurs on the left side, as the left cortical hemisphere monitors only the right hemispace and the right cortical hemisphere monitors both sides.[5] A variant form called allocentric object-based neglect refers to the symptom of ignoring the contralesional side of objects, regardless of the object's egocentric position.[25]

The OD's Role

Management of agnosias and other suspected neurological defects depends on how recently it started. If symptoms present acutely (within 48 hours) the patient must be rapidly transported to a stroke center for evaluation and intervention with thrombolytic agents (most effective within 4.5 hours of ischemic onset).[32]

Symptoms that have been present for 48 hours to two weeks should still be managed with urgency; these patients require evaluation and neuroimaging but this can be best accomplished through coordination with a primary care physician (PCP).[33] A phone call should be placed to the PCP immediately, ideally for same-day evaluation. The PCP may elect to have the patient evaluated in the emergency department or as an outpatient.

Mitigation of diabetic and hypertensive risk factors as well as initiation of aspirin or statin drugs will likely be initiated by the PCP within 24 hours.[33] More often, symptoms are vague and chronic when discovered and require investigation to uncover the nature of the problem along with its underlying cause. After stroke, the most common causes of adult-onset neurological disorders are Alzheimer's disease and demyelinating disease.[34]

The SA patient will look at this image and see the smaller letters Es, but not the larger A they make up.

The role of the optometrist is to refer to neurology, communicating that all ocular findings are normal (unless visual fields are affected) and that the symptoms require further investigation. The OD also plays a valuable role in providing rehabilitative and low vision services along with education on the nature of their visual perception disorder. ■

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Dr. Gurwood is a professor at Pennsylvania College of Optometry at Salus University.

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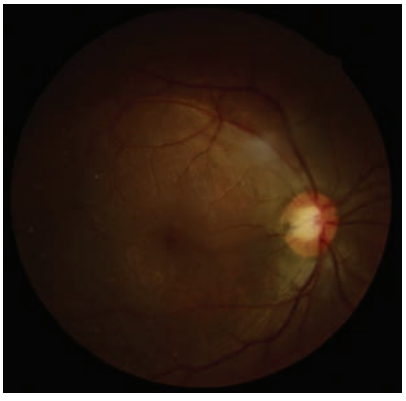
Larry Alexander Resident Case Report Contest Winner

When and How To Treat Optic Disc Pit Maculopathy

This rare presentation has both medical and surgical treatment options. Be prepared with comprehensive knowledge of both. **By Christian Corzo, OD**

In April 2016, optometry lost a giant when the author of the seminal work Primary Care of the Posterior Segment, *Larry Alexander, OD, died. In addition to being an optometric physician, author and educator at the University of Alabama Birmingham School of Optometry, Dr. Alexander was a past president of the Optometric Retina Society (ORS). That group has chosen to honor his legacy by accepting case reports from optometric residents across the country relating to vitreoretinal disease. As selected by the board of the ORS, this case won the third annual Larry Alexander Resident Case Report Contest.*

A patient who presents with congenital optic disc pit (CODP) is at risk for developing optic disc pit maculopathy (ODPM). Many published studies have explored surgical techniques for treating this condition. Despite our current understanding of this complication, no consensus regarding the optimal treatment for ODPM exists. In addition, the literature shows no clear consensus on the mechanism of pathogenesis or the origin of the subretinal fluid, making it more difficult to determine the optimal surgical technique for the treatment of such a complex condition.



The patient presented with a temporal ODP and an elevated macula with surrounding radiating stellate pattern.

Here, we review the presentation, diagnosis and surgical options for a patient who presented with this condition.

Background

CODP presents as small, round, hypopigmented excavated defects usually within the temporal (or inferior temporal) portion of the optic disc.[1] They are grayish white and typically unilateral (although 10% to 15% of cases are bilaterally).[1,2] CODP defects vary in both depth and size. Their average diameter is 500µm and can be up to 0.16mm deep. CODP occurs in one out of 11,000 patients.[1] No sex nor genetic predilections are associated with this condition.[3,4] Due to their congenital nature, they are often diagnosed on the patient's initial eye exam.[3] Though visual acuity (VA) is rarely affected, field loss is possible. If field loss is present, the defect corresponds with a paracentral arcuate scotoma or an enlarged blind spot or both.[5,6]

Any patient who presents with a CODP is at risk for developing ODPM. This complication presents as a serous-neurosensory detachment in conjunction with the presence of an optic disc pit. Occasionally, serous maculopathy presents together with a retinoschisis. ODPM can occur in 52% of patients with CODPs. Patients with temporally located pits are at the greatest risk for developing serous maculopathy.[5]

Typically, maculopathy occurs in patients between 30 and 40 years

old.[6-8] In the presence of this complication, VA is usually reduced to 20/70 or worse.[7]

Though spontaneous resolution occurs in 25% of patients, the majority of cases have a poor visual prognosis, with gradually decreasing vision, leading to a final VA of 20/200 or worse.[7,9] The cause of ODPM remains unclear.[7]

Surgical intervention has proven to be extremely valuable in managing these patients; yet, due to the rarity of such a unique condition, few cases exist published in academic literature.

Despite our current understanding of this condition, researchers have reached no consensus regarding the optimal treatment.[6]

Not Rare in Your Chair

A 29-year-old African-American female presented with a chief complaint of constant, painless, longstanding unilateral vision loss. She reported that her vision gradually began to decrease eight months prior to her exam and has remained poor since that time. She denied any ocular trauma, but her history was remarkable for compound myopic astigmatism in both eyes, and her medical history was remarkable for asthma.

Her best-corrected VA was 20/400 OD and 20/20 OS. When retinoscopy findings were compared with the patient's current spectacle prescription, a significant hyperopic shift was noted in the right eye along with a dim light reflex, which was greater in the right than the left eye.

Dilated fundus evaluation and posterior segment photos of the right eye show a deeply pigmented circular hole in the temporal neuroretinal rim of the optic nerve. The macula and the temporal juxtapapillary retina appeared elevated, and a radiating stellate pattern surrounded the macula.

Optical coherence tomography (OCT) of the right eye showed an accumulation of serous fluid separating the sensory retina and the retinal pigment epithelium (RPE). Cystic pockets were noted in the outer retinal layers (nasal to the fovea) on OCT.

Differentials

The differential diagnosis for a serous retinal detachment is dependent upon the underlying etiology. The four etiologies are neoplastic, inflammatory, vascular and congenital. Neoplastic causes include choroidal melanoma, choroidal hemangioma and choroidal metastatic lesions, while inflammatory causes include Vogt-Koyanagi-Harada disease, posterior scleritis and sympathetic ophthalmia. Common vascular etiologies include choroidal neovascularization, Coats' disease, malignant hypertension and familial exudative vitreoretinopathy. Congenital lesions include CODP, morning glory disc anomalies and choroidal coloboma.[10]

Based on our patient's clinical findings (the presence of an optic pit in conjunction with a neurosensory serous retinal detachment and the absence of any underlying neoplastic, inflammatory or vascular etiologies), the patient was diagnosed with ODPM.

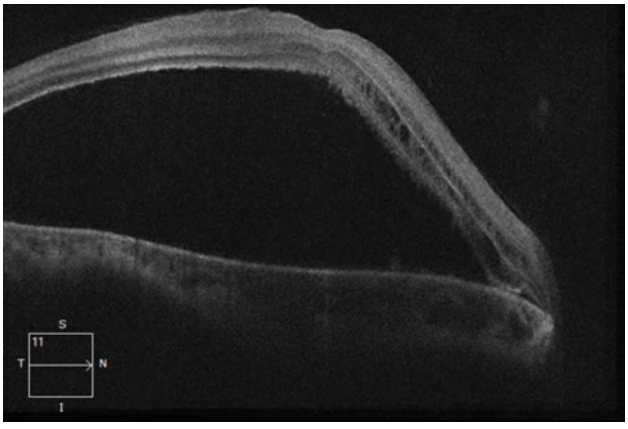
Treatment Options

One of the initial treatments for ODPM was the use of oral corticosteroids and acetazolamide. This treatment proved ineffective in a majority of cases; though the retinal fluid would be initially absorbed, the serous fluid would later reappear after the discontinuation of treatment. As a result, the use of oral corticosteroids and acetazolamide is no longer a valid treatment options.[11]

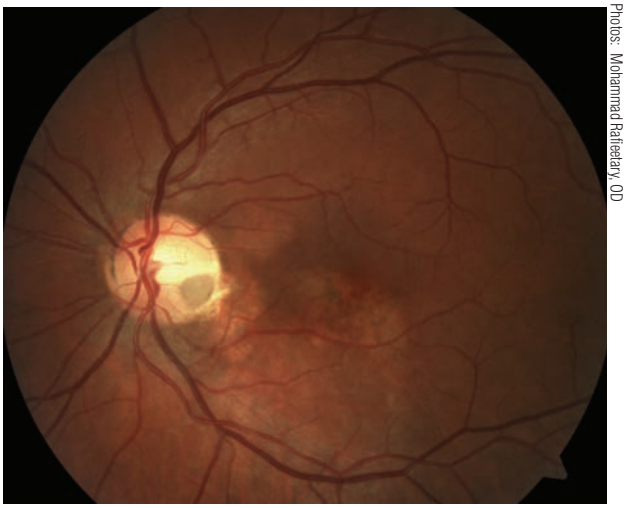
Another early treatment modality was the use of argon laser photocoagulation. Researchers suggest that laser scars, created temporally to the disc margin, would create a chorioretinal barrier that would prevent the passage of fluid from the optic pit to the underlying subretinal space.[11]

Though fluid reabsorption and retinal reattachment was reported in some patients, this treatment had a low initial success rate with visual field defects arising as a consequence.[11,12] This was especially true in cases were serous maculopathy was present in conjunction with a retinoschisis.[11,12] Though laser photocoagulation provides a strong adhesion between the photoreceptors and the RPE, this procedure alone fails to prevent intraretinal fluid migration.[11]

Intravitreal gas injection



This 21-line macular OCT shows, in our patient's right eye, a non-rhegmatogenous serous retinal detachment.



Photos: Mohammad Rafieetary, OD

This fundus image of another patient helps reveal that subretinal and intraretinal fluids are at the root of the reported central vision loss.

has also been proposed as a treatment option, although this is more successful when combined with laser photocoagulation.[13,14]

Another option for these patients, macular buckling surgery, involves positioning a scleral sponge at the posterior aspect of the globe behind the macula along the 6 o'clock to 12 o'clock meridian. An intraoperative B-scan is required for exact placement of the macular buckle. Though this procedure is highly successful, its complexity has prevented it from gaining much popularity.[15]

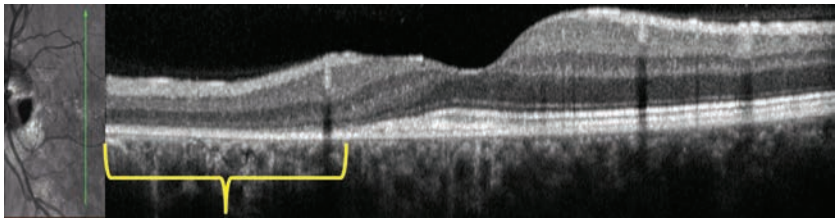
The current literature favors pars plana vitrectomy (PPV) as the treatment of choice for ODPM. PPV is most commonly performed in combination with different surgical procedures such as barricade photocoagulation, intravitreal gas injection and internal limiting membrane (ILM) peeling.[16] One study found the use of combination PPV, laser photocoagulation and intravitreal gas injection improvemed VA in 90% of patients with a complete resolution of maculopathy in 70%.[17]

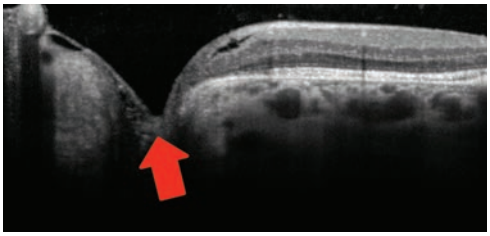
PPV is generally performed within an hour or two of laser photocoagulation. Removing the vitreous gel during this time frame allows the gas bubble to compress the retinal layers juxtapapillary to the optic disc. This allows for formation of intraretinal scars during the seven-to-10 day postoperative face-down period, seizing the migration of intraretinal fluid.[11,16]

Researchers hypothesize that vitreous traction could be responsible for ODPM development. Much has been published on the resolution of ODPM following posterior vitreous detachment (PVD) induction with combined surgery. In one series, vitreous adhesion to the macular surface was noted during surgical intervention.[18] Such literature has acted as supportive evidence for vitreous traction in the role of maculopathy development.

A 2004 study evaluated 11 patients who underwent PPV, PVD induction, laser photocoagulation and gas tamponade for ODPM and found a complete resolution of serous maculopathy in all cases with an 18% recurrence rate.[19] In another, 11 patients underwent PPV, PVD induction and gas tamponade without laser.[20] Complete resolution of maculopathy was noted in 91% of the patients with no reccurrences.[20] Based on the pre-

Photos: Mohammad Rafieetary, OD





OCT imaging of the patient seen on page 87 shows outer retinal disruption (in yellow brackets, above), indicative of possible serous detachment in the past. At right, cavitation in the area of the pit is shown with the red arrow.

viously mentioned series, the use of laser photocoagulation may not be required for surgical success.

One surgical method that has been surrounded by controversy is the use of ILM peeling. Although many researchers have advocated its use, the benefits of this surgical technique remain questionable. In one study, 10 patients underwent PPV, PVD induction, ILM peel and gas tamponade with laser photocoagulation. Complete resolution occurred in 50% of patients and 70% gained greater than or equal to two lines of VA. However, complications of macular holes were seen.[21] In a different study, seven patients were treated in a similar manner and complete resolution was achieved in 86% of eyes with 71% reaching a final VA of greater than or equal to 20/30. In that case, four eyes resulted in full-thickness macular holes after one month.[22]

Possible indications for ILM peeling would be when maculopathy is found in conjunction with an epiretinal membrane or in the presence of overlying vitreoschsis. Given the number of cases that have been successfully performed in the absence of ILM peeling along with the relatively high incidence of macular hole development ILM peeling is generally avoided unless there is an underlying pathogenic role contributing to serous maculopathy due the ILM.[11,20-24]

Some authors suggest the removal of any glial tissue overlying (or adjacent to) the optic pit. One study looked at nine patients undergoing PPV, PVD induction, laser photocoagulation and gas tamponade. Serous maculopathy resolved in six out of six patients in which the glial tissue surrounding the optic pit was removed. It only resolved in two out three patients in which the glial tissue was not removed.[25] A 2008 case report demonstrated the benefits of glial tissue removal in a 45-year-old male who was diagnosed with ODPM. PPV, PVD induction and gas tamponade with glial tissue peeling was performed and retinal reattachment was achieved in six months with a final visual acuity of 20/20 without any visual field defects.[26] Though glial tissue peeling has proven beneficial in a limited group of studies, precaution must be taken when performing this procedure due to the glial tissue's close proximity to the optic nerve.[25,26]

Research suggests that sealing the CODP during surgery to prevent the passage of fluid into the intraretinal and subretinal spaces can prove beneficial.[27] Other techniques used to seal a CODP include using an autolohous scleral flap, inverting peeled ILM and using a Tisseel fibrin sealant.[6]

Recently, investigators have proposed a new strategy in ODPM treatment—the creation of inner retinal fenestrations temporal to the optic disc. In theory, this procedure works by disturbing the pressure gradient, which pushes fluid from the inner retinal layers into the subretinal space. This creates retinal fenestrations that allow for the diversion of fluid back into the vitreous.

In one of the largest published series on ODPM treatment, investigators noted complete resolution of maculopathy in 94% of eyes with a final VA greater than or equal to 20/30 in 56% of eyes. Retinal fenestrations can close prematurely during the early postoperative period.[28]

Clinical Pearls

No established guidelines for the treatment of ODPM exist, nor has a clear consensus on the mechanism of pathogenesis or the origin of the subretinal fluid been reached.[6,11] This makes it difficult to determine the optimal surgical technique. Typically, ODPM is treated with a combination of juxtapapillary retinal laser photocoagulation, PPV, gas tamponade and ILM peeling.[16]

Additional surgical elements may include subretinal drainage, glial tissue peeling, and sealing the CODP.[11] PVD induction is worthwhile to perform to relieve any vitreoretinal traction.

Treatment with laser photocoagulation is never performed alone and macular buckling is rarely performed due to its level of complexity.[15,18] Though PPV with

intraretinal fenestrations have provided promising results but further studies are required.

Spontaneous resolution can occur in 25% of cases but most have a poor visual prognosis if left untreated.[6,29] Patients presenting with ODPM should be referred to a retina specialist promptly. Prophylactic parapapillary laser has not been studied as a preventative measure against ODPM.[30] Patients should be advised to return to clinic immediately if any visual changes are noted. Yearly comprehensive eye exams with the use of at-home Amsler gird monitoring should be considered.

Our patient was issued a new polycarbonate spectacle prescription for full-time wear. She was educated on the condition and referred to a retinal specialist for a consultation.

Due to the longstanding nature of the condition, a guarded visual prognosis was emphasized. The patient was then instructed to return to clinic in three months for another evaluation. ■

Dr. Corzo is a graduate of Illinois College of Optometry and an optometry resident at Nova Southeastern University College of Optometry where he specializes in primary care with an emphasis on ocular disease.

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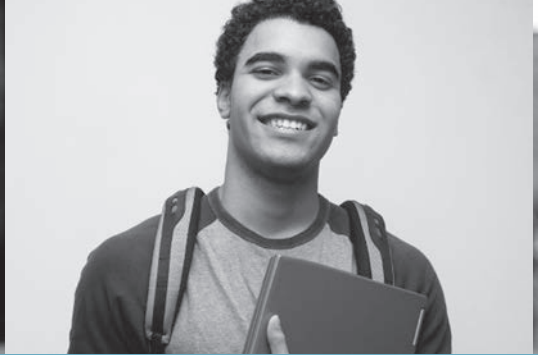


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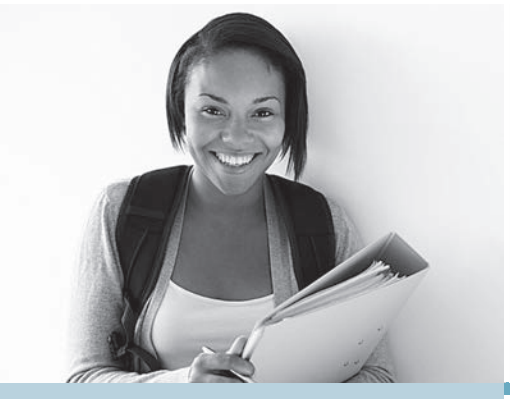

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PEER • REVIEWED • PRA • ARTICLE

When Drugs Cause Dry Eye

To identify and treat patients more effectively, know these systemic drugs and ocular drops linked to dry eye. **By Juan Ding, OD, PhD**

We all know that dry eye is one of the most common complaints of patients walking into an eye doctor's office. Dry eye is associated with older age, the female sex and prolonged use of digital screens. It can also be associated with and exacerbated by certain systemic and ocular medications.

Understanding which medications may affect dry eye helps practitioners identify, diagnose and treat patients' dry eye more effectively.

Systemic Meds That Affect Dry Eye

An estimated 22 of the top 100 best-selling drugs may cause dry eye.[1] Overall, 18 classes of drugs can negatively affect dry eye.[2] To date, most studies have looked at drug classes, rather than specific drugs, in terms of the effect on dry eye.

Also, most studies only demonstrate association, meaning that dry eye is associated with taking certain drugs, but the research cannot state definitively that dry eye is caused by taking these drugs.

One way of evaluating the risk for developing dry eye when taking a medication is to calculate the odds ratio (OR). An OR of 1 means a patient has equal risk for dry eye when taking this drug compared

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Estimated Time to Complete Activity: 2 hours

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Educational Objectives: After completing this activity, the participant should be better able to:

- Identify various drug classes, and perhaps some of the most commonly prescribed drugs in particular, that cause or exacerbate dry eye.
- Recognize the possible or theorized mechanisms of action between these drugs and dry eye.
- Effectively alter dry eye treatment to accommodate the patient's systemic drug regimen.
- Discuss how the patient's systemic therapy may be modified to better treat dry eye.

Target Audience: This activity is intended for optometrists engaged in the care of patients with dry eye.

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.

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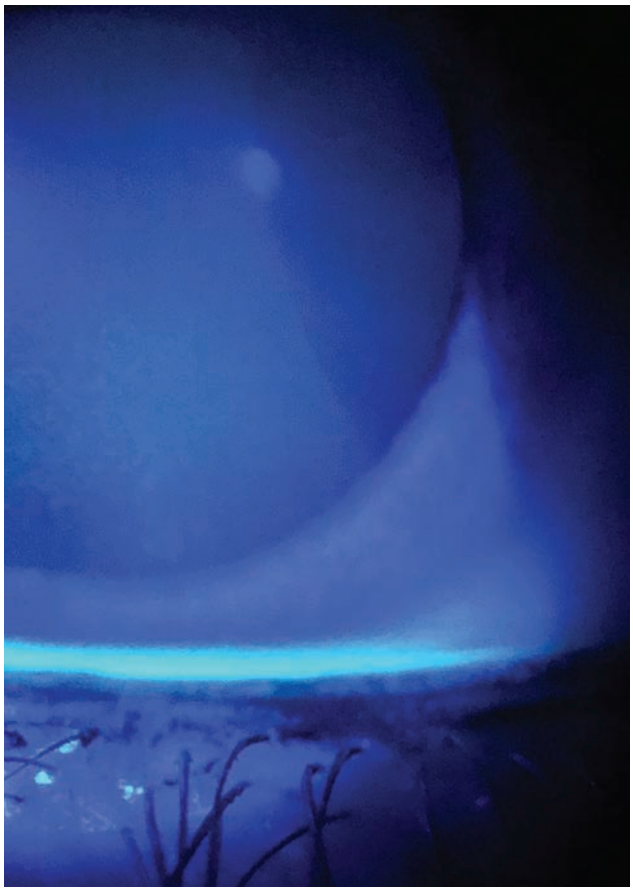
with patients not taking the medication. The higher the OR, the more likely a drug is associated with dry eye. For example, the odds ratio of antihistamines is 1.67, which means the likelihood of having dry eye when taking antihistamines is 1.67-fold compared with controls.

Table 1 summarizes the odd ratios of certain classes of systemic drugs in the order of highest to lowest OR in relation to dry eye risk. These data are compiled in the Tear Film & Ocular Surface Society (TFOS) Dry Eye Workshop (DEWS) II report on iatrogenic causes of dry eye.[2]



Corneal superficial punctate keratitis in a young adult taking several antidepressants.

How Systemic Drugs Affect Dry Eye

There are several possible mechanisms by which systemic drugs may cause dry eye: by decreasing tear production; by changing nerve input and reflex secretion; by inducing inflammation on secretory glands; and by direct secretion into the tears.[2] Drugs that are secreted into tears include aspirin, chloroquine, clofazimine, docetaxel, ethyl alcohol, hydroxychloroquine, ibuprofen, isotretinoin and amiodarone.[1] These may cause mechanical irritation, forming drug crystals or deposits in the tears or cornea, or may result in tear hyperosmolarity.[3-5]

Anticholinergics and adrenergics. Many systemic drugs have anticholinergic activity, which may explain their sicca effects. The anticholinergic activity can affect lacrimal gland acini, conjunctival mucus-producing cells and meibomian gland epithelial cells via cholinergic receptors.[2,6] This leads to reduced tear volume and quality. Drugs with anticholinergic activity include antidepressants, antipsychotics, anti-Parkinson's drugs, H1-antihistamines, decongestants and benzodiazepines.

On the other hand, adrenergic agents such as beta-blockers and alpha-agonists may cause changes in tear volume and quality, likely mediated by protein kinase C and intracellular calcium concentration.[2]

Chemotherapeutic drugs. These are known to cause dry eye syndrome. They may do so by inhibiting tear secretion or by affecting the meibomian glands, which are holocrine glands that constantly undergo cell renewal.[7] Some chemotherapeutic drugs may also cause blepharitis, such as epidermal growth factor receptor (EGFR) inhibitors and the proteasome inhibitor bortezomib, for example.[8] EGFR inhibitors may also impair corneal epithelial healing, leading to corneal epithelial defects.[9] Examples of EGFR inhibitors include monoclonal antibodies cetuximab and panitumumab, and the small-molecule EGFR inhibitors erlotinib and gefitinib.

Another dry eye-causing chemotherapy agent is trastuzumab, a monoclonal antibody directed against HER2 and used to treat HER2-overexpresssing breast and gastric cancers.[10] Orally available BRAF inhibitors vemurafenib, dabrafenib, and encorafenib—used for treating metastatic melanoma with a $BRAF^{V600}$ mutation—may also lead to dry eye. Trials of vemurafenib showed that dry eye occurred in 2% and conjunctivitis occurred in 2.8% of those taking it.[11]

Retinoic acid. Isotretinoin, a form of retinoic acid commonly known by its former brand name Accutane, is used to treat severe acne and certain cancers. Isotretinoin is secreted into the tears by the lacrimal glands and can cause atrophy of the meibomian glands, which can lead to changes in lipid secretion, tear osmolality and tear film stability.[12,13]

At the molecular and cellular level, research has found that isotretinoin inhibits meibomian gland epithelial cell proliferation, increases cell death, alters the expression of more than 6,000 genes and inactivates important survival signaling pathways.[14] Interestingly, isotretinoin has not been proven to directly cause harm to the conjunctiva or cornea, nor does it reduce lacrimal

gland tear secretion.[2]

Sex hormones. Researchers have extensively studied the important role androgens play in ocular surface tissues and dry eye. Androgens are critical in normal functions of the lacrimal glands, meibomian glands, conjunctiva and cornea. Androgen deficiency causes reduced tear volume and tear break-up time, increased meibomian gland disease (MGD) and dry eye signs and symptoms, as reviewed in the TFOS DEWS II *Sex, Gender, and Hormones Report*.[15] In fact, androgen decline with age is a common contributor to the age-related increase in dry eye in both men and women, and research shows androgen decline affects dry eye more than the decline of estrogen and progesterone in menopausal women.[15]

On the other hand, research shows estrogens have a negative effect on lacrimal glands and possibly meibomian glands as well, though data regarding estrogen is less well defined and appears to depend on sex and dosage.[15] For example, high levels of estrogen in hormone replacement therapy (HRT) increases the risk of dry eye.[15]

Polypharmacy. This refers to concurrent use of five or more medications, whether prescription or over-the-counter (OTC). Most of our knowledge of drug interaction is derived from research that studies two-drug interaction, and little is known about the additive effects when a patient is using three or more drugs.

Researchers suspect that polypharmacy, regardless of which individual drug is used, may increase the risk of dry eye, due to unknown and complicated multi-drug interactions.[1] Given that 36.7% of people older than age 60 use more than five drugs, it is no wonder that dry eye in the elderly is further exacerbated by their medication use.[17]

Table 1. Systemic Drugs Associated with Increased Risk for Dry Eye[1,2]

Drug Class	Odds Ratio	Examples
Antidepressants/ antipsychotics	2.54	*Antidepressants:* agomelatine, amitriptyline, bupropion, clomipramine, citalopram, desipramine, doxepin, duloxetine, fluoxetine, fluvoxamine, imipramine, mianserin, mirtazapine, nortriptyline, paroxetine, reboxetine, sertraline, tianeptine, trazodone, venlafaxine *Antipsychotics:* aripiprazole, brompheniramine, carbinoxamine, chlorpheniramine, chlorpromazine, clemastine, clozapine, cyproheptadine, dexchlorpheniramine, fluphenazine, haloperidol, lithium carbonate, olanzapine, perphenazine, promethazine, quetiapine, risperidone, sulpiride, thiethylperazine, thioridazine, thiothixene, trifluoperazine, ziprasidone
Anxiolytics/ benzodiazepines	2.35	alprazolam, diazepam, eszopiclone, lorazepam, zolpidem, zopiclone
Inhaled steroid use	2.04	No specific drugs listed in literature
Antihypertensives	1.98	*Adrenergic blocking:* acebutolol, atenolol, carvedilol, labetalol, metoprolol, nadolol, pindolol, clonidine, prazosin, oxprenolol, propranolol *Diuretic:* bendroflumethiazide, chlorothiazide, chlorthalidone, hydrochlorothiazide, hydroflumethiazide, indapamide, methyclothiazide, metolazone, polythiazide, trichlormethiazide
Anti-infectives	1.88	No specific drugs listed in literature
Hormone replacement therapy	1.69	estrogen/progesterone, medroxyprogesterone
Antihistamines	1.67	azelastine, brompheniramine, carbinoxamine, cetirizine, chlorpheniramine, clemastine, cyproheptadine, desloratadine, dexchlorpheniramine, diphenhydramine, doxylamine, epinastine, fexofenadine, hydroxyzine, ketotifen, loratadine, olopatadine, promethazine, pseudoephedrine, tripelennamine, triprolidine
Systemic corticosteroids	1.60	No specific drugs listed in literature
Antiulcer agents	1.44	No specific drugs listed in literature
Vasodilators	1.37	No specific drugs listed in literature
Antiandrogen therapy	1.35	alfuzosin, doxazosin, finasteride, leuprorelin, tamsulosin, terazosin
Sulfonylureas	1.30	No specific drugs listed in literature
Analgesics/ antipyretics	1.28	aspirin, ibuprofen
Cardiac glycosides	1.28	No specific drugs listed in literature
Antineoplastics	n/a	busulfan, cetuximab, cyclophosphamide, docetaxel, erlotinib, gefitinib, interferon, methotrexate, mitomycin c, panitumumab, vinblastine, verteporfin
Retinoids	n/a	isotretinoin
Antivirals	n/a	acyclovir

Ocular Medications That Affect Dry Eye

In addition to systemic drugs, certain eye drops can also induce or worsen dry eye. These include many popular glaucoma drops, allergy drops, antiviral drops, NSAIDs and common mydriatics that are used diagnostically in our offices (*Table 2*).

Various eye drops cause allergic, toxic and/or immune/inflammatory effects. This may occur due to the interaction with the tear film and disruption of the lipid layer through detergent effects. These drops can also directly damage goblet cells, the conjunctival and corneal epithelia, corneal nerves and meibomian glands.[2]

One common ingredient of most preserved eye drops is benzalkonium chloride (BAK), which disrupts the tear film by altering tear film osmolarity.[18] BAK also has pro-inflammatory and toxic effects on the ocular surface.[19]

These cases highlight the link between dry eye and topical medication use:

Case 1. A 25-year-old female complained of blurry vision and difficulty focusing while using a computer for two months. Her medical history was significant for depression and anxiety; she has been on escitalopram and buspirone for six months and omeprazole PRN for acid reflux. Otherwise, she was healthy and not taking any other medications, including OTC. Her entering uncorrected visual acuity was 20/40 in each eye, but with difficulty. Her refractive error was minimal. Her slit lamp exam was remarkable for 3+ corneal superficial punctate keratitis (SPK) in the lower half of both eyes with a reduced tear meniscus. Her eye examination was otherwise unremarkable.

She was started on OTC artificial tears in both eyes four times a day and warm compress of eyelids twice a day. She was scheduled to return in two weeks for follow up.

Table 2. Topical Drugs That May Cause Dry Eye[1,2]

Drug Class	Examples
IOP-lowering drops	*Beta-blockers* betaxolol carteolol levobunolol metipranolol timolol *Alpha-agonists* apraclonidine brimonidine *Carbonic anhydrase inhibitors* brinzolamide dorzolamide *Cholinergic agents* pilocarpine *Prostaglandins* bimatoprost latanoprost travoprost unoprostone
Allergy drops	emedastine olopatadine
Antiviral agents	acyclovir idoxuridine trifluridine
Decongestants	naphazoline tetryzoline
Mydriatics and cycloplegics	cyclopentolate tropicamide hydroxyamfetamine
Preservatives	benzalkonium chloride
Topical and local anesthetics	cocaine proxymetacaine tetracaine
Topical NSAIDs	bromfenac diclofenac ketorolac nepafenac

At the next visit, her visual acuity had improved to 20/30 and the SPK significantly improved to grade 1. Accommodative dysfunction was suspected due to her ability to see 20/20 with only +0.25-0.25x180.

Sure enough, her accommodative amplitude was 4D in each eye and NRA/PRA at +2.00/-1.25. Given this finding, PALs were prescribed with distance MRx and +1.50 add. Since her dry eye was well controlled, she was instructed to continue the artificial tears and warm compress, and was scheduled for follow up in three months. If her dry eye gets worse, meibography may be employed to examine the health status of the meibomian gland. Because 70% of dry eye has an MGD component, it is often necessary to treat MGD to treat dry eye successfully long-term.

Escitalopram and buspirone are commonly prescribed antidepressants. As discussed earlier, certain antidepressants have the potential to cause dry eye. In addition, they can affect the accommodative system of the eye.[20] We should be on the lookout when a young and otherwise healthy patient presents with dry eye and/or accommodative dysfunction for a possible association with their antidepressant use.

Case 2. A 31-year-old female patient with cutaneous melanoma was taking dabrafenib and trametinib for four months before developing redness, photophobia and mild eye pain. Her ocular exam revealed bilateral iritis as well as dry eye. Her iritis resolved with

1% prednisolone acetate, but her dry eye persisted. Fortunately, the dry eye responded well to preservative-free artificial tears.

Dabrafenib is known to cause uveitis, but an often-overlooked side effect is dry eye. In drug-induced uveitis such as this, we should also check for dry eye, which sometimes causes persistent symptoms even after uveitis is resolved.

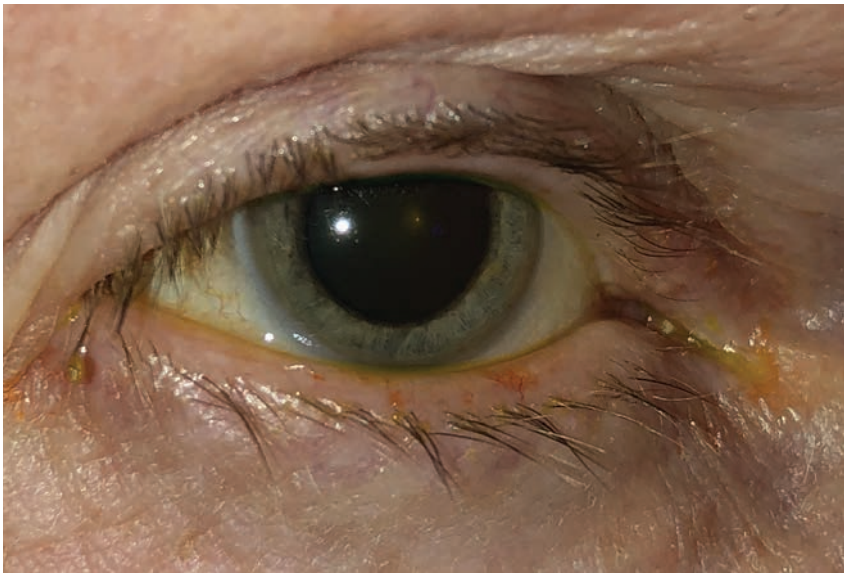
Case 3. A 25-year-old male graduate student took isotretinoin for six months to treat acne. Afterwards, he developed persistent dry eye. He has tried numerous OTC artificial tears, warm compresses, punctal plugs, autologous serum drops, meibomian gland probing and LipiFlow (Johnson & Johnson Vision), but is still battling with pain and burning every day. This case exemplifies how extremely challenging it can be to treat MGD induced by isotretinoin.

Case 4. A 70-year-old female with ocular rosacea and nasal allergies complained of a gritty feeling and tearing of both eyes for one year. She is a classic example of a patient at increased risk for developing dry eye from ocular rosacea alone. Added to this, she's taking antihistamines orally for her longstanding allergies, which has further exacerbated the dry eye.

In situations such as this, we should let patients know of the multiple risk factors for dry eye, implement more aggressive dry eye management and stress the importance of consist day-to-day eyelid hygiene and use of lubrication.

Treating Drug-Related Dry Eye

We can treat iatrogenic dry eye with various therapies. First line is artificial tears and eyelid hygiene. If these are not adequate to control dryness, a topical steroid may be used in the short term, as well as Restasis (cyclosporine 0.05%, Allergan) and Xiidra (lifitegrast 5%, Novartis) long-term.



This patient has both ocular rosacea and dry eye.

Punctal plugs are good options for aqueous-deficient dry eye. But for the majority of patients who have evaporative dry eye, MGD may be treated with LipiFlow, TearCare (Sight Sciences), iLux (Alcon) or another targeted heat delivery system to the eyelids. For dry eye patients with rosacea, intense pulsed light can be especially helpful. Finally, for severe dry eye, amniotic membrane, autologous serum drops and therapeutic scleral lenses may provide relief.

Patient education is key. Rather than brushing off patients with comments such as, "you just have some dry eye," it is usually better to offer an explanation as to why they are suffering from the condition. For example, you should explain the impact of HRT on dry eye to a postmenopausal woman on HRT who has an exacerbation of dry eye for a few months. The treatment may not necessarily discontinue, but the patient should understand that the gritty feeling in her eyes may be expected, and that she should follow the recommended dry eye treatment.

Consider the following alternatives for patients with dry eye-related to systemic medications:

- ***Change to another drug (class).*** This is a nice concept but sometimes difficult in practice. Again, most research looks at drug class rather than individual drugs, making it hard to know which drug may be a better choice; perhaps the entire class will have similar effects on dry eye. Changing medications is certainly worth trying and perhaps for a given patient, an effective drug can be found that lessens dry eye problems. It is important to communicate with the patient's primary care provider or treating physicians for alternative medications that may have fewer dry eye effects.
- ***Reduce dosage.*** A reduced medication dosage may be achievable if the medication is still effective in treating the patient's main health condition. As optometrists, we should communicate with the treating physician to see if a reduced dosage is possible.
- ***Change dosing time schedule.*** If a patient's medication does not require a specific time to take, we may advise the patient to take it at a different time to avoid peak concentration or side effects that may

interfere with their daily activities. For example, certain medications may be taken before bed rather than in the morning so that the dry eye effect occurs while the eyes are shut, reducing the effect on daily activities such as driving or working.

• ***Discontinue a medication.*** The determination to switch or terminate a medication suspected to cause dry eye has to be on a case by case basis. For example, chemotherapeutic drugs are rarely worth stopping to alleviate the comparatively minor side effect of dry eye.

For acne, isotretinoin is usually reserved for severe forms and typically for younger patients. The duration of isotretinoin therapy is typically four to six months. The long-term compromise in patient quality of life would be devastating if they develop irreversible MGD from this treatment. Although some patients' dry eye will resolve a few months after discontinuation of isotretinoin, for some the dry eye persists for many years or may be permanent.[12] One retrospective study evaluating 1,741 subjects on Accutane discovered that 1% of these patients developed irreversible dry eye.[21]

Some dermatologists send patients for an eye evaluation prior to initiating isotretinoin therapy. This examination should include a careful examination of the patient's ocular surface, including the meibomian glands, and a check for other dry eye contributing medications. If no preexisting MGD or dry eye exist, and the patient is not on other medications that may be related to dry eye (e.g., antidepressants or antihistamines), you may decide to clear the patient for the therapy, with close monitoring of ocular surface signs and meibomian gland evaluation.

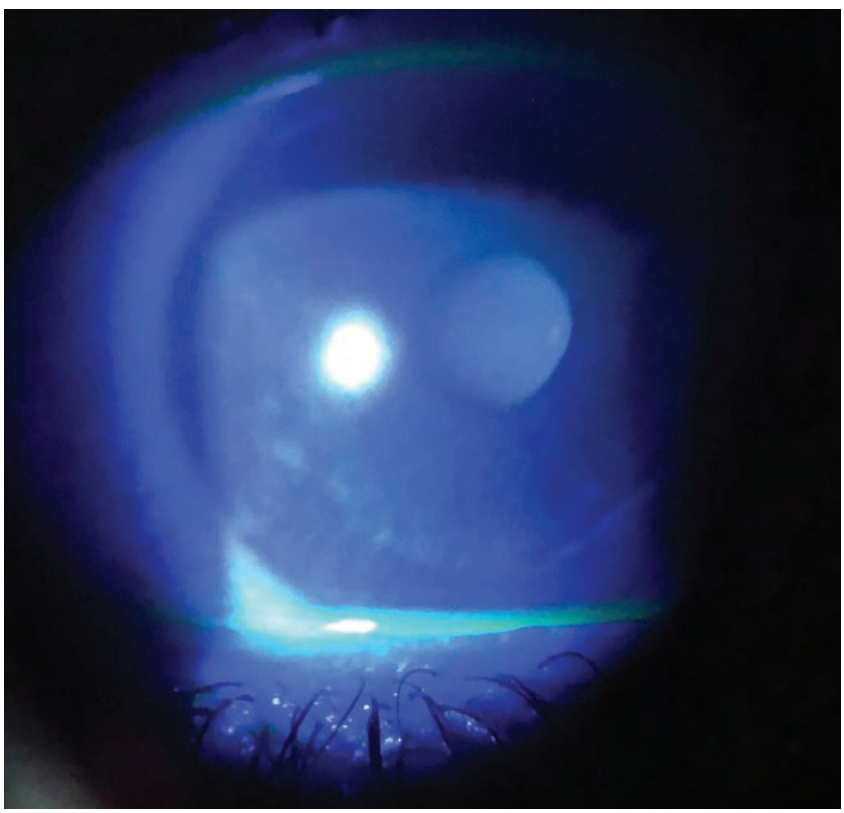
Should early signs of dry eye emerge, the patient should start immediate dry eye therapy such as artificial tears, eyelid hygiene and MGD treatment. Communicate with the treating dermatologist to seek alternative therapy.

For patients with dry eye related to topical ocular drops, consider the following options:

• ***Switch to a drop with a different preservative.*** One major issue for chronic eye drop users is the preservatives in the drops. Glaucoma patients may need to be on IOP-lowering drops for decades, and many patients develop redness/irritation after instilling drops. Several IOP-lowering drops employ preservatives other than BAK, which may be better tolerated than BAK-containing drops and may serve as alternative medications for glaucoma. Travatan Z (travoprost, Novartis) is preserved with SofZia, which contains borate, zinc and sorbitol. Alphagan P (brimonidine, Allergan) is preserved with Purite, a stabilized oxychloro complex that has broad antimicrobial activity through oxidative damage. While these have lower toxic activities than BAK, the TFOS DEWS II *Iatrogenic Report* cautions that they have not been fully investigated in terms of dry eye.[2]

• ***Go preservative free.*** Preservative-free glaucoma drops are also available, which can be much better for dry eye. Examples include Zioptan (tafluprost, Akorn), Cosopt PF (dorzolamide-timolol, Akorn) and Timoptic in Ocudose (timolol maleate, Valeant Pharmaceuticals).

• ***Consider surgery.*** The problem with preservative-free glaucoma drops is that they can be much more costly than preserved ones. Patients may save opened containers, which may lead to contamination. Other alternatives include laser procedures, such as selective laser trabeculoplasty or minimally invasive glaucoma surgeries to reduce drop dependence.



Dry eye in a patient with melanoma taking chemotherapeutic medications.

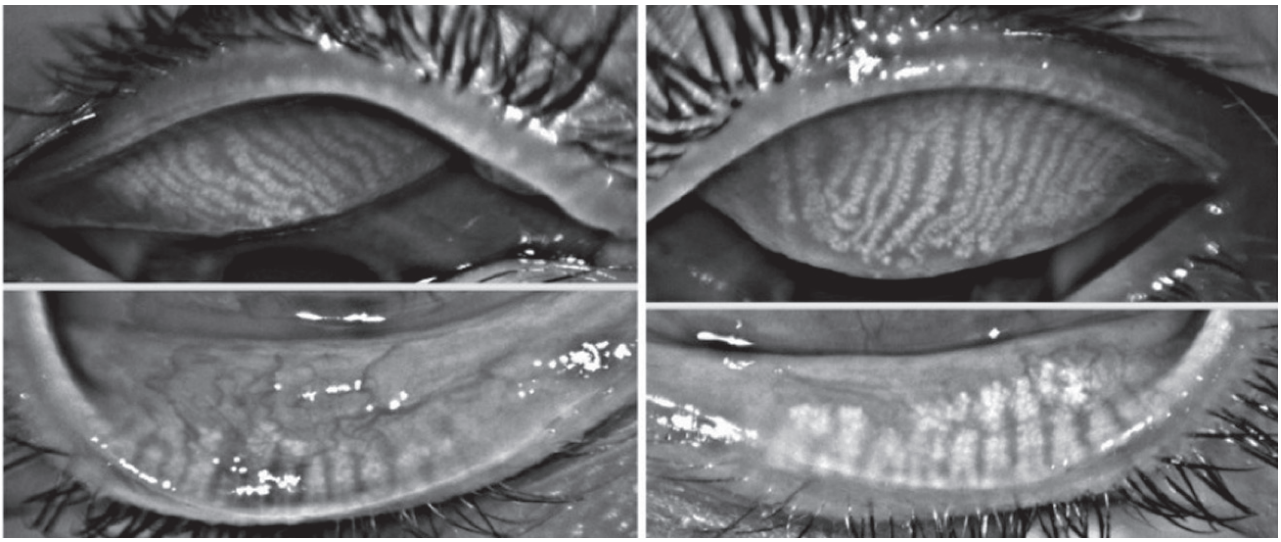


Photo: Nancy Mcnamara, OD, PhD

This is what Accutane-induced dry eye looks like on meibography.[12]

• ***Switch allergy drops and/or add artificial tears.*** Another ocular med that may be used long-term is an allergy drop. For patients with allergic conjunctivitis, these may be used for months or even year-round. If dry eye gets worse while on olopatadine or emedastine, for example, consider alternative drops such as ketotifen, azelastine, epinastine, alcaftadine or bepotastine.

If a patient has concurrent allergy and dry eye, always tell them to use artificial tears as well, and educate patients that their allergy drops will not help with dry eye, and their dry eye needs to be managed in addition to their allergies.

• ***Advise against red eye-reducing drops.*** For patients with allergies or chronic red eyes, another potentially abused eye drop is OTC redness relievers containing decongestants such as naphazoline and tetryzoline. As optometrists, we should inform patients that these drops will not only cause rebound redness if used long term, but also may cause dry eye, making their symptoms worse.

A good redness-relieving drop that does not cause rebound redness is Lumify (brimonidine, Bausch + Lomb), now available OTC, which patients can use in conjunction with other dry eye treatments.

We live in a world of pharmaceuticals, where almost every medical condition has a potential solution involving pills, drops, injectables, inhalers, transdermal patches or other delivery methods. Unfortunately, dry eye—the number one reason patients visit eye care practitioners—is affected by a multitude of drugs, both prescription and OTC.

As optometrists, we are in a unique position to help patients find possible causes of their dry eye. One of the ways we do that is by knowing the ever-increasing list of medications that can induce or aggravate dry eye. This is essential to providing patients with proper education and successful dry eye treatment. ■

Dr. Ding is an assistant professor in the Department of Ophthalmology & Visual Sciences at the University of Massachusetts Medical School and started the optometric service at the Eye Center at UMass Memorial Medical Center. She has conducted and published research on dry eye disease and MGD, and was a coauthor of the TFOS Dry Eye Workshop II 2017.

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OSC QUIZ

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1. Which of these statements related to drugs and dry eye syndrome is true?
a. Systemic medications not used to treat eye diseases will rarely cause eye-related side effects such as dry eye syndrome.
b. Many systemic drugs used to treat other organ systems may affect dry eye.
c. Eye drops are the only medication that causes ocular surface irritation.
d. None of the above.

2. Which of the following classes of drugs is *not* known to be associated with dry eye?
a. Antihistamines.
b. Antidepressants.
c. Anticoagulants.
d. Antihypertensive drugs.

3. How many drugs are generally considered to constitute polypharmacy?
a. Two.
b. Three.
c. Four.
d. Five.

4. Which of the following antidepressants is associated with dry eye?
a. Paroxetine.
b. Sertraline.
c. Trazodone.
d. All of the above.

5. Which of the following antipsychotics may cause dry eye?
a. Chlorpromazine.
b. Lithium carbonate.
c. Thioridazine.
d. All of the above.

6. Which of these antihypertensive drugs is known to be associated with dry eye?
a. Beta-blockers.
b. ACE inhibitors.
c. Diuretics.
d. Both a and c.

7. Which of these hormone drugs may cause dry eye side effects?
a. Growth hormone.
b. Estrogens.
c. Corticosteroids.
d. Both b and c.

8. The following systemic medications are known to be secreted into tears, *except*:
a. Aspirin.
b. Ethyl alcohol.
c. Hydroxychloroquine.
d. All of the above are secreted into tears.

9. What is the mechanism of Accutane-induced dry eye?
a. Causing reduced tear volume.
b. Damaging conjunctiva.
c. Acting on cornea directly to cause toxicity.
d. Inducing MGD.

10. How do anticholinergic drugs cause dry eye?
a. By affecting lacrimal glands.
b. By affecting conjunctival mucus-producing cells.
c. By affecting the meibomian gland.
d. All of the above.

11. Which of these drugs is *not* considered to have anticholinergic activities?
a. Antidepressants.
b. Corticosteroids.
c. Decongestants.
d. Benzodiazepines.

12. What is *not* a mechanism by which adrenergic agents cause dry eye?
a. Reducing tear volume.
b. Modulating intracellular calcium levels.
c. Causing MGD and blepharitis.
d. Changing protein kinase C levels.

13. Which of these drugs known to cause retinal changes may also cause dry eye?
a. Hydroxychloroquine.
b. Amiodarone.
c. Thioridazine.
d. All of the above.

14. Which of these chemotherapeutic drugs is known to be associated with dry eye?
a. BRAF inhibitors.
b. EGFR inhibitors.
c. HER2 inhibitors.
d. All of the above.

15. Which class of glaucoma drugs (preservatives aside) does *not* cause dry eye symptoms?
a. Prostaglandin analogs.
b. Alpha agonists.
c. Beta-blockers.
d. None of the above.

16. A 65-year-old man with moderate stage primary open-angle glaucoma has been on timolol and latanoprost for one year before developing ocular redness and irritation, especially after applying drops. All of these are good therapy approaches, *except*:
a. Discontinue glaucoma drops, as the patient's quality of life is more important.
b. Switch latanoprost to Travatan Z and timolol to Timoptic in Ocudose.
c. Start the patient on preservative-free artificial tears.
d. Switch his glaucoma drops to Cosopt PF.

17. All of these are used as a preservative in eye drops, *except*:
a. BAK.
b. Glycerol.
c. SofZia.
d. Purite.

18. Which of these allergy eye drops may actually worsen dry eye?
a. Ketotifen.
b. Olopatadine.
c. Alocril.
d. Alomast.

19. Of the following patients with complaints of dry eye, which is most likely drug-related dry eye?
a. 60-year-old female with Sjögren's syndrome currently taking Plaquenil.
b. 30-year-old healthy female office secretary taking vitamin D supplement only.
c. 22-year-old healthy male on antidepressant and no other medications.
d. 40-year-old male with blepharitis and MGD, taking Viagra and no other medications.

20. Which of the following topical drops is not associated with dry eye?
a. Cycloplegics and mydriatics.
b. Glaucoma drops.
c. Topical NSAIDs.
d. None of the above.

Examination Answer Sheet

When Drugs Cause Dry Eye

Valid for credit through October 15, 2022

Online: This exam can be taken online at www.reviewsce.com. Upon passing the exam, you can view your results immediately and download a real-time CE certificate. You can also view your test history at any time from the website.

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Answers to CE exam:

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2. Ⓐ Ⓑ Ⓒ Ⓓ
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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. Identify various drug classes, and perhaps some of the most commonly prescribed drugs in particular, that cause or exacerbate dry eye. ① ② ③ ④ ⑤

22. Recognize the possible or theorized mechanisms of action between these drugs and dry eye. ① ② ③ ④ ⑤

23. Effectively alter dry eye treatment to accommodate the patient's systemic drug regimen. ① ② ③ ④ ⑤

24. Discuss how the patient's systemic therapy may be modified to better treat dry eye. 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:

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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 _____

Lesson 118755 RO-OSC-1019

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

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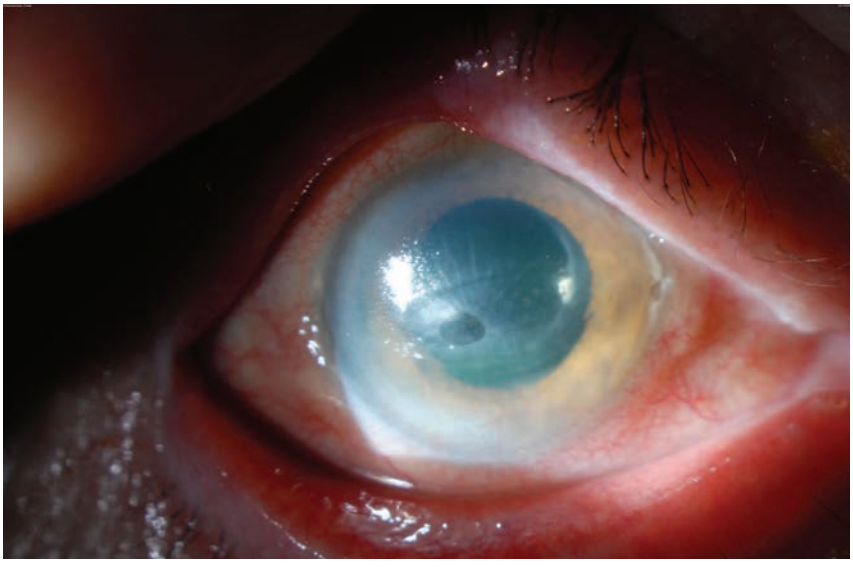
Challenge Accepted

Neurotrophic keratopathy can be difficult to manage, but ever-expanding treatment options are enhancing our ability to achieve successful results.

Edited by Joseph P. Shovlin, OD

Q Shingles has devastated the corneal sensation in one of my patients who has a persistent corneal defect from the resulting neurotrophic cornea. I've tried serum tears and amniotic membranes twice to no avail. How should I approach this?

A For the cornea specialist, neurotrophic keratopathy is one of the most challenging conditions to manage. Its chronic and relentless nature causes frustration among patients, who often try multiple different medications without seeing any significant improvements. Fortunately, our arsenal of tools to manage this disease has broadened significantly over the past five to 10 years, with newer tools demonstrating increasing promise.



A non-healing corneal ulcer in an eye with a history of herpes zoster ophthalmicus. Note the heaped-up epithelial edges. The radial striae indicate significant corneal thinning.

Maneuvering Management

Neurotrophic keratopathy severity can be broadly classified into three stages.[1] Stage one (mild) involves ocular surface irregularity, while stage two (moderate) presents with non-healing epithelial defects and stage three (severe) involves corneal ulceration with subepithelial tissue loss. Stage three is particularly concerning because of its potential to progress to corneal perforation.

While stage one disease can be treated with frequent lubrication (typically preservative-free artificial tears every two hours during the day), stages two and three often require more aggressive management. Punctal plugs, serum tears, bandage contact lenses, self-retained amniotic membranes and amniotic membrane transplantation are often used to promote healing. Referring patients to an oculoplastic surgeon for a temporary tarsorrhaphy—surgical closure of the eyelids over the corneal defect—often results in resolution of the defect. Christopher J. Rapuano, MD, chief of the cornea service at Wills Eye Hospital, recommends this course of action for this patient.

Another potential solution is referring patients to a contact lens specialist for a scleral lens fitting. By vaulting over the cornea, scleral lenses create a tear-filled space that keeps the ocular surface hydrated and protects the cornea from the microtrauma associated with blinking. Occasionally, serum tears are used with scleral lenses to bathe the cornea in the serum tears throughout the day. An additional benefit of a scleral lens includes refractive error correction.

Recently, interest has grown in the use of recombinant human nerve growth factor (rhNGF) agents to improve corneal health in patients with neurotrophic keratopathy. The REPARO trial evaluated the efficacy of rhNGF agents in promoting corneal epithelial healing in cases of moderate-to-severe neurotrophic keratopathy.[2] Compared with vehicle-treated eyes, those treated with rhNGF agents achieved higher rates of healing at four and eight weeks.[2] This effect appears to last for at least one year.

The commercially available rhNGF agent, Oxervate (cenegermin-bkbj, Dompé), entered the market in January of this year and should be administered six times a day for eight weeks. Common adverse effects of the drug are eye pain and hyperemia. In this patient's case, Zeba A. Syed, MD, co-director of the cornea fellowship at Wills Eye Hospital, believes it is reasonable to start Oxervate now or after the epithelial defect has healed from the tarsorrhaphy.

Finally, in rare circumstances, we can look to our plastics colleagues to assist us in performing corneal neurotization procedures. This surgery provides an alternate source of innervation that serves to stabilize the corneal epithelium and help ensure success if a future keratoplasty is needed for corneal opacity secondary to neurotrophic keratopathy.

We can achieve corneal sensory reconstruction using one of several nerves, such as a branch of the sural nerve, with the supratrochlear nerve serving as the donor nerve. There is documented evidence of improved corneal sensation six to eight months post-procedure.[3]

These treatment regimens may be used in combination to rehabilitate the ocular surface, but ultimately deciding which management path to pursue depends on disease severity, prior treatment regimens, clinician experience and comfort level.

Although neurotrophic keratopathy is a difficult clinical condition to manage, the recent expansion of treatment options has enhanced our ability to achieve successful results. ■

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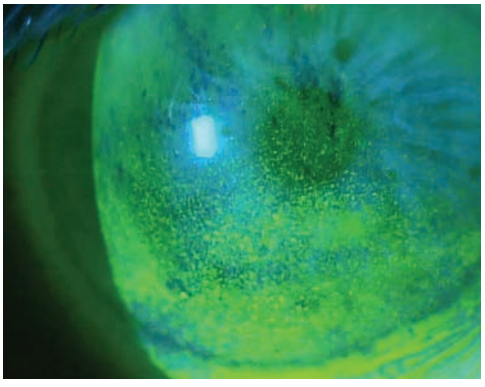
Pressure on the Surface

Glaucoma patients using topical therapy often spiral into chronic dry eye. Here's why.

By Paul M. Karpecki, OD

As optometrists, we are integral to treating both dry eye disease (DED) and glaucoma at the earliest stages, yet our care is too often reactive. Research shows the comorbidity of DED in patients treated topically for ocular hypertension and glaucoma could be as high as 59%.[1] We have many strategies in our arsenal to help patients get the glaucoma treatments they need without necessarily compromising their ocular surface health. These include smart medication prescribing and glaucoma surgery, whether it's traditional options or minimally invasive glaucoma surgery (MIGS).[2-4]



Corneal superficial punctate keratitis in a patient on multiple topical glaucoma medications.

Glaucoma Med Conundrum

Topical IOP-lowering medications, the mainstay of glaucoma therapy, are a godsend for those who need them. But all five classes of medication used—beta blockers (β-blockers), carbonic anhydrase inhibitors (CAIs), prostaglandin analogs (PGAs), alpha 2-adrenergic agonists and rho-kinase inhibitors—come with adverse reactions, many of which are localized to the ocular surface. These can impact everything from ocular surface health to diagnostic testing and compliance.

Ocular surface. Allergy, toxicity, immuno-inflammatory effects, superficial punctate keratitis, conjunctival inflammation and disruption of the tear film are all among the many side effects of both the glaucoma medications and the compounds used to preserve them.[5,6]

Preservatives such as benzalkonium chloride (BAK) are classified as quaternary ammonium compounds that act as a detergent and can disrupt the cell membrane, reduce epithelial cell proliferation and decrease corneal epithelial tight junctions.[7,8] These detergents disrupt the lipid layer, resulting in a decreased break-up time and the potential for evaporative dry eye issues.[9] Although both preserved and preservative-free PGAs cause meibomian gland dysfunction (MGD), preserved forms are more toxic to the meibomian glands, resulting in a greater reduction of mean acinar cell density and area, less homogeneity and significant alterations to secretions.[10] All of this means the meibomian glands are more prone to atrophy.

One study looking at the effects of both β-blockers and PGAs on meibomian gland morphology found a positive correlation between topical treatments and decreased gland structure and function.[11]

PGAs are pro-inflammatory. Such chronic inflammation not only affects epithelial cell function but can also damage the structure and function of sensitive oil-producing meibomian glands.[12] One study found more than 90% of patients on PGAs had MGD.[13] PGAs also induce a significant involution of meibomian lipoid activity.[14]

A recent study found signs of corneal and conjunctival ocular surface disease were evident in 44% of patients treated with PGA therapy.[5] Goblet cells on the conjunctiva, the main source of mucoproteins, are essential in preventing conjunctival epithelial disquamation, inflammation and cell death. Numerous studies show a significant loss of goblet cell density with long-term use of preserved glaucoma medications, and PGAs in particular.[15-18]

The prevalence of blepharitis, MGD and dry eye is more than twice as high after the commencement of topical glaucoma therapy compared with before therapy, and with each additional medication the risk of an adverse event or possible exacerbation of DED multiplies.[5,19-21]

Long-term topical glaucoma therapy can so drastically disrupt the ocular surface that it can impact future surgical options if the condition continues to progress.[22] For example, a disrupted ocular surface results in inaccurate keratometry readings, which could directly affect intraocular lens calculations. It can also lead to scarring that can make shunt and trabeculectomy procedures difficult or less effective.

Diagnostic testing. Tear film disruption can affect the reliability and reproducibility of diagnostic testing such as visual fields.[23] One study found treating glaucoma patients' DED for one week with just artificial tears improved their visual field test duration, mean deviation and the number of depressed points.[23]

Compliance. Topical glaucoma therapy's effect on the ocular surface can lead to decreased medication compliance—a known risk factor for disease progression.[24] Patients who have problems with their topical glaucoma medication are at higher risk for poor compliance, with typical medication non-adherence ranging between 50% and 60%.[5,25]

Smart Prescribing

While keeping IOP under control is a primary aim, practitioners must balance this alongside the practical realities that are inherent with the long-term use of topical therapy.

Preservatives such as BAK are added to topical medications to prevent microbial proliferation after opening; however, they frequently cause or aggravate ocular surface disease in glaucoma.[27,28] For glaucoma treatment to be effective, clinicians must minimize the side effects to promote compliance.[29]

Manufacturers are increasingly developing preservative-free drops and combination therapies aimed at minimizing exposure to preservatives and other irritants that could potentially affect ocular surface health, comfort and compliance.

We've all witnessed the effects of ocular surface disease in glaucoma and the consequences of long-term comorbidity. We can, and should, do everything in our power to address this growing problem. It's up to us to introduce patients to the treatment possibilities that can address their glaucoma without compromising the ocular surface.[2] ■

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

Cut the Drops with Surgery

Alternative therapies such as selective laser trabeculoplasty (SLT) or MIGS may eliminate medication-induced effects, reducing the risk for dry eye symptoms.

The largest MIGS trial illustrates the capacity of this approach to reduce or eliminate patients' dependence on medications. The HORIZON trial compared the efficacy of the Hydrus microstent (Ivantis) plus cataract surgery vs. cataract surgery alone in mild to moderate glaucoma. Two years post-op, 78% of patients with the combination surgery were medication-free, an improvement of 30% over those with cataract surgery alone.[4]

Studies show MIGS procedures have a lower risk profile than conventional surgery—a fact that is slowly leading to a shift in care.[2,3] But adoption remains low, possibly due to our tendency to wait before referring patients for cataract surgery and our hesitancy to educate patients on surgical options. We can and should provide stronger recommendations.

Until recently, surgery was an unappealing option. With filtering surgery, we worried about infection, hypotony, inflammation and bleb-related complications. And although SLT is an excellent treatment option for many, it demonstrates diminished efficacy over time.[26] MIGS, on the other hand, offers an entirely different approach and effectively addresses the needs of patients with mild to moderate glaucoma. What's more, MIGS has a good safety profile and patients recover rapidly.[10]

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Be Home Before Dark

Can imaging help find a course of action for a patient with lifelong myopia who is now experiencing progressive vision loss? **By Mark T. Dunbar, OD**

A 72-year-old Caucasian female presented with a longstanding history of blurred vision in both eyes all her life. In fact, she reported growing up having high myopia until she had cataract surgery in both eyes approximately 10 years earlier. Her vision improved following cataract surgery but it was still never perfect. She reported a slow progressive loss of vision in her right eye over the past three or four years. The left eye sees better but, she reported, it's not as sharp as it used to be.

Her medical history is significant for Type II diabetes for more than 10 years. Her blood sugar is controlled and her last hemoglobin A1c was 6.0. She had a head injury relating to a motor vehicle accident approximately 12 years ago, which resulted in a cerebral hemorrhage. She now gets occasional seizures. This has been stable.

Evaluation

On examination, her best-corrected visual acuities were 20/80 OD and 20/40 OS. Her confrontation visual fields were full-to-careful finger counting OU. Her ocular motility testing was normal, and the pupils were equally round and reactive to light without an afferent pupillary defect. The anterior segment was significant for the presence of clear posterior chamber intraocular lenses in both eyes. Her tensions by Tonopen (Reichert) measured 14mm Hg OU.

On dilated fundus exam, the vitreous was clear. The optic nerves were tilted with a small cup, and there was peripapillary atrophy present around both optic nerves. Obvious retinal changes were visible in the macula of each eye. An SD-OCT was performed and is available for review (*Figure 1*).

We continued to follow this patient closely. On exam one year later, her vision eye had declined to 20/400 OD and 20/80 OS. The SD-OCTs are available for review (*Figure 2*).

Over the next two years, the vision in her right eye never stabilized or improved and now her left eye is starting to decline. The OCT from two years later is also available (*Figure 3*).

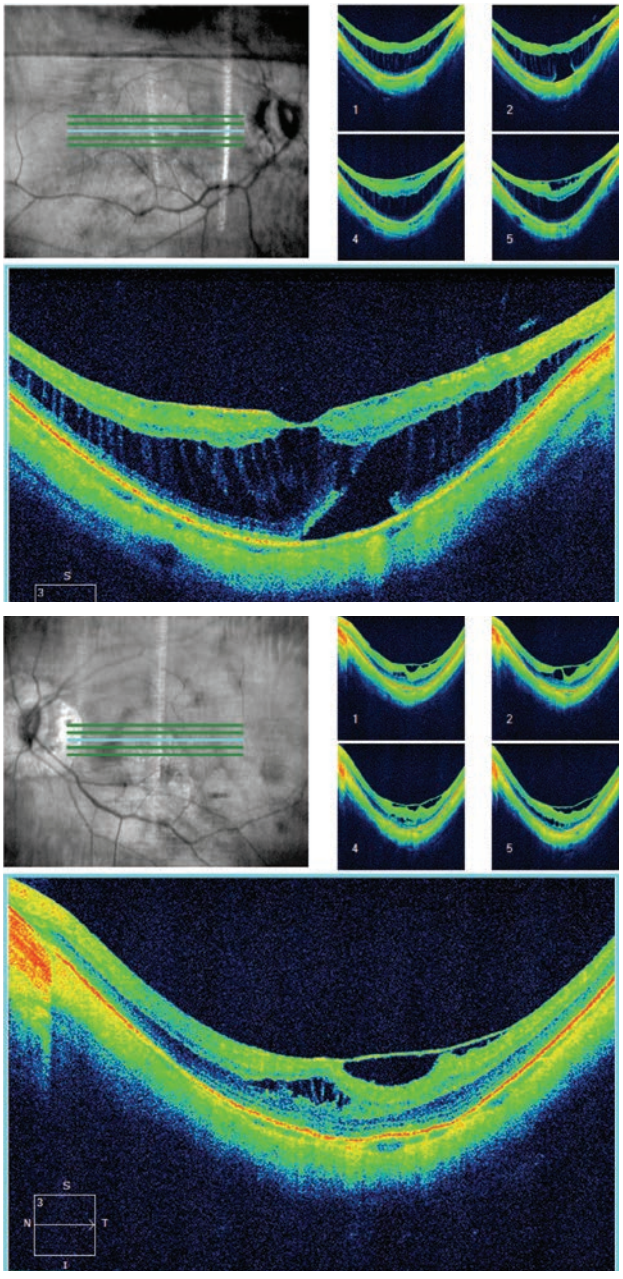

Fig. 1. These OCT images of the right (at top) and left eye on initial presentation. Note the obvious changes in both maculae.

Take The Retina Quiz

1. On her initial visit, what is the most likely diagnosis?
a. Cystoid macular edema.
b. X-linked juvenile retinal schisis.
c. Myopic tractional maculopathy.
d. Central serous retinopathy.

2. On the second visit, the right eye is worse. How would you characterize the right eye based on the OCT?
a. Neurosensory retinal detachment and loss ellipsoid zone.
b. Retinal pigment epithelium detachment.
c. Impending macular hole.
d. Full-thickness macular hole.

3. On the third visit, how would you characterize the right eye at the follow up?
a. Full-thickness macular hole.
b. Pseudo-macular hole.
c. Chorioretinal scaring.
d. Choroidal neovascularization.

4. On the third visit, how should the left eye be managed?
a. Observation.
b. Anti-VEGF injection.
c. Pars plana vitrectomy, membrane peel.
d. Intravitreal injection of Ocriplasmin.

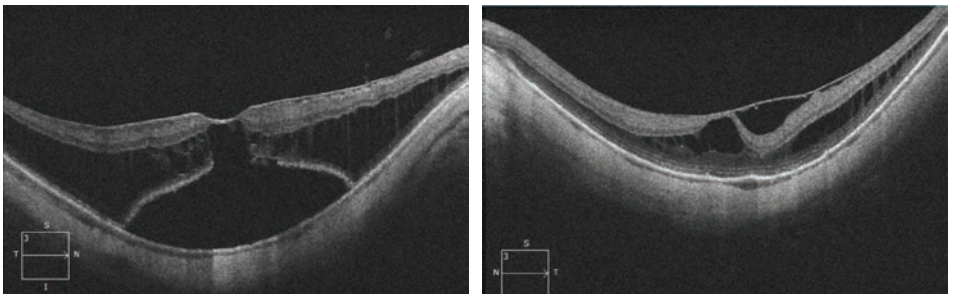

Fig. 2. This set of OCT images were taken one year later.

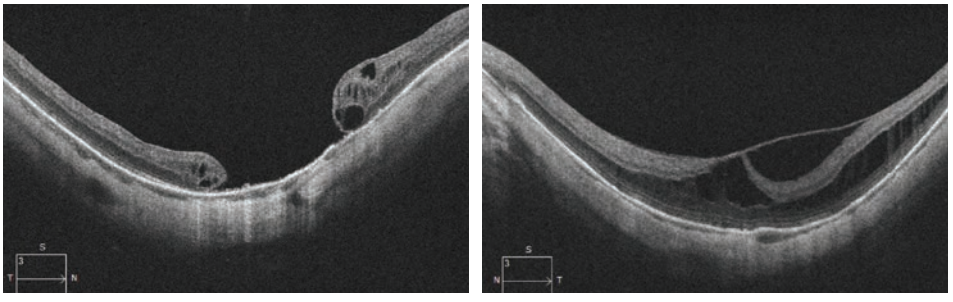

Fig. 3. These OCT images were taken two years later.

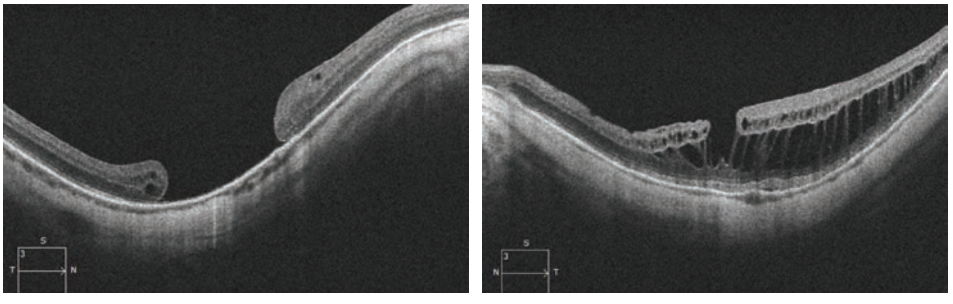

Fig. 4. These images were gathered four years later.

Diagnosis

Even though our patient is pseudophakic, it is clear from her history and clinical presentation that she was highly myopic as a child and has myopic degenerative changes in her posterior pole. It's not surprising the OCTs provide the greatest insight into what's happening with her vision. In both eyes, right worse than left, they clearly show schisis-like cavities within the sensory retina and obvious retinal thickening. On the OCT in the right eye, you can also see a pocket of subretinal fluid posterior to the schisis as well as an obvious epiretinal membrane in the left eye resulting in persistent traction on the macula. This clinical picture is consistent with a diagnosis of myopic traction maculopathy (MTM).

Discussion

This condition develops from a complex interaction of tractional forces on the macula in highly myopic eyes due to an adherent vitreous cortex in the presence of myopic degenerative changes, such as posterior staphyloma.[1] The hallmark of this condition is the presence of a schisis-like thickening in the outer retina. In addition, patients may also have epiretinal membrane, lamellar or full-thickness macular hole (FTMH) and foveal retinal

detachment (FRD).[1-2] It is thought to be present in 9% to 34% of highly myopic eyes.[2]

Beyond the features that are easily visualized on clinical exam with myopic degeneration, such as peripapillary atrophy, lacquer cracks, posterior staphyloma and even epiretinal membrane, the schisis-like cavities are essentially invisible when looking with indirect ophthalmoscopy at the slit lamp with a 90D or 78D lens. It wasn't until 1999 when OCT emerged that we fully recognized the clinical spectrum of this condition.[3]

The natural course of MTM is unclear, especially when paired with a FRD. Some investigators believe that most patients will progress to a FTMH when FRT is present.[2] In eyes that have had vitrectomy after the development of a FTMH, visual outcomes are worse than if a vitrectomy was done before the development of a FTMH.[2] That was the concern with our patient where it was clear on the second OCT that she had progressed to an impending FTMH with significant FRD. Our patient did have a vitrectomy and membrane peel within a week of that visit, and it appeared to be successful, but two weeks after the surgery, she developed a FTMH anyway, which was followed a month later by total retinal detachment. She had a second surgery, which was successful in re-attaching the retina, but the macular hole remained and has since been stable. This is evident on the third OCT where the FTMH can be seen. The OCT shows a complete loss of the sensory retina and photoreceptors in the macula.

Unfortunately, as luck would have it, the left progressed over a two-year period. She noted that her vision in the left eye had declined, and indeed the acuity dropped from 20/30 to 20/80 with an increase in the foveal macular traction. She underwent a vitrectomy and membrane peel in her left eye, which has been successful despite the persistent schisis cavities present on OCT exam. Her acuity returned to 20/30 where it has been stable now for almost two years. The OCT is remarkable in that vitreomacular adhesion and traction that was present before appears to be absent but unfortunately, the schisis is still present and now she has a lamellar hole. The hope is that without persistent macular traction, this will remain stable. She continues to be followed closely. ■

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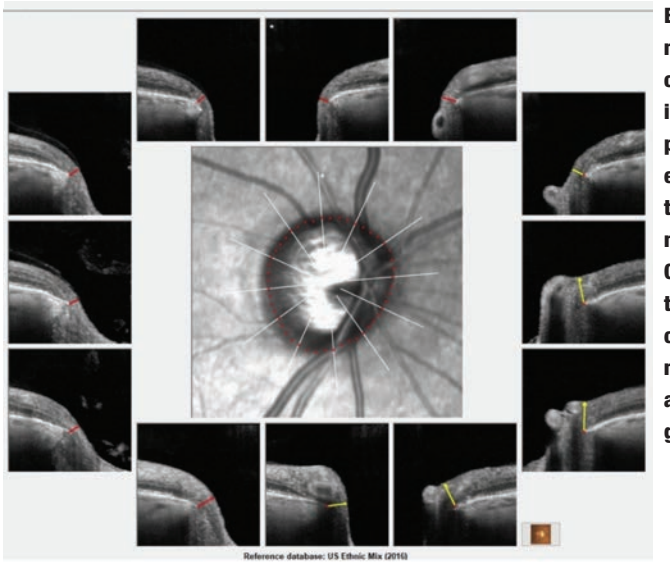
Clean House, But Have a Plan

Hitting the reset button on meds takes a methodical approach. **By James L. Fanelli, OD**

A 62-year-old Caucasian male with a 15-year history of bilateral glaucoma presented as a new patient having just moved to the area. His last visit to his previous eye doctor was approximately nine months earlier, at which time he was apparently told that everything was stable and to make sure he was seen by a glaucoma doctor once he settled.

His chief complaints were chronic and progressively worsening red eyes, the left moreso than the right, which tended to worsen as the day went on. He reported this was a long-term issue and felt as though his symptoms were aggravated after he administered his glaucoma medications. He reported good compliance with his medications.



Bruch's membrane opening overview image of the patient's right eye. Note the thinned neuroretinal rim OD from 6 o'clock to 12 o'clock, consistent with moderately advanced glaucoma.

Examination

At this visit, his glaucoma medications included: brimonidine 0.2% QAM OU, Alphagan P (brimonidine tartrate, Allergan) QD mid-day OU, 1% pilocarpine HS OU and generic latanaprost HS OU. His systemic medications included atorvastatin 20mg QOD, bupropion 100mg QD, gabapentin 300mg BID and Proventil (albuterol, Merck) PRN. He reported no allergies to medications. Significant in his ophthalmic history were two laser treatments to his right eye approximately six years ago and one session to his left eye at about the same time. He reported the laser surgery was for his glaucoma.

He also reported previously trying a series of different glaucoma medications, in an attempt to better control his IOP and minimize the irritation that most of his glaucoma medications caused. Most important was his recollection that he did not tolerate Lumigan (bimatoprost, Allergan) and also had significant issues with dorzolamide on a TID basis, as well as Simbrinza (brinzolamide/brimonidine tartrate, Novartis).

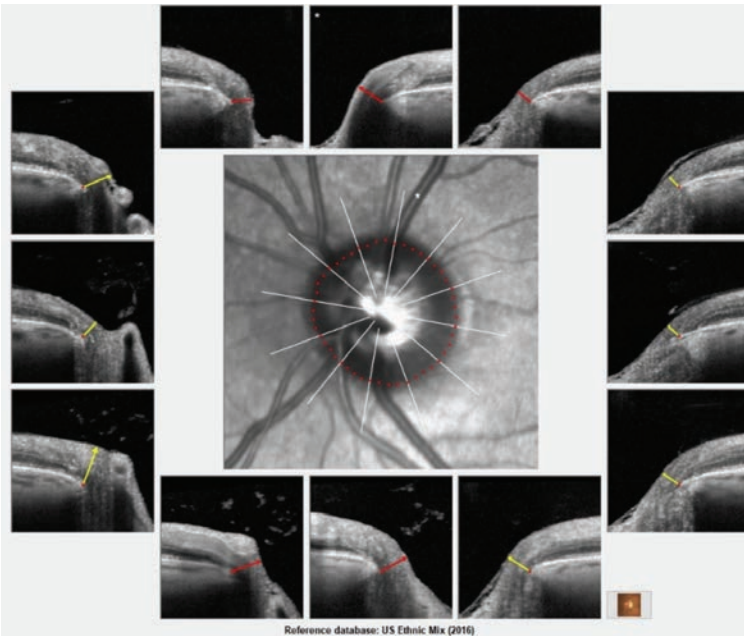
Systemically, he had mild chronic obstructive pulmonary disease (COPD), recurring migraines and hypercholesterolemia. Entering uncorrected visual acuities were 20/20 OD and 20/20 OS. Minimal hyperopic presbyopic refractive error was found upon refraction and was correctable to 20/20 OD, OS, OU.

A slit lamp exam revealed moderate conjunctival and episcleral hyperemia in both eyes. We also noted concurrent mild conjunctival chemosis and fine papillae on the palpebral conjunctiva OU. Angles were wide open on slit lamp estimation. Applanation tensions were 28mm Hg OD and OS at 4:20pm. Pachymetry was 555µm OD and 561µm OS.

The patient was dilated, and his crystalline lenses were clear. Stereoscopic examination of his optic nerves was remarkable for cup-to-disc ratios of 0.8x0.85 OD and 0.65x0.65 OS. The neuroretinal rim temporally of the right eye was quite thin and eroded. The left was also thin, with a well-

perfused remaining rim.

His macular evaluations were essentially unremarkable, with lack of foveal reflexes bilaterally, and fine retinal pigment epithelium granulation consistent with his age. The retinal vasculature was characterized by mild arteriolarsclerotic retinopathy, consistent with his history of hypercholesterolemia. His peripheral retinal exams were unremarkable.



Bruch's membrane opening overview of the patient's left eye demonstrating moderate glaucomatous optic neuropathy.

While he was dilated, we took the opportunity to obtain stereo optic nerve images.

At this point, it was clear that two important items needed attention. First, as the patient relayed in his history, he has been bothered with irritated eyes for several years, which can certainly be exacerbated by the glaucoma medications. Secondly, he has moderately advanced glaucoma OD>OS, and I was not convinced that IOPs of 28mm Hg were an adequate target IOP. So, we determined that we needed to obtain better IOP control and better patient comfort.

Treatment

I didn't immediately replace all his glaucoma medications. That might send the message that I have no faith in his previous doctor, who I don't know, but who the patient has trusted for many years. Also, he presented with a history of having tried many medications and having various reactions to them, so trying something new can be a shot in the dark insofar as selecting one that won't cause problems; it seems as though most do cause him problems. At this stage of the disease, we need to be careful not to let the IOP get further out of control while we hunt for that perfect combination of therapies. And lastly, his eyes are already chronically inflamed and introducing any new is not likely to diminish those symptoms. For these reasons, my management plan in cases such as these is to take things slowly. When only one thing at a time is changed, it's easier to see the effects.

There were a couple of interesting things about his current medication regimen, including the use of pilocarpine as well as two forms of brimonidine.

I chose to have him discontinue the generic brimonidine, increase the Alphagan P to BID, continue the pilocarpine and continue the latanaprost. From a glaucoma medication perspective, the only thing I did was to get him off the higher concentration of brimonidine and substitute that for an additional dose of Alphagan P. But I also chose to help facilitate reduction of his inflammation, and hopefully reduction of his symptoms, by adding a steroid. My go-to steroid in cases like this is fluorometholone because it is a potent anti-inflammatory agent, but one that does not penetrate well into the anterior chamber, reducing the likelihood of an IOP increase. The steroid was prescribed BID for two weeks. The patient was asked to return in a month for threshold field testing, gonioscopy, Heidelberg retinal tomography (HRT 3, Heidelberg) and OCT imaging of both optic nerves and macular ganglion cells.

Follow Up

The patient complied and returned as requested. At this visit, he reported mild reduction in his irritation while on the steroid, but a gradual increase since its cessation. IOPs at this visit were 37mm Hg OD and 33mm Hg OS. We began the slow process of altering glaucoma medications and walking a fine line with topical steroids to reduce inflammation.

Of course, also on the table is surgical intervention. But keep in mind the patient already underwent SLT twice in his right eye and once more for his left. iStent

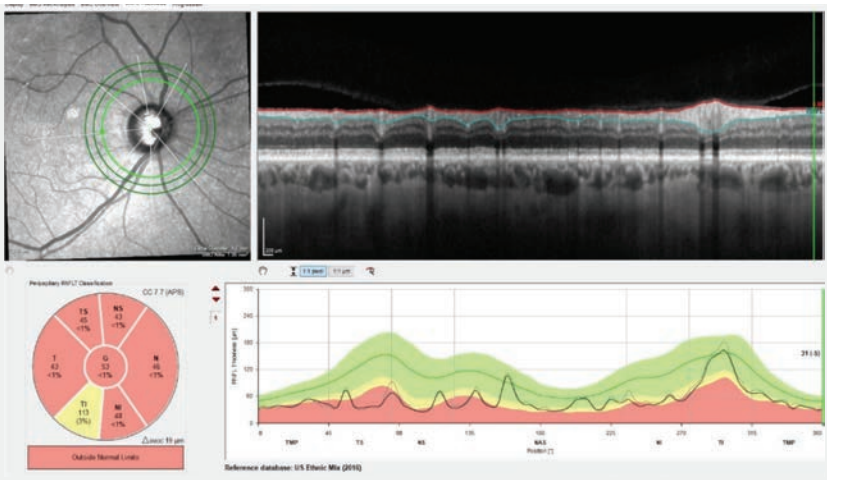
inject (Glaukos) would offer some IOP reduction, but the patient had clear natural lenses, and currently the implantation of iStent inject devices is limited to implantation during cataract surgery. Surgical intervention with tube, shunt or trabeculectomy is also an option, but one I would reserve if absolutely necessary.

At the completion of this visit, I asked him to restart the fluorometholone BID and hold at that dosage until the next visit, decrease the Alphagan P to QD, and add Vyzulta (latanoprostene bunod, Bausch + Lomb) to the regimen. While he was still taking the latanaprost, the Vyzulta gives an additional mechanism of IOP reduction beyond the uveoscleral outflow of other prostaglandins.

My choice in doing this was rather simple; I wanted to see how much of an IOP decrease we would obtain, if any, with the addition of the Vyzulta, knowing full well that his IOPs are now higher than they were before he was put on a steroid. To complicate things further, I was asking him to continue with the steroid for the next few weeks.

Imaging of the nerve and macula correlated to the clinical findings of more advanced disease in the right eye and the visual fields matched the clinical and imaging findings with an arcuate defect noted.

At a subsequent visit, IOPs were 15mm Hg OD and 12mm Hg OS, but he reported that his eyes did not feel much better. While we are making progress on IOP reduction, we are not yet making progress on the discomfort. The addition of the Vyzulta gave us a significant reduction in IOP, which in turn clears the way for further reduction in his other glaucoma medications. Most likely, the biggest contributor to the irritation he's been experiencing is the brimonidine, though the pilocarpine probably also plays a role. Since he was down to using the Alphagan P only once daily, the next logical step was to discontinue it while maintaining all his other medications. He did this and, in a follow up one month later, reported significant improvement in symptoms. His IOPs at this visit were 16mm Hg OD and 14mm Hg OS. We were finally seeing the light at the end of the tunnel.



Three RNFL diameter circle scans of the patient's right eye. Note the stability of the innermost RNFL circle scan of the most recent visit compared with the baseline visit obtained 18 months earlier. Where there is a difference between the scans, the thickness differences overlay major perioptic retinal blood vessels.

As you can imagine, the plan now was to ultimately wean off the steroid, but also with the possibility of eliminating the pilocarpine. So, true to form, I chose to only change one thing at this visit: eliminate the pilocarpine.

Six weeks later, the patient reported continued reduction in irritation. Clinically, the papillae were greatly reduced, as was the episcleral hyperemia OU. IOPs were 16mm Hg OD and 18mm Hg OS. He was only taking the Vyzulta HS OU and the fluorometholone BID OU. The patient was asked to slowly taper off the steroid, using it in both eyes QD for one month then QOD for another month before discontinuing. He was asked to return in four months. He did so and by that visit, he was only taking the Vyzulta. His IOPs were 14mm Hg OD and 15mm Hg OS, and his eyes were clear, white and asymptomatic.

Slow and Steady

When sorting through a case like this, where better IOP control and better ocular comfort are needed, take it slowly. Begin changing things, but do it one step at a time. This way you can see the effects of that one change. By doing so, it makes your treatment path much clearer. While it may take longer to do it this way, it's easier to formulate a plan. Your initial reaction may be to clean house, but sometimes doing that too abruptly leads to confusion as to how to proceed, loss of confidence in you as the doctor and, worst of all, further damage to the patient's eyes. Ultimately, we cleaned house, but we did it in an organized and methodical fashion. ■

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

Out of the Blue

Using trypan blue vital dye in cataract surgery could be the difference between success and failure. **By Christina Tran, BS, and Leonid Skorin, Jr., DO, OD, MS**

Vital dyes are often used in surgical procedures to enhance the visibility of targeted tissues. Some vital dyes used by ophthalmologists include trypan blue, sodium fluorescein, indocyanine green and gentian violet. Of these, trypan blue is the most frequently used in cataract surgery due to its safety, availability and effectiveness.[1] Trypan blue has been used in cataract surgery since the late 1990s to stain the anterior capsule and improve visibility for the surgeon.[1]

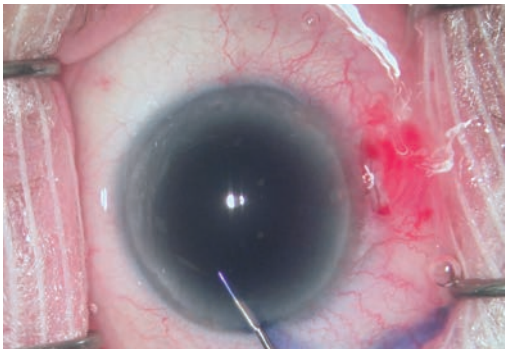


Fig. 1. The surgeon injects the vital dye through the side-port incision to increase visibility during cataract surgery.

Visibility is Vital

The creation of the capsulorhexis in cataract surgery is a delicate procedure that requires careful removal of a circular portion of the anterior lens capsule. This process is far more difficult without visibility of the red reflex and anterior capsule. Poor visibility can increase the risk of radial tears, capsular rupture and intraocular lens (IOL) displacement.[1] Visualization can be facilitated by dimming the operating room lights, using high magnification in the microscope and oblique illumination or applying a high-density viscoelastic such as sodium hyaluronate.[2]

But for mature, traumatic and pediatric cataracts, these techniques are often not enough, and a quick and easy injection of trypan blue is necessary, significantly increasing the success rates of capsulorhexis procedures.[1] This vital dye provides uniform staining of the anterior capsule in 40 to 60 seconds and can help highlight any existing capsular tears.[1] Patients with corneal opacities, abrasions, edema or arcus senilis may also benefit from the use of trypan blue during cataract surgery.[1]

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

Steps for Staining

Various techniques exist for staining the capsule with trypan blue. Surgeons can inject the dye into the anterior chamber under an air bubble or viscoelastic agent or mix trypan blue with a viscoelastic agent and then inject the mixture under an air bubble.[1]

Some surgeons use an even simpler technique: intracamerally injecting trypan blue through the side-port incision following an injection of non-preserved lidocaine (*Figure 1*). The dye remains in the eye for 40 to 60 seconds and is then removed by the injection of a viscoelastic agent into the anterior chamber. As a result, the capsule is stained blue and ready for the capsulorhexis procedure.

Pros and Cons

The application of trypan blue has its advantages, but it's not without the potential for adverse reactions, for which comanaging clinicians should be prepared. Trypan blue has a risk, albeit minimal, of toxicity and spikes in IOP.[1] Rarely, it can cause lens epithelial cell death, which can actually reduce the incidence of posterior capsule opacification.[2] Other studies show that trypan blue can discolor high water content hydrogen IOL implants and inadvertently stain the posterior lens capsule and anterior face of the vitreous.[1] The dye can lead to a decrease in lens capsule elasticity and increase the risk of capsular tears.[3]

Optometrists should note whether a patient had an instillation of trypan blue during surgery and check for any adverse reactions during the early post-op follow-up period. ■

Ms. Tran is a fourth-year student at Pacific University College of Optometry.

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

1. Jhanji V, Chan E, Das S, et al. Trypan blue dye for anterior segment surgeries. Eye (Lond). 2011;25(9):1113-20.
2. Olson RJ, Jin GJC, Ahmed IK, et al. Cataract surgery from routine to complex. Thorofare: Slack; 2011:76.
3. Jacobs DS, Cox TA, Wagoner MD, et al. Capsule staining as an adjunct to cataract surgery. Ophthalmology. 2006;113(4):707-13.

Contact Lenses

New Daily Disposable Keeps Doctors in Charge

Optometrists looking to combat online contact lens sales will soon have an ally with the impending launch of a new daily disposable from a start-up called Eyeris. The hioxifilcon A lens is slated to hit the market in January.

The lens, called Eyeris Dailies, has an 8.5 base curve, a 14.3 diameter and will be available in powers from -13.00D to +6.00D. Optometrists will be able to give their patients access to the lens with the same convenience offered by other online retailers while retaining control over dispensing and pricing, according to the company. If patients choose to order the lens online, the prescribing doctor receives the margin as if it were purchased in their office, the company says.

Diagnostic Devices

Retinal Camera is Delegation-ready

A new offering from Essilor Instruments allows for easy screening and detection of retinal pathologies, the company says. Its Retina 800 non-mydriatic fundus camera is fast to operate, has a space-saving design and provides excellent image quality, a company press release explains. The device's easy to use and intuitive touch-screen interface should allow for no-hassle delegation of fundus photography, saving time and improving patient flow, the company says.

Advanced OCT and Photography in One

The new Maestro2 from Topcon can capture high-resolution non-mydriatic color fundus photos and conventional OCT or OCT angiography scans, the company explains. This multimodal system also now offers the Hood Report for better structure/function analysis of glaucoma, allowing easier comparison of RNFL scans with visual field defects shown on perimetry.

Clinical benefits described in a press release include precise repeatability in follow-up scans for better tracking of disease progress and an extensive portfolio of reports for macular, anterior segment and glaucoma applications. For photography of the peripheral retina, the Maestro2 offers nine preset fields to choose from and the ability to manually control fixation; the device will then create a mosaic image of the fundus. A cataract mode adjusts the scanning position to compensate for opacity, and a 'live fundus' view provides a real-time view of the retina for easier visualization of the optic disc or retinal vessels when determining OCT scan location.

New OCT is as Easy as 1-2-3

The Xephilio OCT-A1 from Canon has a scanning speed of 70,000 scans/sec and a resolution of approximately three microns. An on-board scanning laser ophthalmoscope enables real-time retinal tracking to retain the scan position and protocol for each patient from one exam to the next, eliminating the need for manual adjustment, Canon says.

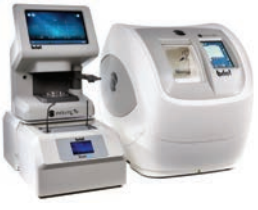
The positioning of the Xephilio's components allows the operator and patient to sit side-by-side. Canon says this can help create a positive and effective user experience. A company press release also highlights the Xephilio's user-friendly interface, explaining that image acquisition is a three-click operation to engage the automated alignment, tracking and acquisition of high-quality images and scans.

Finishing Equipment

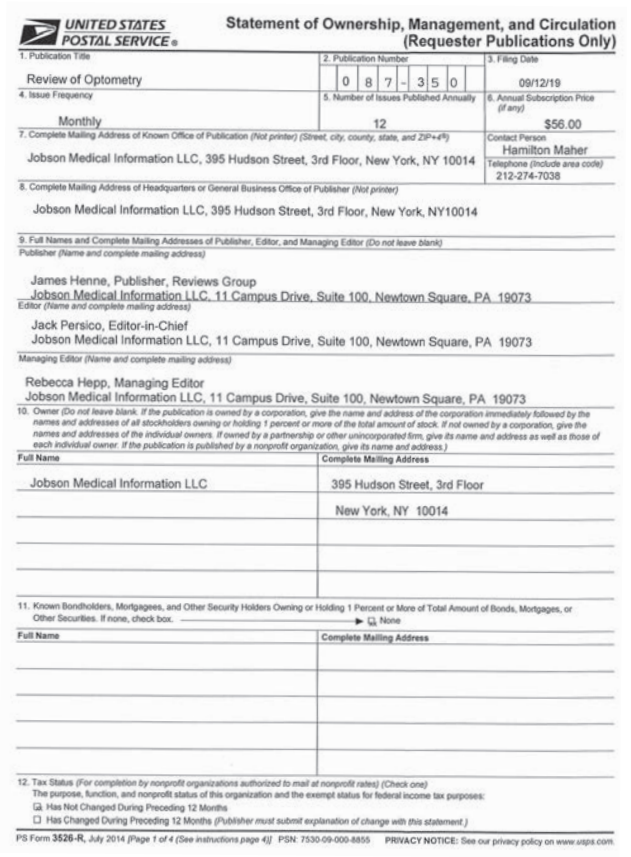
Two Lens Edgers Expand Options

If you have a finishing lab—or want to add one—new options from Briot give you more choices to consider.

The mid-range Evo XS 2 is designed for high-volume in-office labs that may also need a remote tracing option, the company says. The product's advanced tracing capabilities can increase your first-fit rates, according to Briot, while its smooth clamping and soft tracing adapt to any base curve and groove, even high base wrap frames and sport glasses. Its manual blocking process includes parallax-free digital centering and a high-resolution camera system to reveal laser engravings.



Briot's new entry-level edger, Perception 2 Groove, is an all-in-one system developed with new users in mind. Novices might appreciate its high-performance tracing technology, HD camera for parallax-free blocking clarity and better visualization of laser engraving, Briot says. ■



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■ **1-3.** *Music City Fall Classic.* Nashville Marriott at Vanderbilt University, Nashville, TN. Hosted by: Optometric Education Consultants. Key faculty: Greg Caldwell, Joseph Sowka, Jeff Sonsino, Alan Glazier, Michael Depaolis, Joseph Pizzimenti. CE hours: 20. For more information, email optoec@gmail.com, call 954-262-4224 or go to www.optometricedu.com/nashville-2019.

■ **1-3.** *New Technologies & Treatments in Eye Care.* Charleston Marriott, Charleston, SC. Hosted by: REG. Key faculty: Paul Karpecki, Doug Devries, Marc Bloomenstein, Robert P. Woodridge, Jack Schaeffer. CE hours: 19. For more information, go to www.reviewsce.com/charleston2019.

■ **2-3.** *Forum on Primary Eyecare.* Twelve Midtown by Marriott, Atlanta, GA. Hosted by: PSS Eyecare. Key faculty: Ron Melton, Randall Thomas, Jerome Sherman, Robert Rebello, Michael Tolentino. CE hours: 18. For more information, email Sonia Kumari at education@psseyecare.com, call 203-415-3087 or go to psseyecare.com/atlanta-ga.

■ **2-3.** *Maryland Optometric Association Annual Convention.* The Gaylord National Resort and Convention Center, National Harbor, LA. Hosted by: Maryland Optometric Association. Key faculty: Carlo Pelino, Joseph Pizzimenti, Sherrol Reynolds. CE hours: 10. For more information, email Linda Cohen at lindacohen@marylandoptometry.org, call 410-486-9662 or go to www.marylandoptometry.org/page/convention.

■ **3.** *CE-NY: OCT Interpretation in Glaucoma.* Lodge At Welch Allyn, Skaneateles Falls, NY. Hosted by: Continuing Education–New York. Key faculty: Matthew Bovenzi, Mitchell Dul, Anupam Laul. CE Hours: 6. For more information, email Betsy Torres at btorres@sunyopt.edu, call 212-938-5830 or go to www.sunyopt.edu/cpe.

■ **7-11.** *VT2/Learning-related Visual Problems.* Western University, Pomona, CA. Hosted by: Optometric Extension Program Foundation. Key faculty: John Abbondanza. CE hours: 35. For more information, email Karen Ruder at karen.ruder@oep.org, call 410-561-3791 or go to www.oep.org.

■ **8-10**. *ALOA Annual Convention.* Hyatt Regency Ballroom, Birmingham, AL. Hosted by: Alabama Optometric Association. Key faculty: Mile Brujic, Ron Melton, Randall Thomas, Walt Whitley, Mohammad Rafieetary. CE hours: 17. For more information, email Teri Hatfield at teri@alaopt.com, call 334-273-7895 or go to alabama.aoa.org/events/aloa-annual-convention.

■ **8-10**. *Fall Eyecare Conference.* DoubleTree by Hilton Wichita Airport, Wichita, KS. Hosted by: Kansas Optometric Association. CE hours: 13. For more information, email Rachelle Heatwole at rachelle@kansasoptometric.org, call 785-232-0225 or go to www.kansasoptometric.org.

■ **8-10.** *NCOS Fall Congress.* The Westin Charlotte, Charlotte, NC. Hosted by: North Carolina Optometric Society. Key faculty: Bruce Onofrey, Jennifer Lyerly, Nathan Lighthizer, Ron Melton, Randall Thomas. CE hours: 18. For more information, email Christy Santacana at christy@nceyes.org, call 919-977-6964 or go to www.nceyes.org/fall-congress.

■ **9-10.** *CE in Fort Worth.* Dallas Fort Worth Marriott Hotel & Golf Club, Fort Worth, TX. Hosted by: Univeristy of Houston College of Optometry. Key faculty: Marcus Gonzales, Jeffry Gerson, Andrew Kemp, Vladislav Koyfman, Zanna Kruoch, Nikolaos Zagorianos. CE hours: 16. For more information, email UNCHO Continuing Education at optce@central.uh.edu, call 713-743-1900 or go to ce.opt.uh.edu/live/2019/ce-in-fort-worth.

■ **10.** *Optometric Education.* Salus University, Elkins Park, PA. Hosted by: Salus University. Key faculty: Alan Kabat, Tracy Offerdahl, G. Richard Bennett, Josh Kim, Erin Draper, Ashley Maglione, Mark Street. CE hours: 8. For more information, email Natalie Standig at nstandig@salus.edu, call 215-780-1381 or go to www.salus.edu/events.

■ **15-17.** *Monterey Symposium.* Monterey Conference Center, Monterey, CA. Hosted by: California Optometric Association. Key faculty: Alan Kabat, Leonard Messner, Mohammand Rafieetary, James Thimons, Mark Wright, Michael DePaolis, Justin Bazan, W. Lee Ball, Ryan McKinnis, Steven F. Rosinski, Scott Schachter. CE hours: 35. For more information, email Brenda Stewart at brends@coavision.org, call 916-266-5035 or go to www.coavision.org/i4a/pages/index.cfm?pageid=3288.

■ **15-17.** *AZOA Fall Congress.* Hilton Sedona Resort, Sedona, AZ. Hosted by: Arizona Optometric Association. Key faculty: Bryan M. Rogoff, Greg Caldwell, Joshua Duncan, Jay Haynie. CE hours: 14. For more information, email Kate Diedrickson at kate@azoa.org, call 602-279-0055 or go to www.azoa.org/connect.

■ **16-17.** *Orlando Super Weekend.* NSU Orlando Campus, Orlando, FL. Hosted by: Nova Southeastern University. Key faculty: Mark Dunbar, Chandra Mickles, Jessica Steen, Yin Tea. CE hours: 11. For more information, email Vanessa McDonald at oceaa@nova.edi, call 954-262-4224 or go to optometry.nova.edu/ce/index.html.

■ **23-24.** *Everything Therapeutic: San Antonio.* Westin Riverwalk Hotel, San Antonio, TX. Hosted by: Northern Rockies Optometric Conference. Key faculty: William Townsend, Joseph Sowka, David Sendrowski, Robert Prouty. CE hours: 16. For more information, email UNCHO Continuing Education at optce@central.uh.edu, call 713-743-1900 or go to ce.opt.uh.edu/live/2019/everything-therapeutic-san-antonio.

December 2019

■ **5-8.** *Optometric Management Symposium*. Disney's Yacht & Beach Club, Orlando, FL. Hosted by: PentaVision Media, *Optometric Management*. Key faculty: Greg Caldwell, April Jasper, Joseph Sowka. CE hours: 45 total, 25 per OD. For more information, email Maureen Trusky at maureen.trusky@pentavisionmedia.com or go to www.omconference.com.

■ **6-7.** *Annual Tulsa Winter Weekend*. Renaissance Tulsa, Tulsa, OK. Hosted by: Oklahoma College of Optometry. Key faculty: Nathan Lighthizer, Blair Lonsberry. CE hours: 19. For more information, email Callie McAtee at mcateec@nsuok.edu, call 918-316-3602 or go to optometry.nsuok.edu/Continuing-Education/Schedule-of-Events/5th-Annual-Tulsa-Winter-Weekend.

■ **6-7.** *Retina Update*. Fairmont Scottsdale Princess, Scottsdale, AZ. Hosted by: Optometric Retina Society and REG. Key faculty: Mohammad Rafieetary, Steven Ferrucci. CE hours: 12. For more information, go to www.reviewsce.com.

■ **7-8.** *Cornea, Contact Lens & Contemporary Vision Care Symposium*. The Westin Hotel, Memorial City, Houston, TX. Hosted by: University of Houston College of Optometry. Key faculty: Jan P.G. Bergmanson. CE hours: 16. For more information, email UHCO Continuing Education at optce@central.uh.edu, call 713-743-1900 or go to ce.opt.uh.edu/live.

■ **7-8.** *Malinovsky Ocular Disease Weekend*. Rawles Hall, Bloomington, IN. Hosted by: Indiana University School of Optometry. CE hours: 16. For more information, email Cheryl Oldfield at coldfiel@indiana.edu, call 812-856-3502 or go to expand.iu.edu/browse/iuso-ce.

■ **8**. *Contemporary Topics in Optometry*. MBKU Hopping Academic Center, Fullerton, CA. Hosted by: Marshall B. Ketchum University Southern California College of Optometry. Key faculty: Vin Dang, Rachelle Lin, Tomi Luan, Patrick Yoshinaga, Lisa Wahl, Judy Tong. CE hours: 8. For more information, email Bonnie Dellatorre and Antoinette Smith at ce@ketchum.edu, call 714-449-7495 or go to ketchum.edu/ce.

■ **13-14.** *West Coast Optometric Glaucoma Symposium*. Hyatt Regency Huntington Beach, Huntington Beach, CA. Hosted by: REG. Key faculty: Murray Fingeret, Robert N. Weinreb. CE hours: 12. For more information, go to www.reviewsce.com/wcogs2019.

■ **22-29.** *Modern Management of Ocular Disease Cruise*. Royal Carribean's Allure of the Seas, round trip from Fort Lauderdale, FL. Hosted by: Dr. Travel Seminars. Key faculty: Edward L. Paul, Jr. CE hours: 16. For more information, email at info@drtravel.com, call 800-436-1028 or go to www.drtravel.com.

To list your meeting, please send the details to:

Mark De Leon, Associate Editor

Email: mdeleon@jobson.com

Phone: (610) 492-1021

January 2020

■ **10-12.** *AZOA Bronstein Contact Lens & Cornea Seminar*. Hilton Scottsdale Resort & Villas, Scottsdale, AZ. Hosted by: Arizona Optometric Association. Key faculty: Stephen P. Bynes, Blair Lonsberry, Thomas G. Quinn, Jeffrey J. Walline. CE hours: 16. For more information, email Kate Diedrickson at kate@azoa.org, call 602-279-0055 or go to www.azoa.org/connect.

■ **10-12.** *Southwest Congress of Optometry*. Drury Inn and Suites Riverwalk, San Antonio, TX. Hosted by: Optometric Extension Program Foundation. Key faculty: David Cook, Earl Smith, Janice Wensveen. For more information, email Fred Brecheen at drb90@verizon.net, call 972-221-2564 or go to www.oepf.org/calendar.

■ **11.** *Glaucoma Symposium*. Willows Lodge, Woodinville, WA. Hosted by: Pacific University College of Optometry. Key faculty: Murray Fingeret, Howard Barnebey. CE hours: 19. For more information, email Michelena "Miki" Buckingham at mikibuckingham@pacificu.edu, call 503-352-2985 go to www.pacificu.edu/academics/colleges/college-optometry/continuing-education.

■ **18-20.** *Kaskin Invitational Skeffington Symposium*. Embassy Suites Chevy Chase Pavilion, Washington, DC. Hosted by: Optometric Extension Program Foundation. CE hours: 19. For more information, email Jeffrey Kraskin at jlkraskin@rcn.com, call 202-363-4450 or go to www.skeffingtonsymposium.org.

■ **19-25.** *Island Eyes Conference*. Hyatt Regency Maui Resort and Spa, Maui, HI. Hosted by: Pacific University College of Optometry. Key faculty: Thomas Quinn, Christopher Quinn, Susan Cotter, Jay Haynie, William Hefner. CE hours: 30. For more information, email Michelena "Miki" Buckingham at mikibuckingham@pacificu.edu, call 503-352-2985 go to www.pacificu.edu/academics/colleges/college-optometry/continuing-education.

■ **22-25.** *Global Specialty Lens Symposium*. Tropicana Las Vegas, Las Vegas, NV. Hosted by: *Contact Lens Spectrum*, PentaVision Media. Key faculty: Jason J. Nichols, Edward Bennett, Patrick Caroline, Karen DeLoss, Eef van der Worp. CE hours: 57 total, 20 per OD. For more information, email Maureen Trusky at maureen.trusky@pentavisionmedia.com, call 215-514-3729 or go to www.gslsymposium.com.

■ **22-26.** *VT1/Visual Dysfunctions*. 2940 N. Dobson, Suite 11, Chandler, AZ. Hosted by: Optometric Extension Program Foundation. Key faculty: Robin Lewis. CE hours: 35. For more information, email Karen Ruder at karen.ruder@oep.org, call 410-561-3791 or go to www.oep.org.

■ **29-Feb. 2.** *VT1/Visual Dysfunctions in Athens, Greece*. Hosted by: Optometric Extension Program Foundation. Key faculty: Robin Lewis. CE hours: 35. For more information, email Karen Ruder at karen.ruder@oep.org, call 410-561-3791 or go to www.oep.org.

Diagnostic **Quiz**

Put a Red Eye Back in the Pink

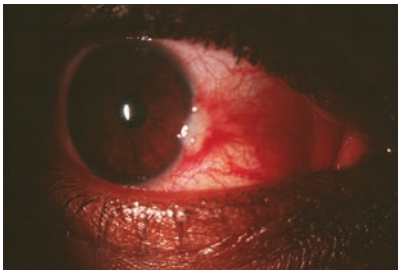
By Andrew S. Gurwood, OD

History

A 42-year-old black male reported to the office with a chief complaint of red, irritated eyes that was present in both eyes, but worse in his left than his right. He explained that the redness had been progressively getting worse and was only temporarily relieved by an over-the-counter anti-allergy drop. His systemic and ocular histories were unremarkable and he denied exposure to chemicals or allergies of any kind.

Diagnostic Data

His best-corrected entering visual acuities were 20/20 OU at distance and near. His external examination was normal with no evidence of afferent pupillary defect. The pertinent anterior segment findings are demonstrated in the photograph. Goldmann applanation tonometry measured 15mm Hg OU. Dilated funduscopy was within normal limits in both eyes, revealing slightly asymmetric cup-to-disc ratios measuring 0.4/0.4 OD and 0.4/0.55 OS with normal peripheries.



This patient's red eye has been getting progressively worse. Can anything about this image and the diagnostic data help explain why?

Your Diagnosis

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

Additional Questions

When a patient with this kind of presentation is in your office, be sure to ask the right questions. You'll want to ask if they experience:

- Pain upon eye movement (which may suggest episcleral or scleral inflammation, while minimal pain suggests superficial inflammation)
- Any indications of severe ocular pain
- Photophobia
- Any history with or previous diagnosis of tuberculosis

Retina Quiz Answers *(from page 107):* 1) b; 2) c; 3) a; 4) c.

Next Month in the Mag

Coming in November, *Review of Optometry* will present an issue focused on promoting eyelid health.

Topics include:

- *The Intersection of Eyelids, Allergy and Dry Eye*
- *In-office and At-Home Interventions for Lid Disease*
- *The Ugly Truth about Cosmetics and Eyelid Disease*
- *A Primer for Diagnosing Lid Lesions*

Also in this issue:

- *Recognizing Secondary Glaucomas* (Earn 2 CE Credits)
- Winners of Review of Optometry's *Biennial Office Design Contest*

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References: 1. Alcon data on file, 2019. **2.** Kern JR, Kappell G, Trinh H, et al. Antimicrobial properties of a novel contact lens disinfecting solution, OPTI-FREE® EverMoist®. *Cont Lens Anterior Eye*. 2011;34(suppl 1):S30. **3.** Alcon data on file, 2010. **4.** Alcon data on file, 2011.