Pigmentary Glaucoma Revisited, *p. 96* • With Disc Edema, Act Fast, *p. 100*

REVIEW® OF OPTOMETRY

March 15, 2020

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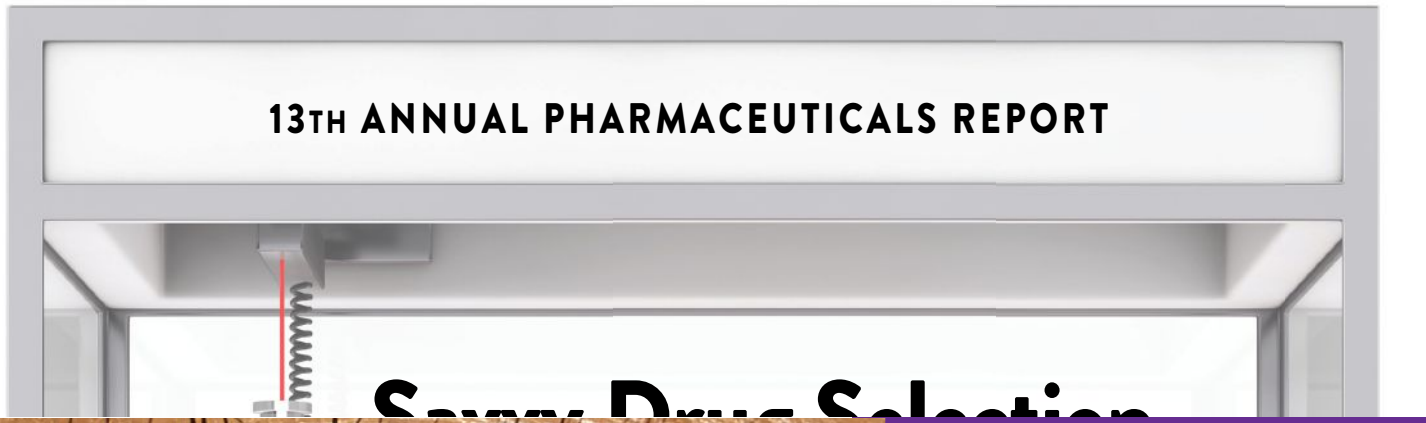

Savvy Drug Selection

References: 1. Korb D, Blackie C, Meadows D, Christensen M, Tudor M. Evaluation of extended tear stability by two emulsion based artificial tears. Poster presented at: 6th International Conference of the Tear Film and Ocular Surface: Basic Science and Clinical Relevance; September 22-25, 2010; Florence, Italy. **2.** Moon SW, Hwang JH, Chung SH, Nam KH. The impact of artificial tears containing hydroxypropyl guar on mucous layer. *Cornea*. 2010;29(12):1430-1435. **3.** Davitt WF, Bloomenstein M, Christensen M, Martin AE. Efficacy in patients with dry eye after treatment with a new lubricant eye drop formulation. *J Ocul Pharmacol Ther*. 2010;26(4):347-353. **4.** Willcox MDP, Argueso P, Georgiev GA, et al. TFOS DEWS II tear film report. *Ocul Surf*. 2017;15:366-403. **5.** Ketelson H, Rangarajan R. Pre-clinical evaluation of a novel phospholipid nanoemulsion based lubricant eye drop. Invest Ophthalmol Vis Sci. 2017;58:3929. **6.** Ogundele A, Ketelson H, et al. Preclinical evaluation of a novel hydroxypropyl-guar phospholipid nanoemulsion lubricant eye drop for dry eye disease. Poster presented at: The 36th World Ophthalmology Congress (WOC); June 16-19, 2018; Barcelona, Spain. **7.** Craig J, Nichols K, Akpek E, et al. TFOS DEWS II definition and classification report. *Ocul Surf*. 2017;15:276-283. **8.** Lane S, Paugh J, et al. An Evaluation of the in vivo Retention Time of a Novel Artificial Tear as Compared to a Placebo Control. Invest Ophthalmol Vis Sci. 2009;50(13):4679. **9.** Benelli U. Systane® lubricant eye drops in the management of ocular dryness. *Clin Ophthalmol*. 2011;5:783-790. **10.** Torkildsen G. The effects of lubricant eye drops on visual function as measured by the Inter-blink interval Visual Acuity Decay test. *Clin Ophthalmol*. 2009;3:501-506.

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Pigmentary Glaucoma Revisited, *p. 96* • With Disc Edema, Act Fast, *p. 100*

REVIEW® OF OPTOMETRY

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VOL. 157 NO. 3 ■ MARCH 15, 2020

IN THE NEWS

Researchers recently assessed SD-OCT RNFL thickness measurements from 684 patients, 101 of whom were glaucoma suspects and found that **37% had partial posterior vitreous detachment (PVD)**, which they associated with **greater retinal nerve fiber layer (RNFL) thickness**. Among the glaucoma suspects, they noted that average RNFL thickness was greater in eyes with partial PVD compared with unaffected subjects.

Liu Y, Baniasadi N, Ratanawongphaibul K, et al. Effect of partial posterior vitreous detachment on spectral-domain optical coherence tomography retinal nerve fiber layer thickness measurements. Br J Ophthalmol. February 12, 2020. [Epub ahead of print].

Topical therapy may be an effective treatment with high closure rates in patients with secondary full-thickness macular holes, a small study of 12 patients suggests. Nine eyes received topical difluprednate with the addition of a topical carbonic anhydrase inhibitor or non-steroidal anti-inflammatory drops. Eight eyes (89%) achieved successful hole closure.

Niffenegger JH, Fong DS, Wong KL, Modjtahedi BS. Treatment of secondary full-thickness macular holes with topical therapy. Ophthalmol Retina. January 28, 2020 [Epub ahead of print].

A new study found **multifocal choroiditis and punctate inner choroidopathy are two distinct clinical conditions**. After five years of follow-up, only one of 343 eyes changed diagnosis due to newly developed peripheral lesions. Analysis prioritized criteria of chorioretinal lesion location and intraocular inflammation and identified two phenotype clusters. The study also found distinct characteristics between the groups, including the development of choroidal neovascularization and different treatment approaches.

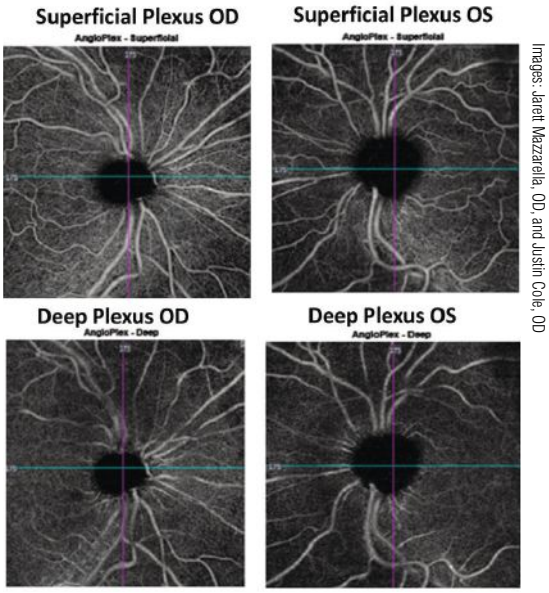
Gilbert RM, Niederer RL, Kramer M, et al. Differentiating multifocal choroiditis and punctate inner choroidopathy: a cluster analysis approach. Am J Ophthalmol. February 3, 2020. [Epub ahead of print].

En Face OCT Influences Treatment Decisions

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

By Bill Kekevian, Senior Editor

Unlike standard OCT, *en face* OCT employs software to construct an image cube of the posterior pole. This produces a transverse image of the retina and choroid at any specified depth—essentially cutting through layers and providing an extensive overview of pathological structures in a single image. For glaucoma suspects, this means clinicians can better observe the structures where glaucoma is first evident. Researchers are now showing that the use of *en face* images seems to influence clinicians' treatment choices.



Images: Jarett Mazzarella, OD, and Justin Cole, OD

***En face* OCT imaging helped uncover clinical signs of pre-perimetric, unilateral pigmentary glaucoma. The superficial and deep capillary plexus segmentation of the OD and OS show significant asymmetry in the peripapillary capillary densities OD>OS.**

The research looked at 30 patients who were examined in three ways: a standard presentation of circumpapillary retinal nerve fiber layer (RNFL) scans, 24-2 perimetry results and *en face* imaging, including RNFL depth scans, custom segmentation of the RNFL and a custom normalized *en face* reflectance probability map.

The researchers asked clinicians to review the images and assess whether glaucoma was likely present using a five-point scale (strongly disagree, disagree, neutral, agree, strongly agree) and to recommend a management plan (start treatment, return in three months, return in six months, recommended yearly OCT and 24-2, discharge as the patient is deemed to be low risk) for the first two presentations. After the *en face* presentation, the initial two questions were asked along with a third one on whether the *en face* image

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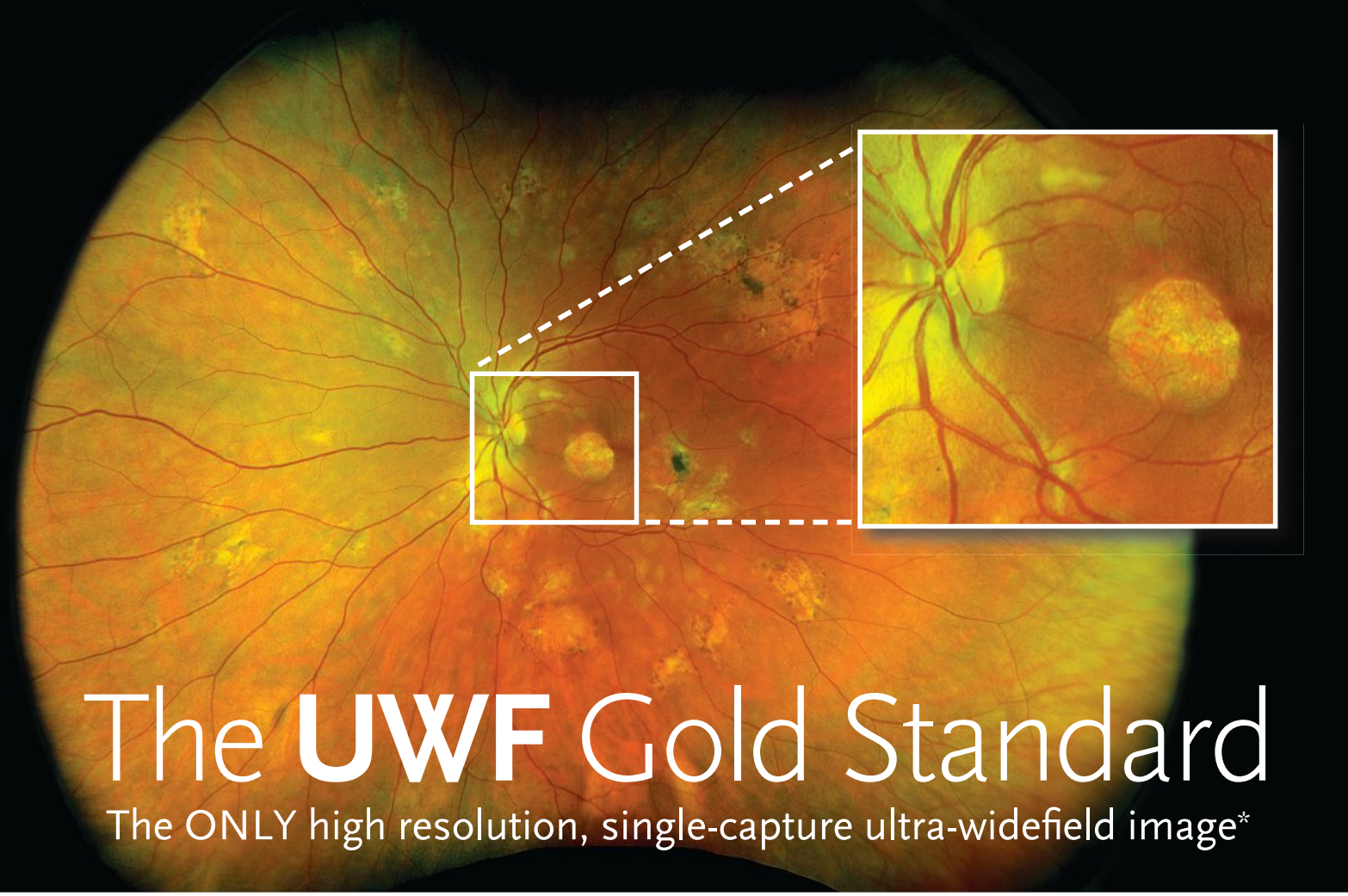

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*Classification & Guidelines for Widefield Imaging Recommendations from the International Widefield Imaging Study Group. Ophthalmology Retina. 2019.

Vermont Rejects Scope Expansion

ODs sought to include some laser procedures, injections and lesion treatment.

Vermont's hope to expand its scope of practice has been dashed by a decision of the state's Office of Professional Regulation (OPR). The office concluded in its final report that optometrists are not properly trained in, nor can they safely perform, the proposed advanced procedures.[1]

The Vermont Optometric Association (VOA) asked the state to consider adding anterior segment laser procedures, injections of the lids and adnexa, and removal of benign lesions. These can include nd:YAG laser capsulotomies, laser trabeculoplasties, laser iridotomy, lid and subconjunctival space injections, as well as intramuscular, subcutaneous and intravenous injections. Removal of benign growths such as pedunculated lesions, papilloma, keratosis, cutaneous cysts and others were included.

The VOA's appeal explained that the "difficult part" of these procedures is pre- and post-op management, which optometrists handle under the current scope of practice.

Vermont's report bemoans an unfulfilled request for information about each school's course offerings, curricula or syllabi from the Association of Schools and Colleges of Optometry (ASCO)—all of which are publicly available, according to ASCO President Elizabeth Hoppe, OD.

The state's report explains that the Vermont Ophthalmologist Society's and the Vermont Medical Society's adamant opposition influenced its decision. As evidence, they pointed to a *JAMA Ophthalmology* 2016 report that showed repeat laser trabeculoplasty procedures nearly double when the initial procedure is performed by an OD rather than an MD.

This research is often cited by medical lobbyists contesting optometric scope expansion, but optometrists charge that it is misleading.[2]

The study looked into the performance of 27 optometrists trained at Northwestern University, a program that recommends performing the 180˚ procedure first and only considering treatment of the other half if IOP doesn't sufficiently decrease.[3] The study said nothing in terms of pressure reduction or complications associated with the full 360˚ procedure, instead framing a second procedure as a "risk."[3]

A rebuttal letter, printed in the same journal, was also delivered to the Vermont legislature.[3] That piece calls the study misleading and explains the inconsistencies regarding the type of treatment under discussion.

The state's report shows the OPR was not convinced that expanding optometry's scope would give patients better access since data suggests there is an ophthalmologist within 30 miles of most residents—and ODs are located near MDs anyway.

1. Layman L, Hibbert S. Vermont Secretary of State Office of Professional Regulation. Study of optometric advanced procedures. legislature.vermont.gov/assets/Legislative-Reports/Optometry-Report-FINAL-2020.pdf. January 15, 2020. Accessed February 3, 2020.
2. Stein J, Zhao P, Andrews C. Comparison of outcomes of laser trabeculoplasty performed by optometrists vs ophthalmologists in Oklahoma. JAMA Ophthalmol. 2016;134(10):1095-1101.
3. Fingeret M. Laser trabeculoplasty use patterns among optometrists and ophthalmologists in Oklahoma. JAMA Ophthalmol. 2016;134(10):1101-2.

En Face OCT Impacts Glaucoma Treatment

Contiued from page 4

was helpful for each of the three formats.

The study shows that, when asked whether the participant illustrates glaucomatous optic neuropathy after viewing the second standard presentation, "agree" or "strongly agree" was selected 29% of the time. When given the third presentation with *en face* reflectance, clinicians changed their ranking 59% of the time (46% toward likely to have glaucoma and 13% toward unlikely to have glaucoma). This shows the addition of *en face* imaging seems to moderately influence clinical decision making in the direction toward selecting "agree" on whether the subject had glaucomatous optic neuropathy and to start treatment, the researchers suggested in their paper.

Clinicians may be more responsive to data from imaging when there is a probability or a value provided rather than strictly visualizing potential defects as in the scroll-through scan, the researchers explained in their paper. By comparison, minimal changes were seen when comparing the first standard setting with the second.

King B, Swanson W, Klemencic S, et al. Assessing the impact of en face retinal nerve fiber layer imaging on clinical decision making for glaucoma suspects. Optom Vis Sci. 2020;97(2):54-61.

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Concerns Surface for New Anti-VEGF

A new anti-VEGF injection, Beovu (brolucizumab-dbll, Novartis) gained FDA approval in October 7, 2019, expanding the portfolio of options for wet AMD. Doctors have been eager to give it a try, with an estimated 46,000 injections administered in the United States as of mid-February, Novartis noted in a statement.

However, on February 23, the American Society of Retina Specialists (ASRS) shared a warning to its members: reports to the society note an increased risk of inflammation among patients who received Beovu. ASRS notes that, since the drug's approval, it has received 14 reports of vasculitis, 11 of which were occlusive retinal vasculitis.

Although alarming, 14 cases out of the estimated 46,000 injections "places these events at a relatively low risk ratio," explains Mohammad Rafieetary, OD, who is a consultative optometrist at the Charles Retina Institute in Germantown, Tenn. "It has been noted in this report that the exact cause of these adverse events are unclear."

Both ASRS and Novartis statements remind doctors that Beovu is contraindicated for patients with active intraocular inflammation.

"Physicians should follow the guidance in the prescribing information that patients with active inflammation should not be injected with Beovu," Novartis said.

The HAWK/HARRIER Phase 3 trials that led to the medication's approval showed that both 3mg and 6mg regimens of brolucizumab were noninferior to aflibercept in best-corrected visual acuity change from baseline at 48 weeks. The trials also revealed a 4% rate of intraocular inflammation and a 1% rate of retinal artery occlusion—incidences that are not far from those reported by other anti-VEGF agents, Dr. Rafieetary says.[1]

"Patient safety is of paramount importance," the company said in the statement. "Novartis stands behind the safety and efficacy of Beovu. In addition to our own internal assessment, we have engaged an external safety review committee to further evaluate these post-marketing cases. We will continue to share details as they become available."

"From a personal experience standpoint, Beovu is very effective agent for neovascular AMD, including for cases becoming recalcitrant to previous treatment with existing anti-VEGF agents," notes Dr. Rafieetary, who is a masked subinvestigator for Novartis's HAWK, MERLIN, KINGFISHER and RAVEN clinical trials. "Beovu or any other therapeutic agents will always come with potential risks. It is incumbent on all practitioners to consider these risks and fully inform the patient of them compared with the benefits and discuss possible alternatives of any treatment and obtain an informed consent for indicated procedural cases."

Doctors who notice Beovu-associated inflammation should follow the patient closely with appropriate imaging, the ASRS said in the warning, as some cases can be subtle or have delayed onset.

Doctors should also report any adverse events to Novartis at 1-888-NOW-NOVA (1-888-669-6682) or the FDA via MedWatch.

Dugel PU, Koh, Ogura Y, et al. HAWK and HARRIER: Phase 3, multicenter, randomized, double-masked trials of brolucizumab for neovascular age-related macular degeneration. Ophthalmology. 2020;127(1):72-84.

Some Wet AMD Patients Retain VA Gain After Treatment

A new study found some patients with wet AMD may be able to retain good vision years after stopping treatment. The investigation found 6.4% of participants retained a visual acuity letter score of 68 (Snellen 20/40) or better after stopping anti-VEGF for at least three years.

A team of US researchers analyzed the results of the landmark CATT trial that made headlines a decade ago for validating the comparable efficacies of bevacizumab and ranibizumab. CATT studied injection patterns and visual acuity trends among 635 eyes with wet AMD randomized into one of four treatment groups: ranibizumab monthly, bevacizumab monthly, ranibizumab as-needed or bevacizumab as-needed. At one year, participants in the monthly groups continued treatment or were switched to as-needed. At year two, participants stopped treatment at the discretion of their ophthalmologist and were followed up again at five years.

The current *post-hoc* analysis compared the eyes of 40 participants who stopped treatment and still had good visual acuity with the remaining 585 eyes from the CATT follow-up study.

Baseline characteristics were similar between the groups, except for a better VA letter score in the study eye (68.8 vs. 61.8) and the fellow eye (78.4 vs. 68) in addition to more blocked fluorescence in the patients who stopped treatment (27.5% and 13.8%, respectively).

The cessation group received fewer injections in year one (5.8 vs. 8.1) and year two (7.7 vs. 13.8) compared with eyes in the as-needed group. The approximate VA letter score at five years was 79 and 57.5 in the cessation and as-needed groups.

These findings suggest that a small proportion of eyes with wet AMD can retain good visual acuity with no treatment for at least three years after two years of treatment, the study noted.

Scoles D, Ying GS, Pan W, et al. Characteristics of eyes with good visual acuity at five years after initiation of treatment for age-related macular degeneration but not receiving treatment from years three to five: Post hoc analysis of the CATT randomized clinical trial. JAMA Ophthalmol. Jan. 30, 2020 [Epub ahead of print].

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References:

1. Xiidra [prescribing information]. East Hanover, NJ: Novartis Pharmaceuticals Corporation; November 2019. **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.

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In clinical trials, 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.

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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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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Skipping Doctor Visits Can Worsen AMD

A new study in *JAMA Ophthalmology* corroborates a point any clinician could tell you: patients who don't seek care fare worse than those who do. Whether or not your patients turn up for their appointments can play a significant role in their ultimate visual outcome, the research shows. The authors advocate for substantial effort to reduce any burden associated with seeing an eye care professional to avoid vision loss.

A research team evaluated the records of 1,178 AMD patients who were originally examined for a clinical trial. These patients were scheduled to return to their doctor once every four weeks. The group's mean number of missed visits was 2.4. The researchers classified the patients as on time (visits every 28 to 35 days), late (every 36 to 60 days) and very late (more than 60 days between visits).

Overall, patients were relatively adherent to their follow-up schedule, with 92.6% achieving complete visit constancy. The investigators compared data between the patients' baselines and last doctor visits and found those with a less-than-stellar record experienced worse visual outcomes. After controlling for covariates, the late and very late groups saw fewer letters than patients in the on-time group.

The researchers found that after only six months, each missed visit was associated with an average visual acuity letter score decline of 0.7. Compared with patients who were on time, those who averaged between 36 to 60 days and more than 60 days between visits lost 6.1 and 12.5 letters, respectively.

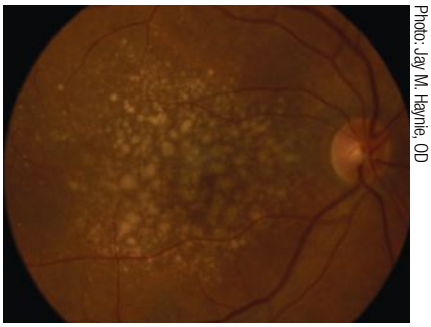


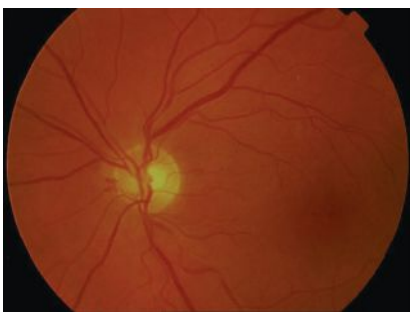
Photo: Jay M. Haynie, OD

Patients with early signs of AMD would do well to adhere to their follow-up schedule to avoid visual decline.

Ramakrishnan M, Yu Y, VanderBeek B. Association of visit adherence and visual acuity in patients with neovascular age-related macular degeneration. JAMA Ophthalmol. February 6, 2020.

Bariatric Surgery Exacerbates PDR

The problems of obesity and diabetes have increased worldwide, and bariatric surgery is becoming an increasingly acceptable management plan. Some recognize the procedure as an elective treatment that facilitates substantial sustained weight loss and induces drastic and rapid glycemic control that consequently results in the remission of type 2 diabetes. But new research suggests it can have negative effects as well. Researchers in Turkey have found that patients with proliferative diabetic retinopathy (PDR) who received bariatric surgery showed more severe retinopathy than patients who did not receive the surgery but were otherwise matched for age, sex, HbA1c levels and follow-up duration.



Clinicians should watch patients undergoing bariatric surgery carefully, as the proceudre may worsen PDR.

The retrospective observational study included 37 eyes of 21 patients with PDR who underwent bariatric surgery. The control group comprised of 37 eyes of 27 patients with PDR who attended the same research hospital for diabetes care without undergoing the surgery.

The first-year HbA1c levels of the bariatric surgery group were significantly lower than those of the control group. However, the surgery patients had significantly higher intraocular hemorrhage, neovascular glaucoma and retinal vein occlusion rates than the control patients. The researchers found that 80.9% of this group had one of these serious eye complications.

The study emphasizes that its results provide evidence that the severity of DR at baseline is an important sign of post-bariatric surgery DR grade. The researchers suggest that continued DR monitoring post-op is particularly important for patients with PDR.

Sever O, Horozoglu F. Bariatric surgery might aggravate proliferative diabetic retinopathy. Acta Ophthalmol. January 7, 2020. [Epub ahead of print].

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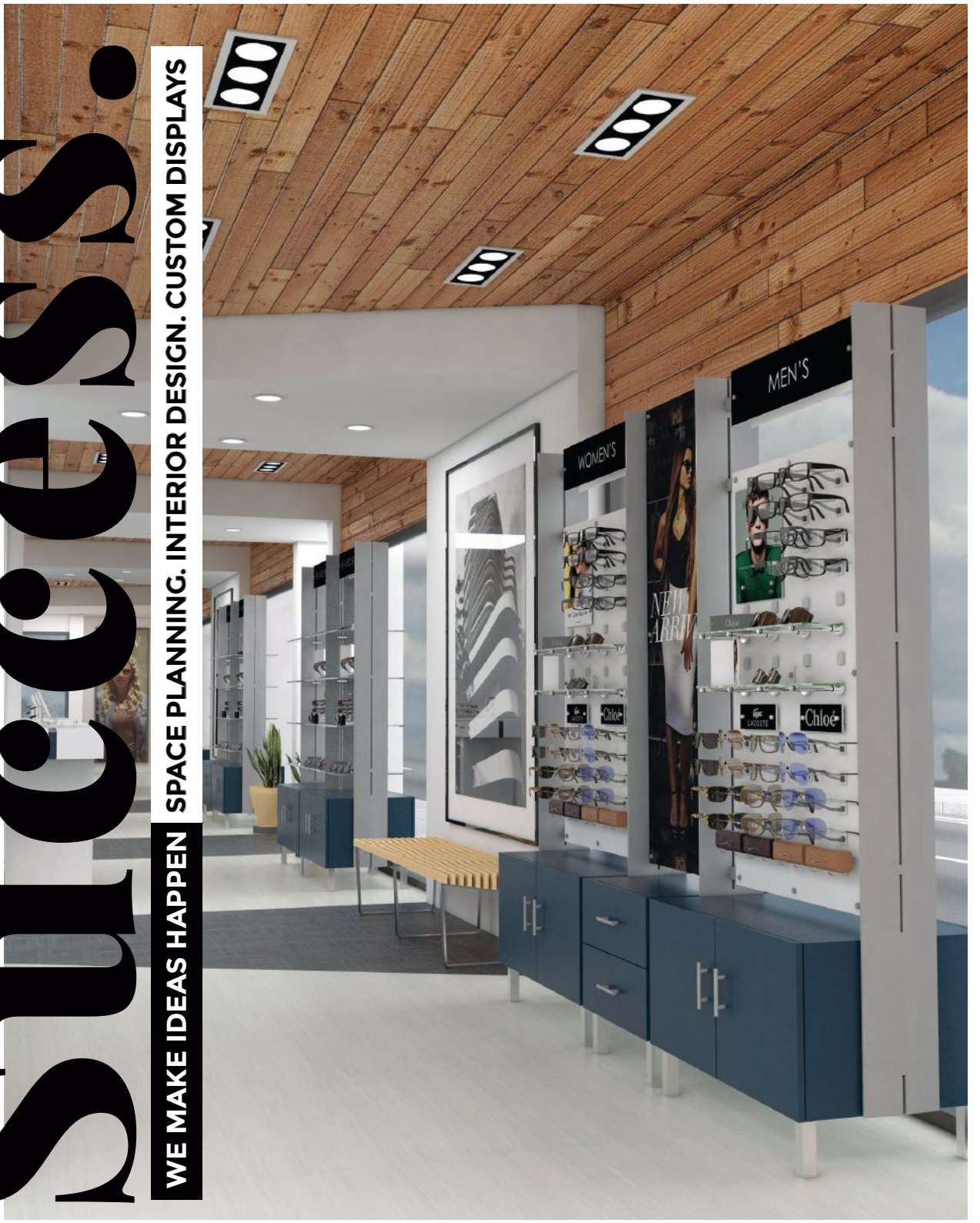

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Glaucoma Surgery-induced LSCD Unique

Limbal stem cell deficiency (LSCD) that develops after glaucoma surgery may have unique characteristics compared with LSCD caused by other factors such as chemical injury or ocular inflammatory disease, a study in *Cornea* reports.

A research team found classic features of LSCD induced by glaucoma surgery included sectoral replacement of corneal epithelial cells by conjunctival epithelial cells but without significant corneal neovascularization or pannus. On the other hand, LSCD caused by a chemical injury or ocular inflammatory disease presented with diffuse limbal involvement and corneal neovascularization.

The study enrolled 41 patients (51 eyes) with LSCD associated with glaucoma. Patients who underwent trabeculectomy and/or aqueous shunt surgery were included.

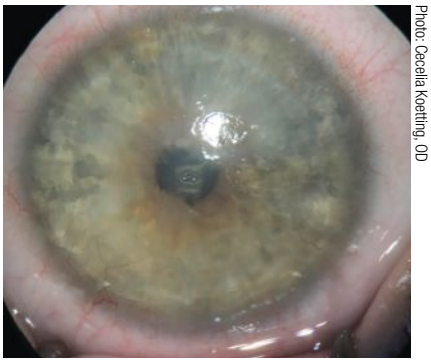

Photo: Cecelia Koetting, OD

LSCD induced by glaucoma surgery might not have the same clinical features as LSCD with other etiologies.

The study found a strong link between the site of the glaucoma surgery and the location of LSCD. Additionally, the researchers observed increased LSCD severity in eyes that had two or more glaucoma surgeries compared with those who underwent a single procedure, although the difference was not significant.

Also of note: the use of topical glaucoma medications appeared to be linked with LSCD severity, while the impact of antimetabolites didn't seem to be a factor.

"In the case of glaucoma, patients often require subsequent surgical revisions to achieve adequate control of intraocular pressure," the researchers wrote in their paper. "The current study reveals a trend toward more severe stages of LSCD in patients with multiple surgeries, suggesting that repeated glaucoma surgery predisposes to increased severity of LSCD, although a larger study is needed to confirm this observation."

Measures to reduce injury to the limbus should be considered to reduce the risk of LSCD, the study noted.

Sun Y, Yung M, Huang L, et al. Limbal stem cell deficiency after glaucoma surgery. Cornea. January 17, 2020. [Epub ahead of print].

Choroid Possible Marker of Preeclampsia

Preeclampsia is a risk factor for future cardiovascular and renal problems as well as the leading cause of maternal and perinatal morbidity and mortality worldwide. Researchers in Kuala Lumpur, Malaysia have determined that subfoveal choroidal thickness is higher in pregnant women with preeclampsia as well as found positive correlations with markers of severity of the condition. They believe that subfoveal choroidal thickness could be used as a novel predictor of preeclampsia's severity.

The study included 50 eyes each from 50 pregnant women with preeclampsia, 50 pregnant women without the condition and 50 age-matched normotensive, nonpregnant women. It defined preeclampsia as blood pressure above 140/90mm Hg on at least two occasions more than four hours apart after 20 weeks of gestation in previously normotensive patients, with significant proteinuria. The pregnant study participants in the two groups were in their third trimester of pregnancies.

The mean arterial blood pressure was higher in the preeclampsia than the normal pregnancy and nonpregnant women (103.0mm Hg vs. 83.2mm Hg vs. 89.5mm Hg, respectively). The subfoveal choroidal thickness of the preeclampsia group was higher than the other groups (370.7µm vs. 344.5µm vs. 315.8µm, respectively).

The study found positive correlations between subfoveal choroidal thickness and mean arterial blood pressure, ocular perfusion pressure and urine protein-to-creatinine ratio in the preeclampsia group. The study also found higher central corneal thickness and lower intraocular pressure (IOP) in pregnant women compared with those nonpregnant. However, there were no statistically significant differences in central corneal thickness, macular thickness or IOP between the preeclampsia and healthy, pregnant groups.

Sharudin SN, Saaid R, Samsudin A, Nor F. Subfoveal choroidal thickness in preeclampsia. Optom Vis Sci. 2020;97(2):81-85.

Corneal 'Speckle' Can Predict Glaucoma

Glaucoma is usually measured with tests that evaluate, among other things, the integrity of the optic nerve and the IOP. But researchers are now pointing to an OCT modality that may be able to identify early stages of the disease in an unexpected structure: the cornea. According to researchers, the corneal speckle of glaucoma suspects has a similar relationship between the parameters of scattering exhibited in glaucoma patients and is distinguishable from that of healthy controls.

Using OCT to measure speckle "can shed more light on the structural characteristics of corneal tissue and their relationship to glaucoma progression," the investigators wrote. They add that "corneal tissue should be given more consideration in future histopathology studies of glaucoma."

To determine this, investigators looked at 64 subjects separated into three groups; those with diagnosed primary open-angle glaucoma (18 patients), glaucoma suspects with normal levels of IOP and uncompromised visual field (24) and age-matched controls (22). The team looked at each patient's corneal speckle using OCT as we as their IOP, visual fields, Heidelberg Retinal Tomography, retinal nerve fiber layer thickness and biometry.

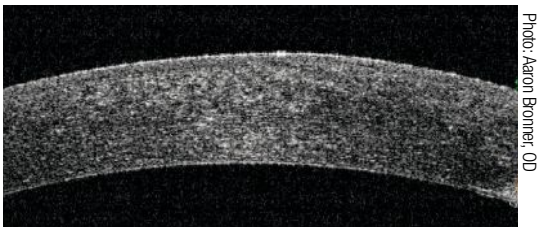

Photo: Aaron Bronner, OD

The corneal speckle captured with AS-OCT just might be a novel way to discover glaucoma.

When measuring IOP, visual field parameters, mean retinal fiber layer thickness and central corneal thickness, the glaucoma suspects all looked similar to the healthy controls and were significantly different than the confirmed glaucoma cases. However, the parameters of the corneal speckle were not significantly different between the groups, but they showed a markedly higher and statistically significant coefficient of determination for glaucoma patients and suspects than that for controls. This indicates "that glaucoma suspects have similar relationship between the corneal scatterer cross section and scatterer density to that exhibited in the glaucoma patients but markedly different from that of healthy controls."

Iskander D, Kostyszak M, Jesus D, et al. Assessing Corneal Speckle in Optical Coherence Tomography. Optom Vis Sci. 2020;97(2):62-7.

Myopia, Screen Time Not Linked

Since Americans have embraced the smartphone as a burgeoning limb extension, apprehension about its ramifications have run amok—especially in eye care. But, according to researchers, the ubiquitous screens we use can be acquitted of at least one charge: an association with myopia.

The investigators point out that, yes, digital screen time is frequently cited as a potential modifiable environmental risk factor for myopia. But, they say, this association is not consistent. For starters, myopia prevalence was already on the rise before digital devices really took off in some countries. So, the team looked through 15 studies that included a total of 49,789 children between the ages of three and 19. Seven studies found an association between screen time and myopia. Five found no association.

This review and meta-analysis summarizes the available relevant evidence, and the researchers found no clear association between screen time and myopia prevalence, incidence or myopia progression. This is one of the first systematic reviews to comprehensively summarize existing data on screen time in children and myopia.

They add that a particular nuance of the study is that screen time may lead to reduced time outdoors, which is associated with elevated myopia, regardless of how kids spend their time indoors. No data exists to clarify whether that indoor time is actually associated with digital device use. But they're not ready to call it a day yet, especially as smartphones continue to dominate ever-greater chunks of our time. "Given the rise in hours spent by children using screens, further studies are warranted using objective screen time measurements," the report reads. ■

Lanca C, Saw S. The association between digital screen time and myopia: a systemic review. Ophthal Physl Opt. January 13, 2020. [Epub ahead of print].

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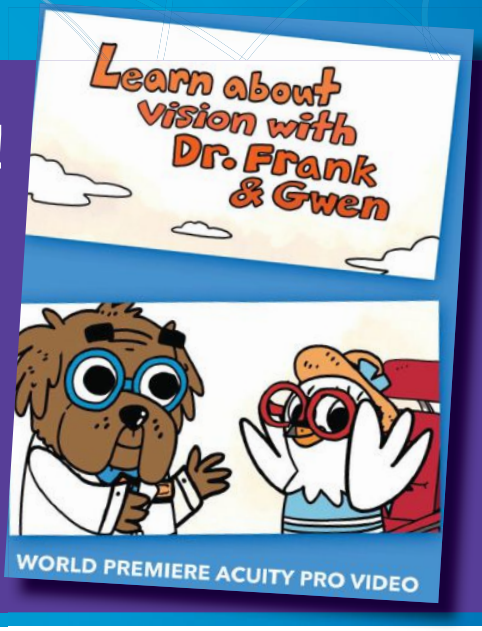

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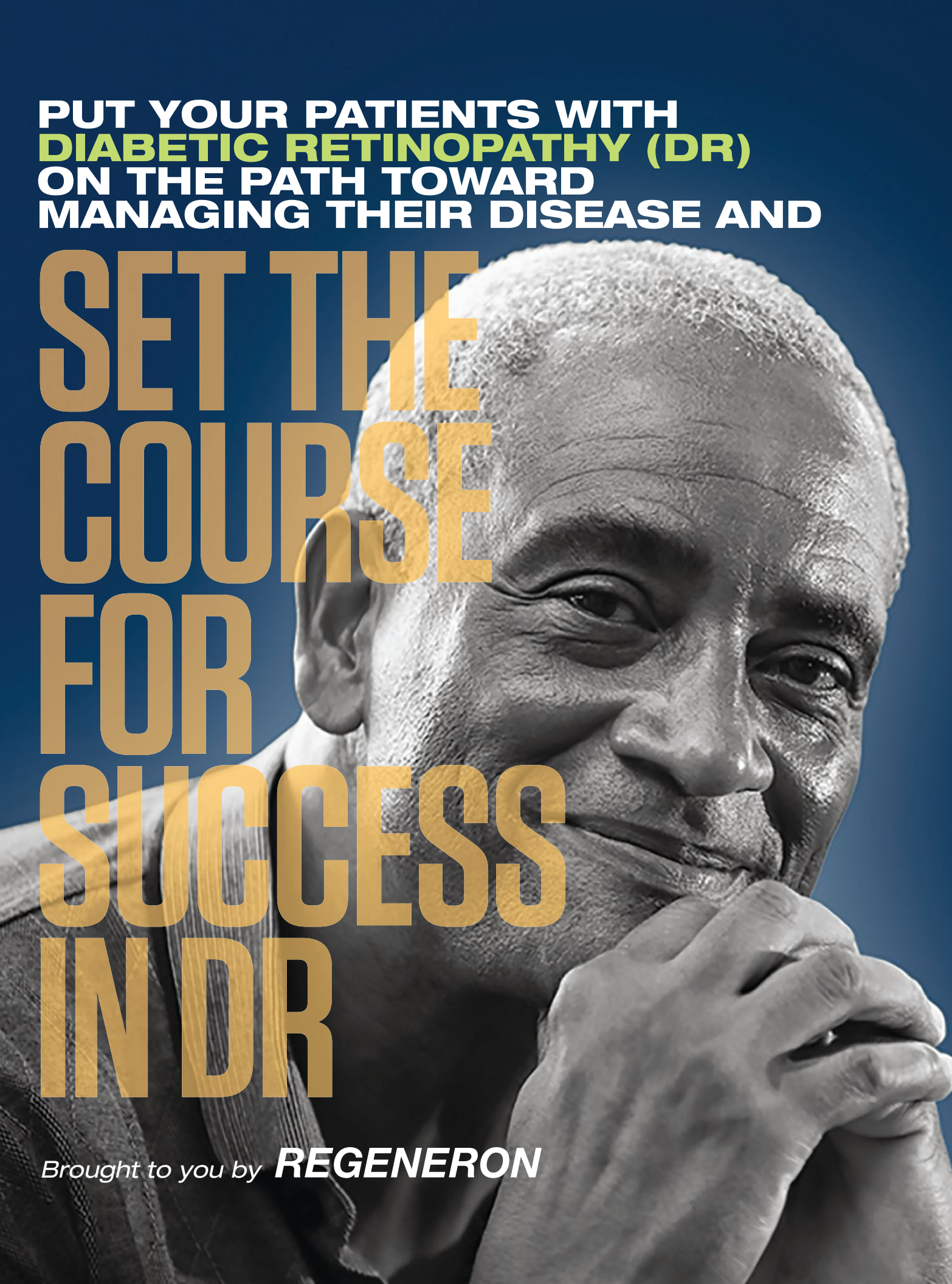
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Educate your patients about the severity of DR, especially when left untreated[3,4]

- Your early and frequent discussions about disease progression, treatment options, and referral will empower patients, which could help them avoid significant vision loss[3,4]

According to the AOA, you should refer patients with[3]:

- Severe nonproliferative DR (NPDR) within 2 to 4 weeks
- Proliferative DR (PDR) within 2 to 4 weeks
- High-risk PDR with or without macular edema within 24 to 48 hours

Ensure patients have followed up with a retina specialist who can treat DR

Monitor your patients with DR[3,4]

The AOA recommends frequent monitoring of patients[3]

- At least every 6 to 8 months in patients with moderate NPDR and more frequently for patients with greater disease severity[3]

Refer patients to a specialist who can treat DR[3,4]

Regeneron is committed to helping you partner with your patients for comprehensive care of DR, as well as for care of other retinal diseases.

AOA = American Optometric Association.

References: 1. Diabetic Retinopathy. Centers for Disease Control and Prevention website. http://bit.ly/2BKTVCTS. Accessed August 7, 2019. **2.** Early Treatment Diabetic Retinopathy Study Research Group. Fundus photographic risk factors for progression of diabetic retinopathy. ETDRS report number 12. *Ophthalmology*. 1991;98(5 suppl):823-833. **3.** Care of the Patient With Diabetes Mellitus: Quick Reference Guide. American Optometric Association website. http://bit.ly/2M22OUJ. Accessed August 7, 2019. **4.** Ferrucci S, Yeh B. Diabetic retinopathy by the numbers. *Rev Optom*. June 15, 2016. http://bit.ly/2KNNJ4E. Accessed August 7, 2019.

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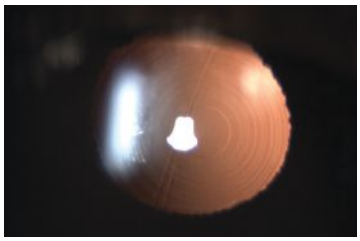

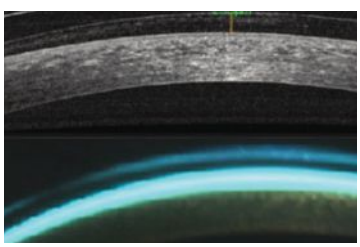

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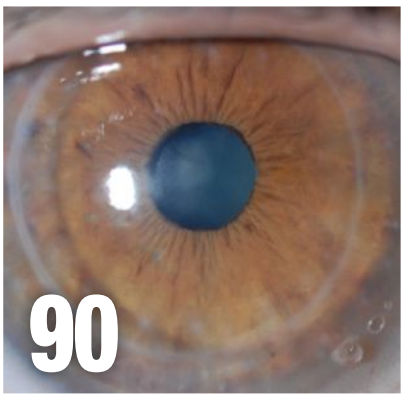

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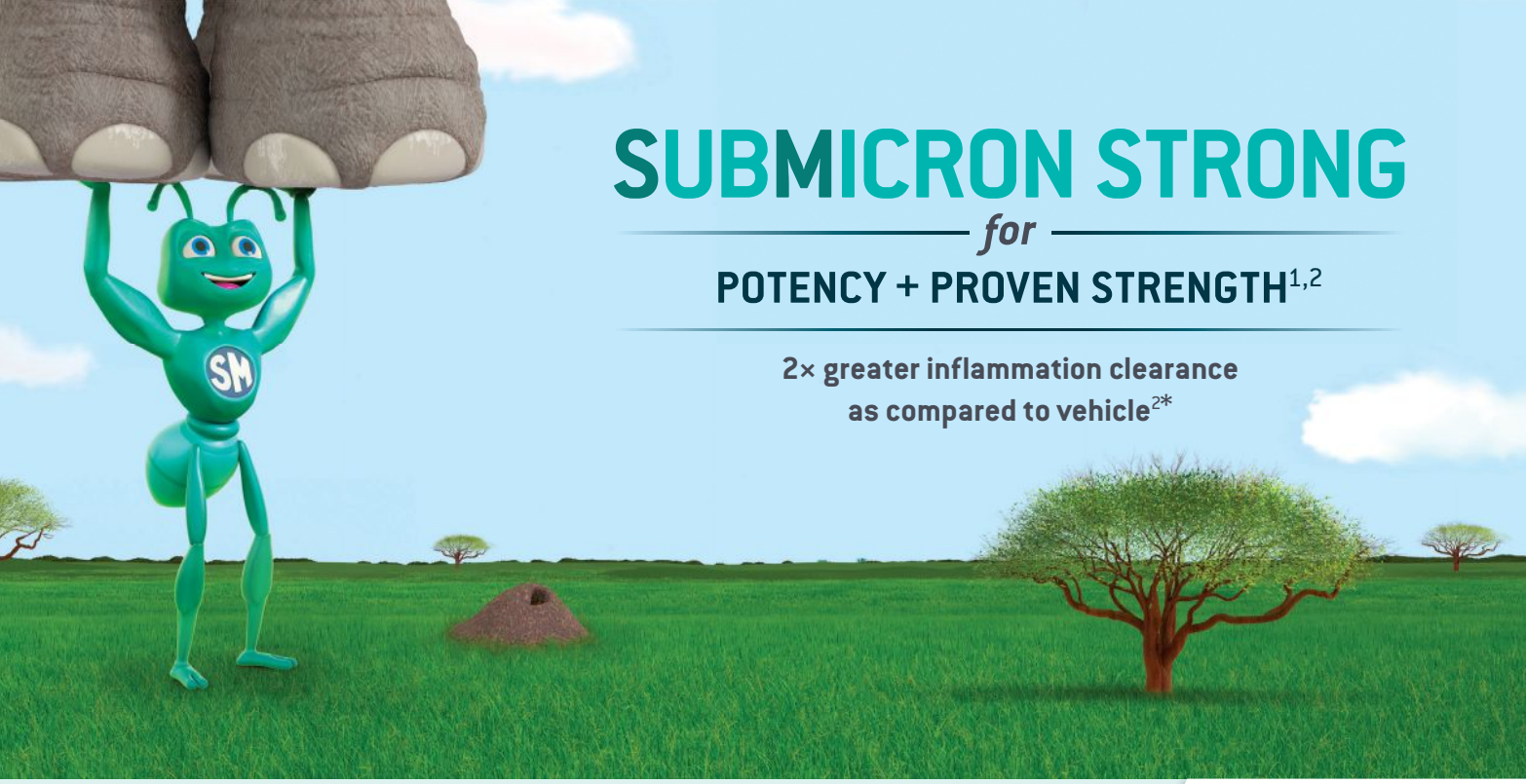

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- **30% of LOTEMAX® SM patients had complete ACC resolution** vs vehicle (15%) at Day 8 (N=371, *P*<0.0001)[1,2†]
- **74% of LOTEMAX® SM patients were completely pain-free** vs vehicle (49%) at Day 8 (N=371, *P*<0.0001)[1,2‡]

†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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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).

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REVIEW OF OPTOMETRY

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Outlook

By Jack Persico, Editor-in-Chief

Screening Measures

A meta-analysis challenges the belief that digital devices turbocharge myopia. But that doesn't let kids off the hook.

In news that would likely make my son jump for joy, it seems digital device use is not associated with myopia development, according to a recent meta-analysis of 15 studies comprising 50,000 subjects.[1] Kids everywhere—well, the ones who read up on medical news—might rejoice at the prospect of an end to parental scolding to 'put down your phone already.' But what should optometrists make of it? This swims against the tide of conjecture linking prolonged screen time with myopia and muddies the waters for ODs looking to advise patients and parents.

Increased near-work trends have been in place at least since 1980, the report notes, long before the ubiquity of digital devices. Worldwide, this chiefly reflects the development of Asian populations that have been transitioning from rural to urban societies. "Education and intensive schooling may have a larger contribution to the increase in myopia prevalence than screen time," the paper explains. Screen use in some ways is merely a substitution for other near work, such as reading or pen-and-paper educational activities.

While screens may not be public enemy #1, the study found, they aren't wholly blameless either. The authors did note a contributory effect. Time outdoors remains the best protective measure against the impetus toward myopia. Because they dissuade kids from outdoor play, digital screens do have some complicity, plus other ill effects.

Here the authors cite a World Health Organization (WHO) report that advises limiting screen time for children under age five, as it "may increase sedentary behavior with negative impact for children's health." For kids between ages one and four, screen time should be no more than one hour per day, the WHO says. Sedentary habits, of course, are a breeding ground for childhood obesity and diabetes, and should be discouraged on those grounds alone, to say nothing of any myopia effect.

How might all this change what you advise? There's no guidance in the report but it seems intuitive to still encourage mitigation of screen time, especially among young kids. Dismayingly, the report notes "the age children start to use smartphone devices is getting younger (22% start at three years old or less) and one in three children one- to six-years old use smartphones between one and two hours per day." If nothing else, such habits expose kids to blue light and decrease blink rates more so than any prior generation.

Like millions of other parents, my wife and I want to encourage our son to develop good habits and avoid bad ones. (Always eager to help the boy, we ate half his Halloween candy last year.) But reports like this that confound the experts leave us somewhat adrift. It's naïve to think kids are going to eschew such a useful (and, yes, fun) thing as a phone or tablet. Still, it pains me to see stats about very young kids being glued to a screen, especially when time outdoors confers so many benefits. To my three-year-old son, I still say, for now: Sorry, kiddo, go fly a kite. ■

1. Lanca C, Saw S. The association between digital screen time and myopia: a systemic review. Ophthal Physl Opt. January 13, 2020. [Epub ahead of print].

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Don't Blame Pharma

Industry bears the brunt of the public's anger for healthcare costs, but the true culprits are more elusive. **By Paul M. Karpecki, OD, Chief Clinical Editor**

Everyone is blaming each other for healthcare costs, and the lack of transparency allows each entity to get away with the accusations. With more transparency, we'll clearly see where the bad actors reside and make appropriate changes.

The Big Players

Think about a company with total revenues of $60.4 billion and operating earnings that grew to $5.0 billion in just the last three months, including a year-to-date growth of 17%. You might think this is a fast-growing biotechnology company. No. It's the largest medical insurance company in the country, United Healthcare.

These companies achieve such growth, in part, by inappropriately using existing tools such as prior authorizations to physicians or through their own pharmacy plan contracts. These blanket prior authorizations cause frustration, inefficiency, increased staff costs and delayed medications. Many doctors simply give up, resulting in patients receiving less-than-adequate medications.

When it comes to pharmacy plan contracts, the margins on insurance claims are regulated by the Affordable Care Act, but the margins on pharmaceutical plans are not. The public signs up on the insurance side, and the company makes up the difference on the highly margined drug plans they offer the patient or business. Because the insurance plans own the pharmaceutical plans, they can require patients to use these products and then raise the drug costs.

The Middlemen

Three pharmacy benefit managers (PBMs) have more than 94% of the market share—mainly because they are owned by the insurance companies. They have a pay-to-play, strong-arm approach with the pharmaceutical companies to take the majority of the profits. As an example, over an extended period of time, Travatan Z (Novartis) made $800 million, $600 million of which—75% of the revenue—went to the PBMs.

Pharmaceutical companies have to pay because the PBMs own the formularies that provide the drugs to their patients and charge exorbitant rates to companies that want their drugs accessible. To compensate for this fee, pharmaceutical companies increase their drug prices, passing the cost on to the patient.

Health insurance brokers are another layer of middlemen. Although some brokers do a great job, the vast majority receive kickbacks from insurance companies to sell the pharmaceutical benefit plans—a vicious cycle of hidden fees that inflate drug prices. Although some brokers don't play these games, 90% of the drugs go through PBMs that offer these kickbacks.

Canada has significantly lower drug prices because they don't have PBMs, health insurance brokers or other layers adding to the cost.

The Solutions

Transparency is finally coming. The president recently signed an executive order on price transparency that will be implemented over the next several months. But we should also write to our congressional representatives to recommend fixing the system rather than trying to find a way to pay for a faulty system.

Entities such as GoodRx are another terrific solution, as they shop for the best cash price of various therapeutic agents. But a word of caution: in my experience, the pharmacies sometimes tell patients they must provide their insurance card and then pay the required higher cost of the drug rather than the cash price provided by the Good Rx app. However, patients don't have to provide their insurance information and can simply pay the cash price if they want.

Some ophthalmic pharmaceutical companies are setting up their own distribution channels at lower costs and hopefully new options continue to be available to our patients.

If you want to better understand today's healthcare concerns, I recommend reading *The Price We Pay: What Broke American Healthcare—and How to Fix It* by Marty Makarey, MD.

The system needs healing, but it's not all the pharma's fault. Instead, the process begins with identifying the areas that lack transparency and shining a light on them. Only then will these issues become clear enough to put proper solutions in place. ■

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

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The Rebate Racket

Whoever thought asking patients to mail in proof-of-purchase to get money back on their contact lenses was a good idea? **By Montgomery Vickers, OD**

Rebates are, at best, wacky. At worst, they are a plot to destroy your will to live. Still, rebates are the feel-good film of the year… the Christmas movie where the girl takes over her sick mother's hardware store and then learns that people can buy hammers and nails online much cheaper than she can buy them wholesale. Don't worry, she'll fall in love with the delivery drone and live happily ever after.

The Problem

So, our labs quietly charge us more per box than they charge some company who purchases 100+ boxes per day, more than we ever could. They then explain that we can "beat" the big box/online prices by dangling the oh-so-seductive rebate in front of the patient like holding a brisket up in front of a starving pit bull.

Does it work? Pretty much, at least for offices that supply a year's batch of contacts as a matter of fact. Not so much for you namby-pamby doctors who fear rejection so much you ask dumb questions like, "How many boxes do you want?" or "Wanna buy lenses here?"

At least the rebate gives every office the chance to make a case for patients to purchase their contact lenses from their trusted doctor who (a) will be sure they are safe and correct and (b) will get them to you in a convenient and timely fashion.

But not all patients are accounting majors. If the online price is listed as $25 per box and the patient needs four boxes a year, that's $100 per year, right? Uh, not exactly. Shipping and handling discounts only kick in at eight boxes, for example.

At your office, the per-box charge is $33, an exorbitant $132 a year! The rebate is $50, assuming the patient remembers to send it in on time. You know they will send it in on Tuesday if it's due Monday, and it will get lost in the mail.

As a consequence, you, their trusted eye doctor for 25 years, are now a crooked jerk for the extra $32, you thief! You would have been better off just handing them $32. Heck, you would have been better off handing them the lenses for free, considering how much time you spent checking it out, calling the sales rep and then discussing how your office decided $33 per box was fair in the first place.

The Solution?

When you gripe to your contact lens sales rep, they just smile. If you gripe a little more, they pull out the big guns and show you the *new* rebates. The design team has done an amazing job, and I think each rebate form is coated with Prozac because you will feel a sudden sense of calm. It's really nice.

Now your contact lens problems are over. Except the new rebate is less than last year's, so now you get to explain why that happened to your long-time patient.

Even better, when you switch a patient from one contact lens company to another, the first rebate is higher than if you keep them in the lens design they have used for years. That's right, contact lens companies like to undervalue loyalty. Weird.

I think the rebate should *increase* each year the patient supports the same company. After, let's say, 10 years, the 11th year's supply should be free. Also, this escalating rebate should be null and void the first time the patient goes online instead of buying from their optometrist.

The origin of the word *rebate* is the old Anglo-Norman French word *rebatre*. It literally meant *to beat back* or *repel*. No wonder I hate rebates. They're dangerous! ■

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- The cumulative percentage of subjects receiving rescue medication of ocular steroid or nonsteroidal anti-inflammatory drug (NSAID) by day 30 was significantly lower in the DEXYCU (517 mcg) treatment group (20%; n=31/156) compared to placebo (54%; n=43/80)[1]

*DEXYCU was studied in a randomized, double-masked, placebo-controlled trial. Patients received either DEXYCU or a vehicle administered by a physician at the end of the surgical procedure. The primary endpoint was the proportion of patients with anterior chamber cell clearing (cell score=0) on postoperative day 8.

INDICATION AND USAGE

DEXYCU® (dexamethasone intraocular suspension) 9% is indicated for the treatment of postoperative inflammation.

IMPORTANT SAFETY INFORMATION

CONTRAINDICATIONS

None.

WARNINGS AND PRECAUTIONS

Increase in Intraocular Pressure
- Prolonged use of corticosteroids, including DEXYCU, 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

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 corticosteroids

Exacerbation of Infection
- The use of DEXYCU, as with other ophthalmic corticosteroids, is not recommended in the presence of most active 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 disease of ocular structures
- Use of a corticosteroid in the treatment of patients with a history of herpes simplex requires caution and may prolong the course and may exacerbate the severity of many viral infections
- Fungal infections of the cornea are particularly prone to coincidentally develop with long-term local steroid application and must be considered in any persistent corneal ulceration where a steroid has been used or is in use. Fungal culture should be taken when appropriate
- 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 infection

Cataract Progression
- The use of corticosteroids in phakic individuals may promote the development of posterior subcapsular cataracts

ADVERSE REACTIONS

- The most commonly reported adverse reactions occurred in 5-15% of subjects and included increases in intraocular pressure, corneal edema and iritis

Please see brief summary of full Prescribing Information on adjacent page.

References: 1. DEXYCU® (dexamethasone intraocular suspension) 9% full U.S. Prescribing Information. EyePoint Pharmaceuticals, Inc. December 2018. **2.** Donnenfeld E, Holland E. Dexamethasone intracameral drug-delivery suspension for inflammation associated with cataract surgery: a randomized, placebo-controlled, phase III trial. *Ophthalmology.* 2018;125(6):799-806. **3.** Data on file. EyePoint Pharmaceuticals, Inc.

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10/2019
US-DEX-1900208

DEXYCU (dexamethasone intraocular suspension) 9%,
for intraocular administration
Initial U.S. Approval: 1958

BRIEF SUMMARY: Please see package insert for full prescribing information.

1 INDICATIONS AND USAGE

DEXYCU (dexamethasone intraocular suspension) 9% is indicated for the treatment of postoperative inflammation.

4 CONTRAINDICATIONS

None.

5 WARNINGS AND PRECAUTIONS

5.1 Increase in Intraocular Pressure

Prolonged use of corticosteroids including DEXYCU 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.

5.2 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 corticosteroids.

5.3 Exacerbation of Infection

The use of DEXYCU, as with other ophthalmic corticosteroids, is not recommended in the presence of most active 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 disease of ocular structures.

Employment of a corticosteroid medication in the treatment of patients with a history of herpes simplex requires 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 culture should be taken when appropriate.

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 infection.

5.4 Cataract Progression

The use of corticosteroids in phakic individuals may promote the development of posterior subcapsular cataracts.

6 ADVERSE REACTIONS

The following adverse reactions are described elsewhere in the labeling:

- Increase in Intraocular Pressure *[see Warning and Precautions (5.1)]*
- Delayed Healing *[see Warnings and Precautions (5.2)]*
- Infection Exacerbation *[see Warnings and Precautions (5.3)]*
- Cataract Progression *[see Warnings and Precautions (5.4)]*

6.1 Clinical Trials Experience

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

The following adverse events rates are derived from three clinical trials in which 339 patients received the 517 microgram dose of DEXYCU. The most commonly reported adverse reactions occurred in 5-15% of subjects and included increases in intraocular pressure, corneal edema and iritis. Other ocular adverse reactions occurring in 1-5% of subjects included, corneal endothelial cell loss, blepharitis, eye pain, cystoid macular edema, dry eye, ocular inflammation, posterior capsule opacification, blurred vision, reduced visual acuity, vitreous floaters, foreign body sensation, photophobia, and vitreous detachment.

8 USE IN SPECIFIC POPULATIONS

8.1 Pregnancy

Risk Summary

There are no adequate and well-controlled studies of DEXYCU (dexamethasone intraocular suspension) in pregnant women. Topical ocular administration of dexamethasone in mice and rabbits during the period of organogenesis produced cleft palate and embryofetal death in mice and malformations of abdominal wall/intestines and kidneys in rabbits at doses 7 and 5 times higher than the injected recommended human ophthalmic dose (RHOD) of DEXYCU (517 micrograms dexamethasone), respectively *[see Data in the full prescribing information]*.

In the US general population the estimated background risk of major birth defects and miscarriage in clinically recognized pregnancies is 2 to 4% and 15 to 20%, respectively.

8.2 Lactation

Risk Summary

Systemically administered corticosteroids are present in human milk and can suppress growth, interfere with endogenous corticosteroid production, or cause other unwanted effects. There is no information regarding the presence of injected DEXYCU in human milk, the effects on breastfed infants, or the effects on milk production to inform risk of DEXYCU to an infant during lactation. The developmental and health benefits of breastfeeding should be considered, along with the mother's clinical need for DEXYCU and any potential adverse effects on the breastfed child from DEXYCU.

8.4 Pediatric Use

Safety and effectiveness of DEXYCU in pediatric patients have not been established.

8.5 Geriatric Use

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

Manufactured for: EyePoint Pharmaceuticals US, Inc. Watertown, MA 02472

Chew on This

A dental abscess may be the cause of a patient's preseptal or orbital cellulitis.

Edited by Paul C. Ajamian, OD

Q **A patient presented with a two-week history of preseptal cellulitis, with no improvement after an ENT consult and four days of hospitalization with IV antibiotics. Other than facial swelling that was hard to the touch, he reported no pain or other symptoms. Any suggestions?**

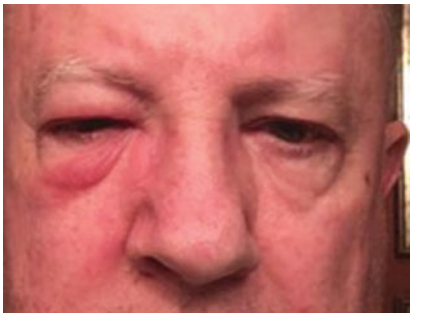
A "Preseptal cellulitis is an infection of the eyelid that occurs anterior to the orbital septum," says Paige Thompson, OD, of SouthEast Eye Specialists in Chattanooga, TN. "Orbital cellulitis is a less common but more serious eyelid infection that involves the tissues posterior to the orbital septum." The orbital septum provides an important boundary between the anterior eyelid and the deep orbital tissues.[1,2]

Presentation and Management

Preseptal cellulitis classically presents with unilateral eyelid edema and erythema, which can affect the skin above and below the lid. The most frequent underlying etiologies include an incipient chalazion, sinusitis and trauma.[3,4]

Preseptal cellulitis is managed with prompt treatment with oral antibiotics. Amoxicillin-clavulanic acid is often preferred, but Keflex (cephalexin, Advancis Pharmaceutical) and doxycycline (tetracyline class) are also used.[5] Monitor patients daily until you note clear improvement.

"Patients should exhibit complete resolution of the condition within one week of antibiotic therapy," Dr. Thompson says. "If preseptal cellulitis is not managed appropriately, infection can spread to the orbital septum via the venous system."



Preseptal cellulitis unresponsive to antibiotics could need dental extraction.

Orbital cellulitis also presents with eyelid edema and erythema. These patients can exhibit proptosis, extraocular motility restriction, afferent pupillary defect and/or vision loss. This condition is most commonly caused by ethmoidal sinusitis, due to the proximity of the sinus cavities to the orbital structures.[1,3,4]

Refer all cases of suspected orbital cellulitis to an orbital specialist or the emergency department for prompt neuroimaging, especially those not responding to conventional therapy. "I prefer a CT scan of the brain, including orbits and sinuses," Dr. Thompson says. After lab work, treat these patients with combined IV antibiotics followed by oral antibiotic therapy.

Tooth Infections

A lesser-known cause of preseptal and orbital cellulitis is dental infection. These infections are specifically associated with maxillary molar abscesses.[6] The patient may not be complaining of localized tooth pain, which can cloud the diagnosis.

To diagnose suspected cases, endodontists can order a cone beam CT scan—a specific type of panoramic x-ray that provides detailed images of the jaw, teeth, nasal cavity and sinuses.[7] "Always consider dental abscess in cases of preseptal cellulitis that are not responding to therapy," Dr. Thompson says. Most of these cases require dental extraction to eliminate the infection.[6,8,9]

Our referral to the patient's dentist and an oral surgeon resulted in the extraction of the infected molar and complete resolution of the facial cellulitis within a few days.

Preseptal and orbital infections may have serious consequences if not treated appropriately. Perform a thorough clinical examination, including visual acuity, pupillary assessment, extraocular motility evaluation and exophthalmometry in each of these cases.

"If the patient does not have a clear history of the common underlying causes we outlined, consider an underlying asymptomatic dental abscess," Dr. Thompson says. ■

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A Peek at the Pinhole

This simple test can quickly categorize a patient's reduced vision as either refractive or pathological. Here's what else it can do. **By Bisant A. Labib, OD**

A patient may present to your office with reduced vision for any number of reasons, ranging from something as benign as uncorrected refractive error to serious pathology. Even in cases in which a combination of factors affect vision, the ability to distinguish between contributory ocular pathology and a refractive error is a must. Despite today's high-tech tools—such as autorefractors or the potential acuity meter (PAM), to name a few—many ODs turn to a tried-and-true low-tech option: the pinhole occluder.

Due to its effects of limiting light scatter and subsequent blur, the pinhole can serve as a quick test to determine the need for further investigation of underlying ocular pathologies if the vision does not improve with pinhole.

Optics Explained

Pinhole occluders are inexpensive and readily available tools that consist of multiple, small apertures amidst a dark background. They are available for purchase or, in some cases, can even be fashioned from punching approximately 1mm to 2mm holes in a dark card.[1]

The pinhole occluder works along the same basis as pupil constriction in bright conditions causing an improvement in visual acuity. Through a smaller pupil, the effects of minor ocular irregularities—such as refractive error or paracentral cornea or lens opacities—are diminished.

The "pinhole effect" is an optical concept suggesting that the smaller the pupil size, the less defocus from spherical aberrations is present. When light passes through a small pinhole or pupil, all unfocused rays are blocked, leaving only focused light to land on the retina to form a clear image.[1,2] In contrast, patients who have very large pupils, whether it is physiological or from pharmacological dilation, experience image "ghosting," or a blurring halo around an otherwise sharp image.[3]

The size of the pinhole aperture or pupil size is an important consideration. While there is great benefit, as mentioned, to limiting marginal and unfocused rays from reaching the retina using a small aperture, a pinhole or pupil that is too small may cause diffraction and loss of resolution.[1] Diffraction occurs when a light wave is fractured by the edge of the aperture, which limits the amount of detail available to form a clear retinal image.[4] Additionally, a pinhole too small reduces retinal illumination. A clinical study evaluating pinhole size concluded that a diameter between 0.94mm to 1.75mm is most effective, with most instruments on the market containing approximately 1.2mm apertures.[5]

Pupil constriction is also involved in the near triad of accommodation, convergence and miosis. A balance of these factors is essential to increase depth of focus and aid in providing a clear near image. The effect of pupil constriction, or use of a pinhole, is such that the subsequent increase in depth of focus lessens the accommodative demand required.[3]

Pinhole in Practice

The obvious benefit of using this optical principle in clinical practice is to discriminate between reduced visual acuity secondary to refractive error and the presence of pathology. When a patient's visual acuity is

not 20/20 despite the use of pinhole, further investigation to determine an underlying cause is warranted.

This is also a useful tool to check best-corrected visual acuity in patients for whom performing a refraction is unnecessary or difficult.[6] In a practical setting, pinhole visual acuity may be used to document vision of a patient returning for a medical follow-up; routine refraction is not indicated, but sometimes the patient does not bring their up-to-date spectacle or contact lens correction to the appointment.

Another worthy application is in the context of measuring potential visual acuity post cataract extraction. Potential acuity pinhole (PAP) is a monocular test using a pinhole occluder to view a near target amidst bright illumination to predict visual status postoperatively.

The patient is first dilated so that they are able to search for a subjectively clearer area that may be less obstructed from lenticular opacities. Patients are then instructed to read the letters on a near card brightly lit with a transilluminator. The importance of using direct, bright lighting is to compensate for the reduced illumination from both the reduced viewing size and the cataract itself. The intensity of the light also helps to diminish the light scattering effect innate to cataracts.[2,3] The best-corrected visual acuity measured using PAP correlates to the expected post cataract surgery visual outcome.

A similar test is the PAM test, which requires additional and more costly equipment for projection. In a study comparing the accuracy of PAP and PAM testing, both methods were similar in determining visual outcome post cataract surgery, though PAM provided a slightly more accurate correlation.[3]

Using pinhole visual acuity can offer the clinician a great deal of important information. It functions as a rapid tool to screen for best-corrected visual acuity without having to employ refractive techniques. It also suggests that if and when a pinhole fails to improve vision, the reduced vision is likely a result of pathology, whether a structural defect or functional change. Additionally, it is a readily available and cost-effective alternative to determine postoperative visual endpoints for patients undergoing cataract surgery. ■

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- Significant improvement in corneal staining as early as 1 month[2,4]
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INDICATIONS AND USAGE

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

IMPORTANT SAFETY INFORMATION

WARNINGS AND PRECAUTIONS

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

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

ADVERSE REACTIONS

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

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

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

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SUN OPHTHALMICS

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

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

INDICATIONS AND USAGE

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

CONTRAINDICATIONS

None.

WARNINGS AND PRECAUTIONS

Potential for Eye Injury and Contamination

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

Use with Contact Lenses

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

ADVERSE REACTIONS

Clinical Trials Experience

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

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

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

USE IN SPECIFIC POPULATIONS

Pregnancy

Risk Summary

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

Data

Animal Data

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

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

Lactation

Risk Summary

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

Pediatric Use

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

Geriatric Use

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

PATIENT COUNSELING INFORMATION

Handling the Vial

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

Use with Contact Lenses

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

Administration

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

Rx Only

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Cranbury, NJ 08512

PLR-00020 2018

SUN OPHTHALMICS

Spring—and Pollen—is in the Air

Are you ready for ocular allergy season? New testing could make it a breeze.

By John Rumpakis, OD, MBA, Clinical Coding Editor

Spring is a great time of year, but it's also when our offices are flooded with patients symptomatic with ocular allergy. Allergies are an extremely commonplace condition, and nearly half of the patients who walk into your practice will present with signs or symptoms of this common affliction.

Fresh Approaches Pay Off

Allergy is a chronic concern, and clinicians must consider a full-scope approach to its management. This begins with the right diagnosis and, sometimes, new approaches to care.

For example, Doctor's Rx Allergy Formula: Ocular Allergy Diagnostic System (Bausch + Lomb) allows you to effectively test, in-office, for up to 58 allergens with regional specificity and have results within 15 minutes.[1] This can help you properly identify the specific allergen involved and guide your treatment. It may also help isolate and differentiate allergic manifestations from other common concomitant ocular surface conditions. Currently, 19 states allow its use in optometric scope of practice.

To code for this test, use CPT code 95004, defined as a "percutaneous test(s) with allergenic extracts, immediate type reaction, including test interpretation and report by a physician." The 2020 CMS National Average Allowable for this code is about $4.32.[2] When submitting your claim, the number of units is 58 (one unit per allergen), with a billing total of $250.56. However, remember that your charges will be individually determined.

Allergy Coding Refresher

Coding and compliance requirements for an ocular allergy visit should be in line with other anterior segment conditions. Often, the first presentation of allergy is discovered during the comprehensive exam's case history, rather than the patient showing up with frank symptoms.

When you discover signs and symptoms during the routine exam and you choose to initiate or change therapy, only code the exam (and refraction) and bill the encounter to the managed vision care carrier, as the patient didn't present with signs and symptoms of ocular allergy. If they did, a comprehensive exam would not be the appropriate code to use.

The subsequent follow-up visit (generally in one week) would meet the requirement of a doctor-directed visit for a specific reason, thus meeting the CC requirements as well as those of medical necessity, which fulfills the requirements for a medical encounter.

For most cases, the return visit would be an E/M visit code. Most likely the level of the code would be either a 99212 or a 99213 based on meeting the criterion of history, physical exam elements and medical decision making for this visit. Sometimes, a 92002/92012 could be appropriate to use as well, provided that you meet the CPT definition of that code, currently defined as "an evaluation of a new or existing condition complicated with a new diagnostic or management problem not necessarily relating to the primary diagnosis, including history, general medical observation, external ocular and adnexal examination and other diagnostic procedure as indicated."

While some disagree with the application of this definition, it is the current 2020 CPT definition.[3]

There are no CCI edits that affect coding a 920X2 and 95004 on the same date; however, there are issues when performing an E/M code and 95004 on the same date of service. Follow-up evaluations to determine the efficacy of your medical therapy are essential for appropriate follow-up and the typical six-month interval between ocular allergy visits.

The ICD-10 codes typically associated with ocular allergy are H10.001 - H10.019 (acute follicular conjunctivitis), H10.43 - H43.439 (chronic follicular conjunctivitis), H10.44 (vernal conjunctivitis) and H10.45 (other chronic allergic conjunctivitis).

Ocular allergy is a prevalent condition in the United States, and making your patients aware that you diagnose and treat ocular allergy could be a boon for your practice. You have all the tools and expertise to provide easily accessible, professional care for your patient—and that's nothing to sneeze at! ■

Send your coding questions to rocodingconnection@gmail.com.

1. Bausch + Lomb. Doctor's Rx Allergy Formula: Ocular Allergy Diagnostic System. www.bausch.com/ecp/our-products/diagnostics/ocular-allergy-diagnostic-system. Accessed February 3, 2020.
2. American College of Allergy, Asthma and Immunology. Proposed 2020 Medicare Physician Fee Schedule. https://college.acaai.org/advocacy/resources/proposed-2020-medicare-physician-fee-schedule. Accessed February 3, 2020.
3. CPT Professional Edition 2020. American Medical Association. 2020:656.

Steroid Wars:
New Drugs Challenge Old Habits

Here's how novel delivery systems and updated formulations may one day overcome the current challenges inherent in topical steroid prescribing.

By Jeffrey Sterling, OD, and Heather Whyte DeMarco, OD

The old medical adage of "first, do no harm" is a guiding principle that has steered those in the medical field since ancient times. The simple directive preaches caution in the uncertain world of diagnostics and therapeutics. In optometry, this mindset often makes clinicians pause before they consider using topical steroids. Adverse reactions from steroid use include, but are not limited to, cataract formation, increased intraocular pressure (IOP), possible secondary infections, delayed wound healing and even central serous retinopathy (CSR).[1,2] Although these events can be serious and are worth considering, the benefits of steroids often outweigh their risks.

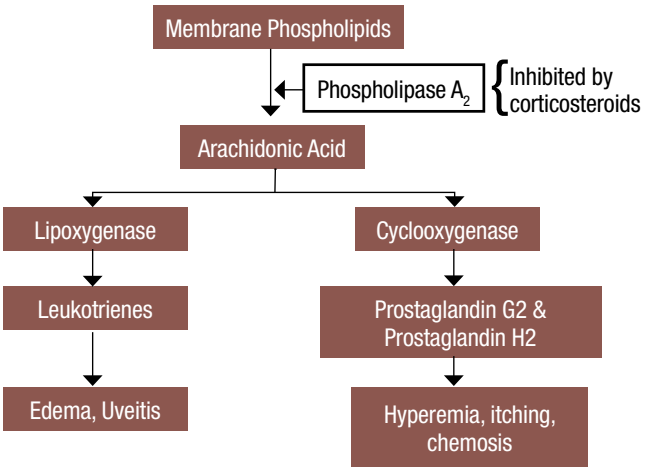

Fig. 1. This is a simplified view of how steroids affect the inflammatory cascade.

Pros and Cons

Inflammation is the body's response to injury or infection, resulting in the release of mast cells, cytokines and other pro-inflammatory mediators.[3] Tissue may turn red, become painful or swell during inflammation. In the eye, inflammation may also manifest with injection, blurred vision and photophobia. Ophthalmic corticosteroids, or glucocorticosteroids, are named accordingly because they are similar to the naturally occurring anti-inflammatory hormone, cortisol. They downregulate the inflammatory pathways by inhibiting phospholipase A_2.[4] Thus, steroids help mediate inflammation by decreasing histamine synthesis, capillary dilation and fibroblast formation (*Figure 1*).

As with all medications, steroids come with the risk of adverse effects. One of the key concerns many optometrists have when prescribing steroids is their effect on IOP. Up to a third of the population may develop induced ocular hypertension after topical steroid use.[5] However, IOP will typically return to pre-treatment levels within three weeks after the cessation of steroid use.[6] Posterior subcapsular cataract formation can also be a result of chronic steroid use, both topically and systemically.[7]

Additionally, steroid use is associated with CSR.[8] When this presents, it is imperative that clinicians ask the patient about all of their steroid use, because even over-the-counter topical and inhaled steroids can lead to ocular side effects. Caution must also be used when prescribing steroids with any corneal insult. This is because steroids can suppress the body's immune response and delay wound healing by downregulating transforming growth factor-β and insulin-like growth factor-I.[9,10] On the other hand, because they lower the immune response, steroids are heavily used after corneal grafts to reduce the likelihood of rejection.[11]

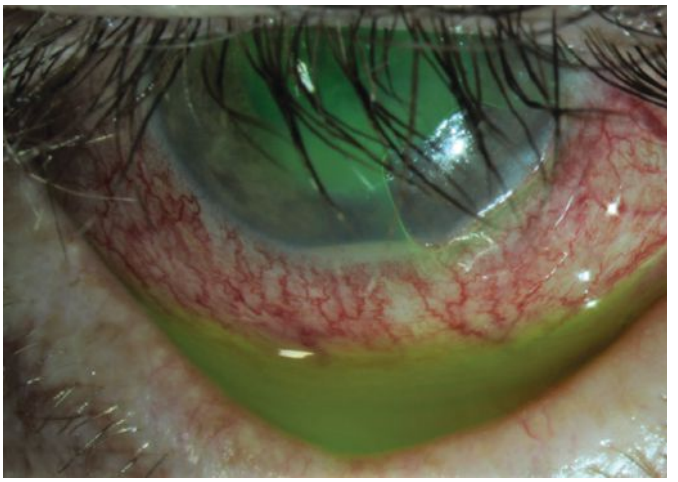

Fig. 2. This patient presented with a corneal abrasion and hypopyon. He was treated successfully with a bandage contact lens, a topical steroid and a topical antibiotic.

Formulation 411

It may seem logical that the incidence and severity of adverse events from steroid use would increase with the strength of the steroid. However, not all steroids are the same, and the medication's strength is only part of the puzzle. Many steroids are available, each with their own formulation that ultimately affects their potency and the risk of side effects. The specific chemical make-up of each steroid plays a major role in its potential to create an adverse event. Steroid molecules may be attached to acetate, alcohol or phosphate bases—with acetates penetrating an intact cornea the best.[12] Additionally, the delivery vehicle also plays a role.

Today's optometrist not only has to decide on the particular steroid and strength to use, but also if they want a solution, suspension, gel or ointment. Several new options have hit the market in recent years, further complicating the decision (*Table 1*). Here is a look at the current options and how newer formulations and delivery methods might change your prescribing habits:

Pred Forte (prednisolone acetate ophthalmic suspension 1%, Allergan) has been the workhorse steroid of choice for generations. This product and its generic formulations are suspensions and, therefore, require vigorous shaking prior to instillation. Still, not all suspensions are created equal. A 2007 comparison of branded vs. generic preparations showed that the branded version had smaller and more uniform prednisone particles, which made it more effective in combating ocular inflammation.[13] Prednisolone is also available in a phosphate form that is a solution instead of a suspension and therefore does not require vigorous shaking. The phosphate formulation, however, lacks the potency of the acetate form.[12]

A potent alternative to Pred Forte is ***Durezol (difluprednate ophthalmic emulsion 0.05%, Novartis)***. This "designer" steroid adds two fluorine atoms to the prednisone molecule to increase its strength.[14] It is approved for the treatment of postoperative pain and inflammation as well as anterior uveitis. Because Durezol is an emulsion, it does not require shaking to deliver an equal amount of active ingredient with each drop. This may be an advantage when treating patients with anterior uveitis who may require dosing every hour with prednisone. Research shows that Durezol four times a day is as good as prednisolone acetate eight times a day; however, it is worth noting that Durezol is more likely to increase IOP than prednisone.[15]

Topical ***dexamethasone*** and ***fluorometholone***, like prednisolone, are corticosteroid suspension eye drops that have been available for years. Both medications are less potent than prednisolone and are considered viable alternatives when clinicians need to mitigate the risks of a stronger steroid.[1]

To further decrease the risk of increased IOP or other events, many optometrists will look to "soft steroids." These are active compounds that deactivate in a predictable manner during systemic absorption.[16] An example of a soft steroid is loteprednol. Two of the oldest and most well-known are ***Alrex (loteprednol etabonate ophthalmic suspension 0.2%, Bausch + Lomb)*** and ***Lotemax (loteprednol etabonate ophthalmic suspension 0.5%, Bausch + Lomb)***. The weaker of the two, Alrex, is approved for temporary relief of the signs and symptoms of seasonal allergic conjunctivitis.

Lotemax, with more than twice the potency of Alrex, is approved for pain and inflammation after cataract surgery along with other inflammatory conditions such

Table 1. Newly Approved Steroid Medications

Brand	Generic	Preparation	Approval	Notes
Inveltys	Loteprednol etabonate 1%	Suspension	Postoperative inflammation and pain	BID dosing
Lotemax SM	Loteprednol etabonate 0.38%	Gel	Postoperative inflammation and pain	TID dosing
Dexycu	Dexamethasone intraocular suspension	Injected suspension	Postoperative inflammation	30-day release
Yutiq	Fluocinolone acetonide	Intravitreal implant	Chronic noninfectious uveitis	36-month release
Dextenza	dexamethasone	Intracanalicular insert	Postoperative inflammation and pain	30-day release

as iritis and allergic conjunctivitis.[17] While Lotemax was initially released (and is still available) in a suspension form, it is also available as an ointment and a gel. The ointment is approved for use after multiple ocular surgeries, including LASIK, cataract, minimally invasive glaucoma surgeries and Descemet's stripping endothelial keratoplasty.[17]

Two gel formulations exist, ***Lotemax gel (loteprednol etabonate 0.5%, Bausch + Lomb)*** and ***Lotemax SM (loteprednol etabonate 0.38%, Bausch + Lomb)***. Both have similar indications as the suspension and ointment. One major benefit of the gel formulation is that it ensures a consistent amount of active drug in each drop without the need for shaking.[18] Additionally, the gel vehicle remains on the ocular surface longer than a suspension, which helps maximize absorption potential.[19]

The newest member of the family is Lotemax SM, which uses what the manufacturer calls "submicron technology" to help the medication adhere to the ocular surface. This is designed to improve penetration to the targeted tissues, including presence within the aqueous humor.[20,21]

With four different Lotemax products to choose from, clinicians have to consider several factors when deciding what to prescribe, including efficacy, availability and affordability. Ointments are great because they don't require shaking, stay on the eye and can provide some lubrication. However, they can blur vision, and some patients may struggle to instill the proper one-half inch ribbon dose. Lotemax gel and Lotemax SM do not need shaking and may require less dosing, but new products may be more expensive and not as widely available.

In August 2018, ***Inveltys (loteprednol etabonate ophthalmic suspension 1%, Kala Pharmaceuticals)*** was approved for postoperative pain and inflammation after ocular surgery. The manufacturer states that Inveltys uses mucus-penetrating nanoparticles to penetrate the ocular surface and reach its target tissue.[22] Inveltys is unique among ocular steroids in that it was approved for twice-a-day dosing; other corticosteroids require at least three to four drops a day after surgery.[23] This may be particularly useful for patients who require the help of others to instill their medications, have dexterity limitations or have active lifestyles and may be at risk for poor compliance. Postoperative regimens are usually determined by the surgeon, but if you are comanaging cataract surgery, it may be worth your time to discuss this option with your referring surgeon.

Dropless Options

Because patient compliance usually increases as the number of medications and dosage decreases, companies have been working to provide steroid options with decreased dosing. To that end, two new medications have been approved that hope to eliminate the need for postoperative steroid drops altogether.

Dexycu (dexamethasone intraocular suspension 9%, EyePoint Pharmaceuticals) is indicated for the treatment of postoperative inflammation. This drug is injected behind the iris and into the posterior chamber after cataract surgery and is slowly released for up to 30 days.

The second drug is ***Dextenza (dexamethasone ophthalmic insert 0.4mg, Ocular Therapeutix)***; it delivers a corticosteroid to the eye by way of an intracanalicular insert. Dextenza is a preservative-free insert placed in the lower puncta after surgery. The drug is also slowly released over a 30-day period. Since it is resorbable, there is usually no need to remove it.

The big advantage of these alternative pathways is minimizing patient burden. Fewer drops often equal better compliance and can reduce the amount of preservative on the corneal surface, which could lead to increased patient comfort. Optometrists may also like the dosing control provided by Dextenza and Dexycu. With these medications, there is less worry about the consistency of drug delivery to the target tissue. Side effects can still occur and need to be monitored in the postoperative period.

While not used in optometric practices, injectable steroids are also available. ***Ozurdex (dexamethasone 0.7mg intravitreal implant, Allergan)*** is approved for the treatment of macular edema from vein occlusions, diabetic macular edema and for non-infectious uveitis. After insertion, the implant releases 100ug/ml to

ZERVIATE delivers the proven power of cetirizine—a leading oral allergy medication—now targeted to the eyes.[1,2,a]

- Clinically meaningful and rapid reduction in ocular itch vs vehicle at 15 minutes and 8 hours (P<0.0001 at all time points measured)[1,3]
- Designed for comfortable delivery with Hydrella™, a glycerin and HPMC formulation[1,4]
- Administered as a single drop, twice daily approximately 8 hours apart[1]

ZERVIATE™
cetirizine ophthalmic solution, 0.24%
FORMULATED WITH HYDRELLA™

Learn more at MyZerviate.com

STUDY DESIGN: The pivotal trials for ZERVIATE included two Phase 3, double-masked, randomized, vehicle-controlled, parallel-group studies involving 201 patients. Study 2 required more severe allergic conjunctivitis symptoms. Patients were screened for an allergen response using the conjunctival allergen challenge (CAC) model and randomized to receive either ZERVIATE or vehicle. Primary efficacy endpoints were ocular itching and conjunctival redness 15 minutes and 8 hours post treatment instillation.[3]

INDICATIONS AND USAGE

ZERVIATE™ (cetirizine ophthalmic solution) 0.24% is a histamine-1 (H1) receptor antagonist indicated for treatment of ocular itching associated with allergic conjunctivitis.

DOSAGE AND ADMINISTRATION

Instill one drop of ZERVIATE in each affected eye twice daily (approximately 8 hours apart).

IMPORTANT SAFETY INFORMATION

WARNINGS AND PRECAUTIONS

Contamination of Tip and Solution: As with any eye drop, care should be taken not to touch the eyelids or surrounding areas with the dropper tip of the bottle or tip of the single-use container to avoid injury to the eye and to prevent contaminating the tip and solution. Keep the multi-dose bottle closed when not in use. Discard the single-use container after using in each eye.

Contact Lens Wear: Patients should be advised not to wear a contact lens if their eye is red. ZERVIATE should not be instilled while wearing contact lenses. Remove contact lenses prior to instillation of ZERVIATE. The preservative in ZERVIATE, benzalkonium chloride, may be absorbed by soft contact lenses. Lenses may be reinserted 10 minutes following administration of ZERVIATE.

ADVERSE REACTIONS

The most commonly reported adverse reactions occurred in approximately 1%–7% of patients treated with either ZERVIATE or vehicle. These reactions were ocular hyperemia, instillation site pain, and visual acuity reduced.

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

HPMC=hydroxypropyl methylcellulose.

[a]Based on a *U.S. News* report on data from the 2019 *Pharmacy Times* Survey of Pharmacists' OTC Recommendations.

References: 1. ZERVIATE [package insert]. Fort Worth, Texas: Eyevance Pharmaceuticals LLC; 2020. **2.** *U.S. News & World Report.* Antihistamines for allergies. https://health.usnews.com/health-products/top-rec-antihistamines-oral-8. Accessed October 7, 2019. **3.** Meier EJ, Torkildsen GL, Gomes PJ, et al. Phase III trials examining the efficacy of cetirizine ophthalmic solution 0.24% compared to vehicle for the treatment of allergic conjunctivitis in the conjunctival allergen challenge model. *Clin Ophthalmol*. 2018;12:2617-2628. **4.** Malhotra RP, Meier E, Torkildsen G, et al. Safety of cetirizine ophthalmic solution 0.24% for the treatment of allergic conjunctivitis in adult and pediatric subjects. *Clin Ophthalmol*. 2019;13:403-413.

ZERVIATE™ (cetirizine ophthalmic solution) 0.24%

Brief Summary

INDICATIONS AND USAGE

ZERVIATE (cetirizine ophthalmic solution) 0.24% is a histamine-1 (H1) receptor antagonist indicated for treatment of ocular itching associated with allergic conjunctivitis.

DOSAGE AND ADMINISTRATION

Recommended Dosing: Instill one drop of ZERVIATE in each affected eye twice daily (approximately 8 hours apart). The single-use containers are to be used immediately after opening and can be used to dose both eyes. Discard the single-use container and any remaining contents after administration. The single-use containers should be stored in the original foil pouch until ready to use.

CONTRAINDICATIONS

None.

WARNINGS AND PRECAUTIONS

Contamination of Tip and Solution: As with any eye drop, care should be taken not to touch the eyelids or surrounding areas with the dropper tip of the bottle or tip of the single-use container to avoid injury to the eye and to prevent contaminating the tip and solution. Keep the multi-dose bottle closed when not in use. Discard the single-use container after using in each eye.

Contact Lens Wear: Patients should be advised not to wear a contact lens if their eye is red.

ZERVIATE should not be instilled while wearing contact lenses. Remove contact lenses prior to instillation of ZERVIATE. The preservative in ZERVIATE, benzalkonium chloride, may be absorbed by soft contact lenses. Lenses may be reinserted 10 minutes following administration of ZERVIATE.

ADVERSE REACTIONS

Clinical Trials Experience: Because clinical trials are conducted under widely varying conditions, adverse reaction rates observed in the clinical trial 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 7 clinical trials, patients with allergic conjunctivitis or those at risk of developing allergic conjunctivitis received one drop of either cetirizine (N=511) or vehicle (N=329) in one or both eyes. The most commonly reported adverse reactions occurred in approximately 1%–7% of patients treated with either ZERVIATE or vehicle. These reactions were ocular hyperemia, instillation site pain, and visual acuity reduced.

USE IN SPECIFIC POPULATIONS

Pregnancy

Risk Summary

There were no adequate or well-controlled studies with ZERVIATE in pregnant women. Cetirizine should be used in pregnancy only if the potential benefit justifies the potential risk to the fetus.

Data

Animal Data

Cetirizine was not teratogenic in mice, rats, or rabbits at oral doses up to 96, 225, and 135 mg/kg, respectively (approximately 1300, 4930, and 7400 times the maximum recommended human ophthalmic dose (MRHOD), on a mg/m^2 basis).

Lactation

Risk Summary

Cetirizine has been reported to be excreted in human breast milk following oral administration. Multiple doses of oral dose cetirizine (10 mg tablets once daily for 10 days) resulted in systemic levels (Mean C_{max} = 311 ng/mL) that were 100 times higher than the observed human exposure (Mean C_{max} = 3.1 ng/mL) following twice daily administration of cetirizine ophthalmic solution 0.24% to both eyes for 1 week. Comparable bioavailability has been found between the tablet and syrup dosage forms. However, it is not known whether the systemic absorption resulting from topical ocular administration of ZERVIATE could produce detectable quantities in human breast milk.

There is no adequate information regarding the effects of cetirizine on breastfed infants, or the effects on milk production to inform risk of ZERVIATE to an infant during lactation. The developmental and health benefits of breastfeeding should be considered along with the mother's clinical need for ZERVIATE and any potential adverse effects on the breastfed child from ZERVIATE.

Pediatric Use: The safety and effectiveness of ZERVIATE has been established in pediatric patients two years of age and older. Use of ZERVIATE in these pediatric patients is supported by evidence from adequate and well-controlled studies of ZERVIATE in pediatric and adult patients.

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

NONCLINICAL TOXICOLOGY

Carcinogenesis, Mutagenesis, Impairment of Fertility

Carcinogenicity

In a 2-year carcinogenicity study in rats, orally administered cetirizine was not carcinogenic at dietary doses up to 20 mg/kg (approximately 550 times the MRHOD, on a mg/m^2 basis). In a 2-year carcinogenicity study in mice, cetirizine caused an increased incidence of benign liver tumors in males at a dietary dose of 16 mg/kg (approximately 220 times the MRHOD, on a mg/m^2 basis). No increase in the incidence of liver tumors was observed in mice at a dietary dose of 4 mg/kg (approximately 55 times the MRHOD, on a mg/m^2 basis). The clinical significance of these findings during long-term use of cetirizine is not known.

Mutagenesis

Cetirizine was not mutagenic in the Ames test or in an *in vivo* micronucleus test in rats. Cetirizine was not clastogenic in the human lymphocyte assay or the mouse lymphoma assay.

Impairment of Fertility

In a fertility and general reproductive performance study in mice, cetirizine did not impair fertility at an oral dose of 64 mg/kg (approximately 875 times the MRHOD, on a mg/m^2 basis).

PATIENT COUNSELING INFORMATION

Risk of Contamination: Advise patients not to touch dropper tip to eyelids or surrounding areas, as this may contaminate the dropper tip and ophthalmic solution. Advise patients to keep the bottle closed when not in use. Advise patients to discard single-use containers after each use.

Concomitant Use of Contact Lenses: Advise patients not to wear contact lenses if their eyes are red. Advise patients that ZERVIATE should not be used to treat contact lens–related irritation. Advise patients to remove contact lenses prior to instillation of ZERVIATE. The preservative in ZERVIATE solution, benzalkonium chloride, may be absorbed by soft contact lenses. Lenses may be reinserted 10 minutes following administration of ZERVIATE.

Administration: Advise patients that the solution from one single-use container is to be used immediately after opening. Advise patients that the single-use container can be used to dose both eyes. Discard the single-use container and remaining contents immediately after administration.

Storage of Single-use Containers:
Instruct patients to store single-use containers in the original foil pouch until ready to use.

Rx Only

Manufactured by: Renaissance Lakewood, LLC. Lakewood, NJ 08701

Distributed by: Eyevance Pharmaceuticals LLC. Fort Worth, TX 76102

eyevance® pharmaceuticals

ZER-01-20-MS-10

1,000ug/ml of the drug for the first two months and then becomes undetectable after seven to eight months.[24] The use of intraocular steroids for macular edema seems to be decreasing along with an increased use of anti-VEGF drugs, possibly because of the increased risk of elevated IOP with intraocular dexamamethasone.[25]

More recently, ***Yutiq (fluocinolone acetonide 0.18mg intravitreal implant, EyePoint Pharmaceuticals)*** was approved for chronic noninfectious posterior uveitis. The Yutiq implant delivers a sustained release of drug for up to 36 months. This may be a great alternative to an oral systemic steroid; the local injection eliminates the risks of non-compliance and systemic side effects.[26]

Off-label, On Target

The on-label approval for most steroids is for pain after surgery, anterior uveitis or allergy—but there are times when using them off-label may be appropriate (*Figure 2*):

Dry eye. Clinicians often say that early dry is hard to diagnose and easy to treat, while advanced dry eye is easy to diagnose and hard to treat. Symptomatic patients with chronic inflammation from dry eye disease (DED) can get fast relief with topical steroid drops. The Tear Film and Ocular Surface Society's Dry Eye Workshop II reported that topical steroids are useful at breaking the cycle of inflammation associated with DED. Also, patients given steroids before or with the initial treatment of cyclosporin did better than patients who were only given cyclosporin.[27]

Steroid use in DED may be short- or long-term; thus, it may be wise to choose a soft steroid or one that has a lower risk of adverse effects.[28] Flarex (fluorometholone, Eyevance Pharmaceuticals) or loteprednol are good choices and are usually dosed two to four times a day, depending on signs and symptoms.

Corneal ulcers. Many ODs shy away from using topical steroids when the corneal epithelium is not fully intact. This is likely due to the fact the steroids can delay wound healing and have the potential to suppress the immune response, which may lead to an increased risk of infection. However, the Steroids for Corneal Ulcers Trial looked at the use of topical steroids after 48 hours of antibiotics in cases of corneal ulcers and found that long-term vision improve with no safety concerns when steroids were used as an adjunctive therapy for infections not caused by *Nocardia*.[29,30] We have routinely treated non-central corneal ulcers with a combination of steroids and antibiotics with great success. We prefer two-in-one medications such as ***Tobradex (dexamethasone 0.1%/tobramycin 0.3%, Eyevance Pharmaceuticals)*** or ***Zylet (loteprednol 0.5%/tobramycin 0.3%, Bausch + Lomb)*** over two separate

DO's and DON'Ts of Steriod Use

Although steroids provide effective treatment for many ocular conditions, several contraindications exist. Ultimately, the benefit must outweigh the risk. Topical ocular steroids have low systemic absorption, but their side effects can be considerable. Here are a few dos and don'ts when prescribing steroids:[1]

1. *DO* hit the inflammation hard and fast. The eye is considered an immune-privileged site, as it can control immune responses to protect the visual system's delicate components.[2] Thus, evidence of inflammation within the eye is uncommon and should be treated promptly to avoid any harmful effects of chronic inflammation. Typically, treatment with a steroid that can penetrate the cornea and treat the anterior chamber inflammation, such as prednisolone acetate or Durezol, is most appropriate. Clinicians should consider dosing every hour to two hours while awake initially.

2. *DON'T* taper too quickly. Avoiding a fast taper will minimize risk of rebound inflammation. Regarding anterior chamber inflammation, the rule is not to begin your taper until you notice a two-fold improvement in inflammation (based on the Standardization of Uveitis Nomenclature criteria).[3] However, prolonged use of topical steroids leads to an increased risk of potential adverse effects.[4]

3. *DO* refer for potential periocular or intraocular steroid treatment. For intermediate and/or posterior inflammation, cystoid macular edema and recalcitrant cases of uveitis, referral for possible treatment with subtenon's or intravitreal injections is warranted.

4. *DON'T* allow refills. One of the added benefits of topical steroids is that they provide almost instant relief of discomfort. However, they are not always safe and appropriate to use, and patients shouldn't have ongoing access. When another ocular condition presents, patients may be tempted to refill the prescription that made them better previously, but this could be problematic if they have a herpetic, bacterial or fungal infection.

5. *DO* educate your patient on the formulation. Due to the different ophthalmic formulations, clinicians must educate patients on which steroids require shaking. Patients can easily related to the analogy that a suspension is akin to salad dressing; it requires vigorous shaking to ensure that the medication mixes well before installation—an imperative step for successful treatment.

6. *DON'T* be afraid to treat the inflammation in a known steroid responder. Because chronic inflammation can cause significant damage to the immune-privileged site of the eye, quelling inflammation even in a steroid responder is necessary. In these patients, start a topical hypotensive agent along with the steroid and continue until the patient is done with the steroid. Additionally, be sure to check intraocular pressure at each visit.

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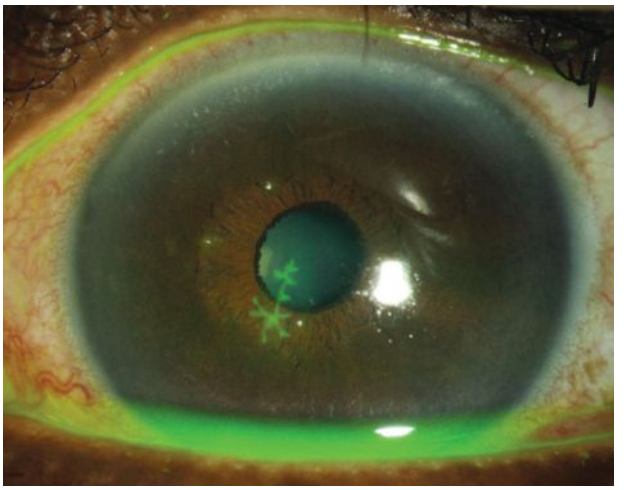

Fig. 3. Clinicians should avoid prescribing steroids for an epithelial herpetic infection, but they are helpful for cases of stromal herpetic keratitis.

prescriptions. While there is a certain amount of risk with this approach, it can be mitigated with careful patient education and close follow-up.

Herpetic stromal eye disease. Certainly, one of the greatest risks when prescribing steroids is that of herpes simplex keratitis. The classic corneal dendrite is pathognomonic for an epithelial herpetic infection (*Figure 3*). When dendritic ulcers are present, clinicians should avoid topical steroids, as they may prolong the infection and lead to a larger geographic ulcer. These ulcers can be treated either by topical or oral antiviral medications.

However, in the case of stromal herpetic keratitis without epithelial ulceration, topical steroids should be prescribed at dosages reflective of the amount of inflammation present. This is typically done in conjunction with oral antivirals to reduce the risk of epithelial involvement.[31]

Steroids are an integral part of optometric practice, and we are fortunate to have many topical options at our disposal. Knowing what makes each drug unique is critical when selecting the best opti for your patient. A strong, deep penetrating steroid may not be needed for mild ocular surface inflammation. Contrarily, a less potent steroid will not get the desired results for severe uveitis. Being aware of the potential adverse effects that may occur when prescribing steroids and how to address them will allow a clinician to treat patients with confidence. In the ever-changing world of pharmaceuticals, the best eye drop for your patient may not be a drop in the near future. ■

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Contents in this publication do not represent the view of the US Department of Veterans Affairs or the United States government.

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Modifying Your Glaucoma Regimen: When, Why and How

Managing this disease requires careful consideration of myriad factors. Here's how you can structure your approach. By Andrew Mackner, OD

As optometrists, we are well positioned to diagnose and treat glaucoma; however, it's not as simple as identifying someone with elevated intraocular pressure (IOP) and then lowering it as much as possible. One report from nearly 20 years ago calculated more than 56,000 ways to treat glaucoma after taking into account all available medications and possible regimens ranging from monotherapy to maximum medical therapy.[1] With new treatments at our disposal, that number is now considerably higher.

In addition, glaucoma itself is a complex condition with varying presentations, rates of progression and responses to treatment. Properly treating the condition first requires the clinician to accurately identify the type of glaucoma, order and analyze appropriate testing and initiate an appropriate treatment plan with suitable follow up—there is no one-size-fits-all treatment protocol.

Although glaucoma care remains more of an art than an exact science, a structured approach allows us to initiate—and tweak—treatment plans for each individual.

Where You Start Matters

The diagnosis and decision to initiate treatment is based on a comprehensive glaucoma workup that includes a detailed case history, slit lamp biomicroscopy, tonometry, pachymetry, gonioscopy, retinal nerve fiber layer (RNFL) and ganglion cell complex imaging, perimetry and dilated optic nerve assessment. One of the most challenging aspects of managing glaucoma is the interpretation of this conglomeration of data.

The dilated fundus exam, with an assessment of the optic nerve head, is one of the most important parts of a glaucoma evaluation. Pay close attention to the cup-to-disc ratio, integrity of the neuroretinal rim and the presence or absence of an optic disc hemorrhage, peripapillary changes and baring or bayoneting of the retinal vasculature. Indications of glaucomatous changes noted during a dilated fundus exam should correlate and be consistent with optical coherence tomography (OCT) and visual field findings to warrant initiation of treatment. Beyond using gonioscopy for the initial diagnosis and classification of glaucoma, this testing should be repeated annually, as the anterior chamber angle tends to narrow with age. When determining whether to initiate treatment, pachymetry values and corneal hysteresis are especially important, as patients with thin corneas and low corneal hysteresis values are at an increased risk of developing glaucoma.[2]

First-line Options

The decision to initiate treatment—and what treatment to start with—should take into account testing, exam findings, the individual's risk factors for developing glaucoma, the burden of long-term treatment, possible adverse effects, inconvenience and cost.[3] Initial therapy with drops or selective laser trabeculoplasty (SLT) are both acceptable first-line treatments.

Prostaglandin analogs have been the preferred initial pharmaceutical treatment due to their ability to effectively lower IOP with a once-daily dose.[4] Research

shows prostaglandin analogs are superior to other topical IOP-lowering classes in controlling diurnal and nocturnal IOP.[4] Prostaglandins are also extremely safe, with no reported systemic adverse reactions.[5] However, ocular side effects include darkening of iris color, lash growth, periocular skin pigmentation, fat distribution changes and conjunctival hyperemia.[4,5] These ocular side effects should be considered, especially when treating unilaterally or in younger patients.

SLT is also a viable first-line treatment option. Recently, the Laser in Glaucoma and Ocular Hypertension Study demonstrated that SLT is both clinically effective and cost-effective as an initial intervention for primary open-angle glaucoma (POAG) and ocular hypertension.[6] The benefits of SLT include eliminating the risk of poor compliance with medications, given the burdens placed on patients who must take one or more topical hypotensive agents daily.

Change Tactics

After initiating therapy, follow-up is guided by the severity of disease. Typically, monitoring patients with serial OCT and visual fields every six to 12 months for mild disease and every six months for moderate to severe disease is sufficient.

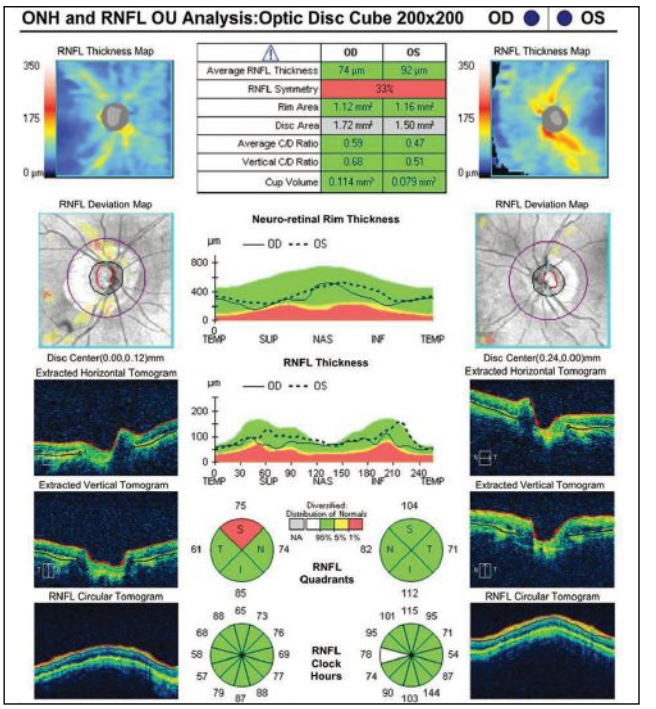
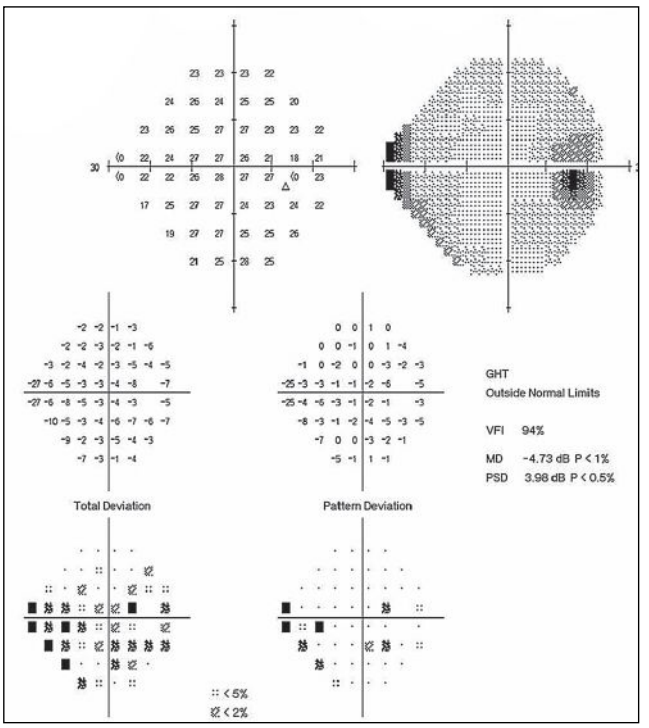
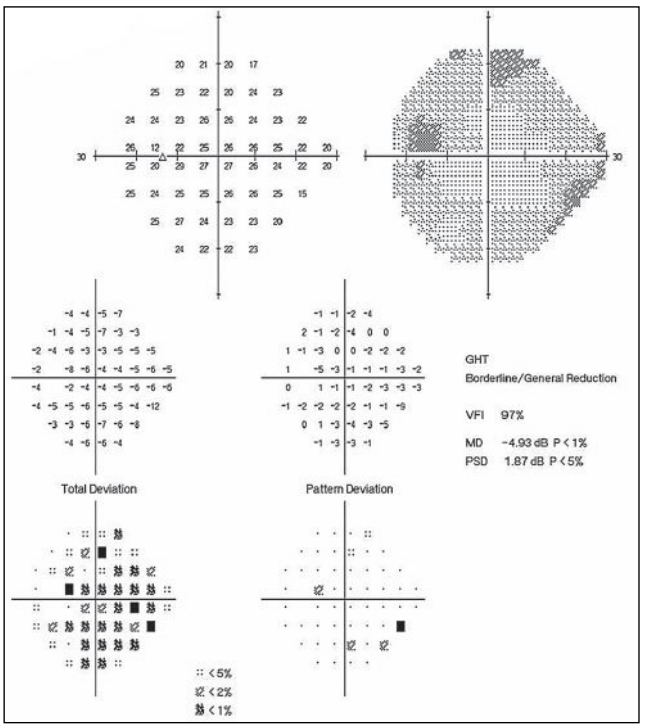


Fig. 1. This 92-year-old white female is currently taking latanoprost OU at bedtime. She has a maximum IOP of 22mm Hg with an IOP on exam of 18mm Hg OU. Corneal thicknesses are 523µm OD and 520µm OS. The patient's testing shows only mild visual field loss. Given her age and clinical picture, it is unlikely she will experience real functional impairment in her lifetime. A mild (even if confirmed) progression of RNFL loss might not be an indication to escalate therapy.





The concept of reaching a target IOP has gained acceptance in glaucoma management; however, it remains unclear whether that target number should be a percent reduction, an absolute number, low IOP fluctuations or low nocturnal IOP.[7] This treatment approach was reinforced by a post hoc analysis from the Advanced Glaucoma Intervention Study, which suggested that lowering IOP to below 18mm Hg at all visits or an average IOP of 12.3mm Hg would halt glaucoma progression.[4] Other landmark glaucoma studies provide evidence for percent reduction in IOP.

Still, practitioners who rigidly adhere to a target level or range may potentially be doing more harm than good, especially in low-risk patients with thick corneas.[4] The target IOP concept is also limited by the fact that glaucoma care involves balancing the complex set of risks and benefits that accompany escalating therapy. Simply adding medications to reach a target IOP, regardless of evidence of disease progression, does not take these considerations into account. Lastly, no randomized clinical control trials show that the use of a target IOP is superior to any other approach.[4]

Instead, clinicians should take into account both structural and functional changes as well as potential adverse reactions, costs to the patient and burden of treatment prior to escalating medical treatment or considering surgical intervention. An experienced clinician takes a dynamic approach, weighing these risks and benefits, with an understanding that these factors change over time. Thus, treatment decisions should be made based on the current disease state of an individual patient and their progression.[4]

OCT and visual field testing play a critical role in evaluating structural and functional changes and monitoring for disease progression. Although white-on-white perimetry has been considered the standard for monitoring glaucoma, progressive RNFL thinning increases the risk of visual field progression, and a significant amount of RNFL damage can occur before a functional defect is apparent on visual field testing.[8,9] OCT analysis provides an objective measure of the RNFL thickness.[8] Repeatable evidence of RNFL thinning on OCT or repeatable visual field progression are indications to consider changing a patient's glaucoma treatment. Recent studies show that loss of the ganglion cell complex may occur prior to loss of the RNFL, and assessment of the ganglion cell complex may be useful in determining progression of patients with pre-perimetric glaucoma.[9]

For example, if a patient's IOP and visual fields are stable but progressive RNFL thinning is confirmed on repeat testing, this would be an indication to consider escalating the patient's treatment. However, if a patient has borderline IOP control and stable RNFL and visual fields, this would be an indication to monitor without changing the treatment regimen. When evaluating testing and making the decision to escalate or initiate therapy, the patient's age, life expectancy and stage of

Glaucoma FAQs

Glaucoma is the leading cause of blindness in the United States, and researchers estimate that three to six million people are at risk of developing the condition because of elevated IOP.[1] Furthermore, large epidemiologic studies show that one third to approximately half of glaucoma cases have an IOP at or below 21mm Hg.[2] Other population-based studies show that only 50% of patients with glaucomatous visual field loss have received an appropriate diagnosis or treatment and that 50% of glaucoma cases may be undiagnosed.[2,3] Within the United States, therapeutic management of glaucoma costs an estimated $2.5 billion annually.[4] With these numbers in mind, not only is early detection and treatment important for preserving a patient's vision and quality of life, it is also important to treat the patient appropriately and avoid over-treatment.

Classification of glaucoma is important to ensure proper treatment paradigms. By definition, glaucoma is a diverse group of conditions that potentially results in progressive damage to the RNFL and associated visual field loss as the disease progresses.[3] Clinically, glaucoma is managed by lowering the patient's IOP because landmark glaucoma treatment trials demonstrate that reduction in IOP is associated with a reduced risk of visual field progression.[5] The Ocular Hypertension Treatment Study helped identify key risk factors for glaucoma patients. It also showed that topical hypotensive therapy is effective in reducing the incidence of glaucoma in ocular hypertensive patients. The study demonstrated that delaying treatment has a small effect on the incidence of POAG in low-risk patients but has a larger effect on reducing the incidence of glaucoma in high-risk patients.[1,3] In the Early Manifest Glaucoma Trial, each 1mm Hg increase in IOP during follow up was associated with a 12% increase in the development of visual field progression.[2] Understanding the fundamental elements of these studies and incorporating their clinical pearls is critically important in the management of glaucoma.

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glaucoma must also be taken into account (*Figure 1*).

Once a patient's clinical picture dictates a change in their glaucoma management, clinicians should remember to make one change at a time. Using a single medication as an adjunct therapy prior to moving to a combination drop allows the practitioner to assess if the patient has an adequate IOP reduction with the new medication. It also allows the practitioner to assess the patient's tolerance of the new adjunct therapy. If the IOP response is inadequate with the initial adjunct therapy or the patient exhibits progression, you still have the option to change the treatment by either recommending SLT or switching the patient to a combination drop.

Many clinicians turn to beta blockers as a first-line adjunct medication for patients already on a prostaglandin analog. This class of medication is typically well tolerated and can be dosed once or twice a day.[4] Beta blockers are contraindicated in patients with asthma, bronchospasm, chronic obstructive lung disease, heart failure, sinus bradycardia, atrioventricular block and cardiogenic shock.[5] A second option for adjunct therapy is a topical carbonic anhydrase inhibitor, which has better control of diurnal IOP fluctuations when used as an adjunct treatment with a prostaglandin analog.[4]

In patients for whom a beta blocker is contraindicated or who do not have an adequate response to a beta blocker, a carbonic anhydrase inhibitor, alpha 2-agonist or the rho-kinasd inhibitor netarsudil (Rhopressa, Aerie Pharmaceuticals) are viable next options for increasing the patient's treatment.

Additional Considerations

Before altering a patient's glaucoma treatment, clinicians should also consider cost, brand name vs. generic and adverse reactions to medication.

Financial burden. Glaucoma medications can be expensive, and the yearly cost of a medication can vary greatly depending on the class, whether it is branded or generic and dosing frequency.[10] Clinicians should consider the patient's formulary, the efficacy of the medication and patient financial obligations while treating glaucoma.

Branded vs. generic. Most commonly, clinicians switch to a generic medication due to cost. While generic medications must contain the same concentration of active ingredients, they often vary in non-active ingredients, bottle design and drop volume.[11] Therapeutic dosage in a topical medication is largely dependent on drop volume, and the potential for smaller drop volume in generic medications means patients could receive less of the active ingredient per instillation, potentially resulting in a lower daily prescribed dose.[11] This lack of dose consistency can be concerning for the long-term management of glaucoma patients.

Generic medications also have different names and come in different packages, which can be confusing for patients, especially for those with low health care literacy.[11] Such patients have poor compliance with glaucoma treatment and worse visual field results on follow up examination.[12] Thus, for certain patients the variability in generic medications may compound with existing confusion from a multi-drop regimen, resulting in worse compliance. In these cases, the consistency of a brand-name medication, despite cost issues, may be valuable in potentially improving compliance and ultimately effectiveness of treatment.

Adverse reactions. One of the main goals of glaucoma treatment is to maximize the IOP-lowering effect while limiting adverse reactions. Most preservatives act as surfactants, which destabilize bacterial cell membranes and result in the destruction of the cell membrane, inhibition of cell growth and reduction of cell adhesiveness. Preservatives also exert this effect on corneal and conjunctival cells. This may result in ocular surface disorders, including superficial punctate keratitis, corneal erosion, conjunctival allergy, conjunctival injection and anterior chamber reaction.[5] Patients who are exhibiting a toxic reaction to a medication or cannot tolerate the medication could benefit from a preservative-free version.

Another common ocular adverse reaction in glaucoma patients is follicular conjunctivitis secondary to an alpha-2 adrenergic agonist such as brimonidine.[5] If the patient exhibits a follicular response, hyperemia or simply does not tolerate this class of medication, an alternative class is indicated. Additionally, for patients with more severe glaucoma or who may require surgical intervention with filtration surgery, consider avoiding alpha-2 agonists, as they can decrease conjunctival mobility and make filtration surgeries more difficult secondary to the chronic follicular conjunctivitis.

New Horizons

For the first time in more than 20 years we now have two new classes of glaucoma medications available to us. Vyzulta (latanoprostene bunod 0.024%, Bausch + Lomb) is metabolized into latanoprost and donates nitric oxide once the medication is in the eye—targeting both uveoscleral and trabecular outflow.[12] Research shows Vyzulta has a safe systemic profile, with the

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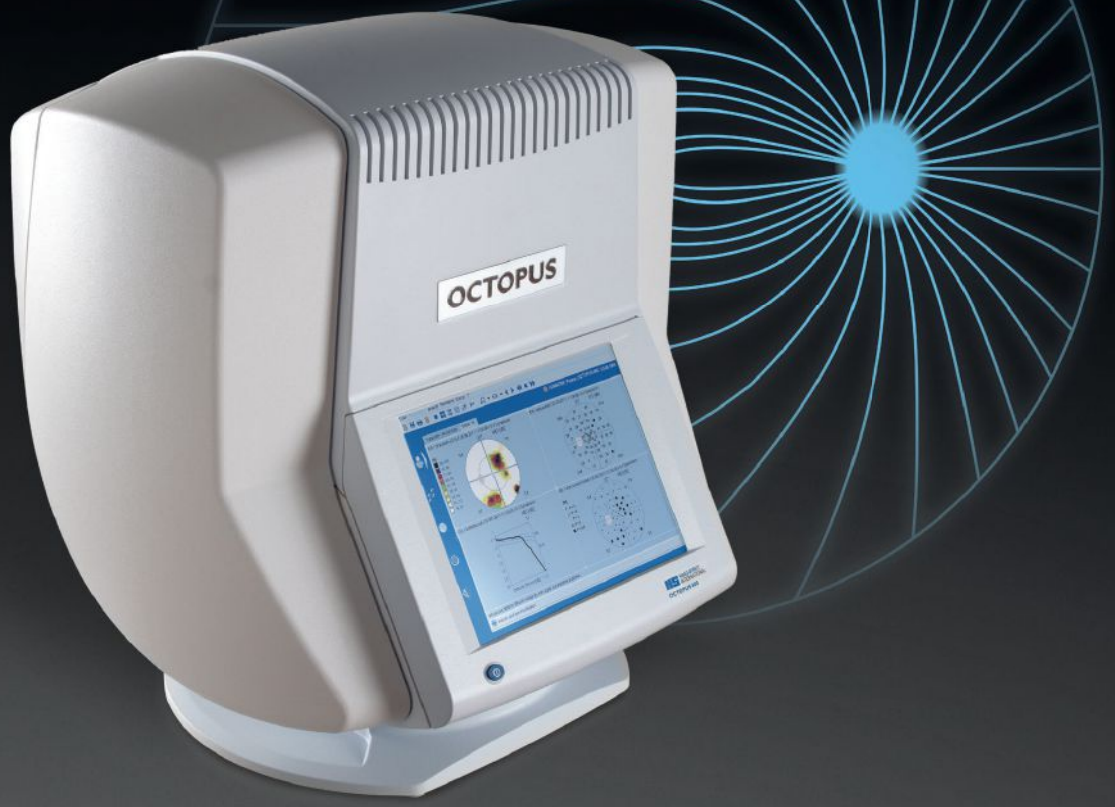

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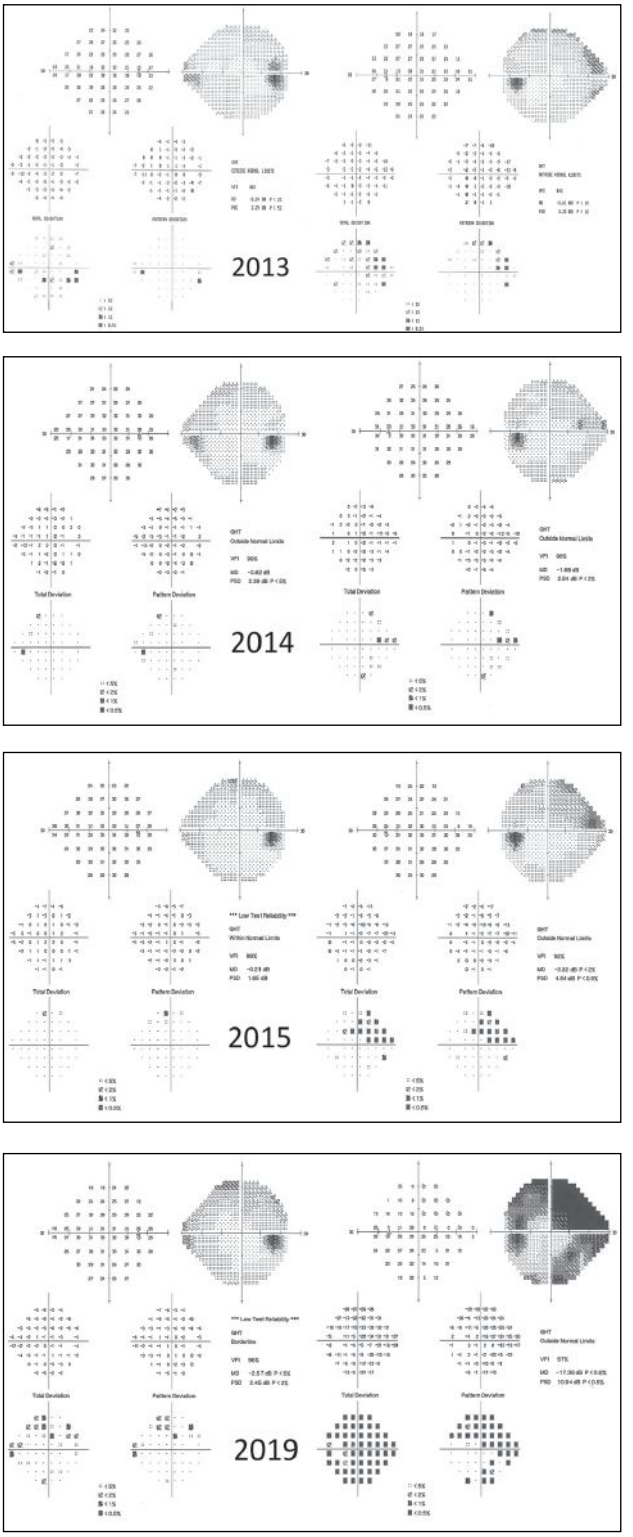

Fig. 2. This 59-year-old male was lost to follow up for four years, during which time he discontinued his drops. His visual fields demonstrate progression. Patients such as this might benefit from surgical intervention rather than topical therapy.

most common reported ocular side effect being hyperemia.[12] The medication has also demonstrated a robust IOP-lowering effect of 9mm Hg.[13] This considerable effect suggests Vyzulta could potentially be used as first-line therapy.

The other new class of topical IOP-lowering medication is the aforementioned Rhopressa, a rho-kinase inhibitor that acts to increase trabecular outflow.[12] This drug class has a safe systemic profile but comes with two unique ocular side effects: small conjunctival hemorrhages and corneal verticillata.[13] These side effects usually resolve upon discontinuing the medication and typically do not have any impact on the patient's vision. Rhopressa had an IOP-lowering effect similar to that of timolol, and research shows it's non-inferior to timolol, making it a suitable first- or second-line adjunct therapy.[12]

Rocklatan (netarsudil 0.02%/latanoprost 0.005% ophthalmic solution, Aerie) is another new topical glaucoma therapy. Clinical trials demonstrate that the fixed combination is more effective at lowering IOP than either medication alone. The once-daily dosing and demonstrated success in lowering IOP suggest that Rocklatan may be a good first-line therapy or a beneficial medication to consider in patients with complex dosing regiments.[14] The same risk/benefit considerations should be given when prescribing these medications as to other topical glaucoma medications.

Surgical Intervention

Minimally invasive glaucoma surgeries (MIGS) are generally ab interno, micro-incisional, conjunctiva-sparing procedures that prioritize patient safety and have a demonstrated efficacy in lowering IOP.[15] MIGS offer a less-invasive option for lowering IOP than other traditional filtering and shunt surgeries.

These devices fall into three main implant categories: (1) increase trabecular outflow by bypassing the juxtacanalicular trabecular meshwork, (2) increase uveoscleral outflow via suprachoroidal pathways or (3) create a subconjunctival drainage pathway.[15] Research shows MIGS can provide IOP control in the mid to low teens while reducing the need for topical hypotensive agents.[15] Optometrists should consider referring patients with mild to moderate POAG and cataracts to surgeons who are performing MIGS procedures.

Clinicians should also consider consultation for surgical intervention in patients who are progressing despite topical therapy with two or three medications, have fixation-threatening visual fields, cannot tolerate topical medications secondary to ocular or systemic

side effects and for whom the use of regular topical therapy is not realistic secondary to their social situation or comorbid conditions (*Figure 2*).[16]

Glaucoma management requires a dynamic approach that carefully weighs the risks and benefits of a particular therapy. Each patient's status will likely change over time, requiring treatment adjustments along the way. A careful assessment of both structural and functional changes as well as potential adverse reactions, costs and the burden of treatment can help you determine when, and how, to escalate treatment.

There is no one-size-fits-all treatment regimen for glaucoma. Rather, changing a patient's glaucoma therapy is a complex art involving careful thought and consideration of the patient's glaucoma status, response to treatment, indicators of progression, medication cost, adverse reactions and the patient's quality of life. ■

Dr. Mackner completed his residency in ocular disease and surgical comanagement at Omni Eye Services in New York and New Jersey. He currently practices at Edina Eye Physicians and Surgeons in Minnesota.

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PEER • REVIEWED • PRA • ARTICLE

How—and Why—to Choose Dry Eye Drugs

Properly pairing a patient with a pharmaceutical requires an understanding of the individual's underlying condition and overall health. By Rachel Grant, OD

For patients with dry eye disease (DED), treatment options were once confined to artificial tears, warm compresses and the occasional off-label steroid. While some of these help soothe symptoms, they do little to truly manage the disease, especially as it worsens. For patients no longer treatable with these at-home remedies, new research shows a cascade of discoveries in both diagnosis and treatment.

Today, optometrists can treat these patients with prescription topical and oral anti-inflammatory medications, point-of-care treatments, heating and expression of glands and even advanced options such as amniotic membranes. While these advances are giving our patients a new lease on ocular comfort, optometrists have much more to navigate. Applying the most appropriate treatment still varies widely within optometric practices.[3]

While no clear consensus exists on prescribing dry eye drugs, understanding how these medications work can help doctors tailor effective management for each patient.

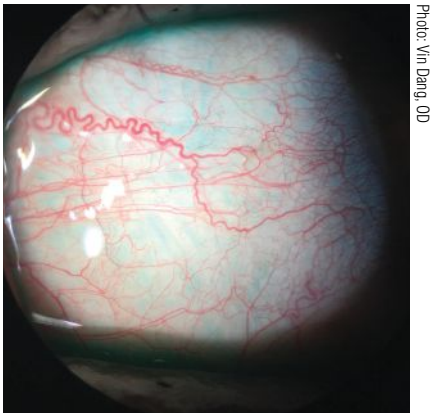

Photo: Vin Dang, OD

This patient's inflamed blood vessels can be seen without any vital dyes.

The Loss of Homeostasis

When it comes to treating DED patients, timely and effective options can be complicated by the need to accurately detect and identify underlying etiologies. In 2017, the Tear Film and Ocular Surface Society (TFOS) shed new light on DED with its second Dry Eye Workshop report (DEWS II). That research recasts DED as a multifactorial condition characterized by a loss of homeostasis of the tear film and accompanied by ocular symptoms that lead to tear film instability and hyperosmolarity.[1] This turned on its head the old paradigm of categorizing DED as either aqueous-deficient dry eye (ADDE) or evaporative dry eye (EDE). With the DEWS II's conclusions, these are no longer considered completely separate ocular surface conditions, as researchers have found up to 70% of DED patients have mixed etiologies.[2] At the forefront of that loss of homeostasis is inflammation.

The TFOS DEWS II report also taught us "in DED, tear hyperosmolarity is considered to set up a cascade of signaling events within surface epithelial cells, that leads to the release of inflammatory mediators and proteases. Such mediators, together with the tear hyperosmolarity itself, are conceived to cause goblet cell and epithelial cell loss and damage to the epithelial glycocalyx. Damage is reinforced by inflammatory mediators from activated T-cells, recruited to the ocular surface. The net result is the characteristic punctate epithe-

liopathy of DED and a tear film instability which leads at some point to early tear film break-up. This break-up exacerbates and amplifies tear hyperosmolarity and completes the vicious circle of events that lead to ocular surface damage."[1] This vicious inflammatory circle is a common pathway that all forms of DED enter, regardless of etiology.

Inflammation

On the ocular surface, the acute phase of inflammation results in the degranulation of mast cells and the subsequent release of histamine and phospholipids. The inflammatory stimuli also initiates the degradation of the cell membrane via phospholipase A_2 and subsequently leads to the formation of arachidonic acid (AA). The latter is metabolized by 5-lipoxygenase (LOX) and cyclooxygenase isoenzymes (COX-1/COX-2), resulting in the recruitment of white blood cells and the formation of prostaglandins and thromboxane A_2, respectively.[4,5] Prostaglandins play a role in pain response and increase vessel permeability.

One of the many impacts of this cascade is the production and release of inflammatory cytokines such as interleukin-1 (IL-1), tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6) and matrix metalloproteinases (MMPs) by the ocular surface epithelial cells.[1,5] This results in the activation of antigen-presenting cells and increased expression of adhesion molecules such as intercellular adhesion molecule-1 (ICAM-1) and selectins by the conjunctival vascular endothelium.[5] This facilitates recruitment of additional inflammatory cells to the ocular surface.

Chronic inflammation involves the processing of antigens by ocular antigen-presenting cells and naive T-cells. Primed CD-4 T-cells adhere to activated vascular endothelium and enter the ocular tissue. Cytokines produced by activated T-cells, such as interferon gamma (IFN-Y), amplify the immune response by increasing adhesion molecule expression by ocular blood vessels.[5] Arachidonic acid metabolites such as leukotrienes and prostaglandins are actively involved in the development of inflammatory disease.[4]

Recognizing the significance and complexity of inflammation in DED gives us the opportunity to identify gaps or overlap in treatment, provided we can identify where each prescription fits along the cascade.

Corticosteroids

These topical meds stimulate the production of a glycoprotein called lipocortin. Formed lipocortin inhibits the activity of phospholipase A2, which releases arachidonic acid and ultimately results in the formation of prostaglandins and thromboxane A2.[6] Steroids also inhibit the formation of IL-1, ICAM-1, MMPs and cytokines.[6] These actions produce anti-inflammatory and immunosuppressive effects on a localized level as steroids block both the LOX and COX-1/COX-2 pathways of the inflammatory cascade, reducing vasodilation, vascular permeability and stabilizing cellular membranes.[7]

Clinical trials show significant improvements for patients with moderate to severe ADDE, with improvements in corneal staining and general injection.[6] As a result, these medications are often highly effective short-term anti-inflammatory options for patients with ocular surface inflammation.

Choosing the most appropriate corticosteroid for your patient requires knowledge of both the medication's mechanism of action and your particular patient. From a clinical standpoint, loteprednol has numerous ophthalmic indications and also comes in a wide array of

Table 1. Anti-Inflammatory Drugs for Dry Eye Disease

Brand Name	Generic Name	Manufacturer	Preparation	Additional Information
Lotemax SM	Loteprednol etabonate 0.38%	B+L	Gel drops	For steroid responders and glaucoma patients; 0.38% approved for TID, 0.5% approved for QID
Lotemax gel	Loteprednol etabonate 0.5%	B+L	Gel drops	
Lotemax ointment	Loteprednol etabonate 0.5%	B+L	Ointment	
Inveltys	Loteprednol etabonate 1%	Kala	Suspension	BID; for post-op inflammation/pain
Pred Forte	Prednisolone acetate 1%	Allergan	Suspension	Shake well before use, consider prescribing name brand
Alrex	Loteprednol etabonate 0.2%	B+L	Suspension	
FML	Fluorometholone alcohol 0.1%	Allergan	Suspension	
Restasis	Cyclosporine 0.05%	Allergan	Emulsion	BID dosing, may consider short-term topical steroid in conjunction
Cequa	Cyclosporine 0.09%	Kala	Emulsion	BID dosing
Xiidra	Lifitegrast 5%	Novartis	Solution	BID dosing
Doxycycline/ minocycline		Generic	Oral	50mg to 100mg once or twice a day or 20mg long-term
Azithromycin	Azithromycin	Generic	Oral	For MGD and rosacea
Azasite	Azithromycin 1%	Akorn	Solution	Off-label for MGD/lid disease

Table 2. The ABCs of Inflammation

Innate Defenses	General Function
Collectins	Proteins that protect against bacteria, yeast and some viruses
Complement	Group of proteins in plasma and other body fluids that stimulates inflammation, attract phagocytes and enhances phagocytosis
Defensins	Peptides produced by neutrophils and other granulocytes; cripple microbes by cell membrane/wall deterioration
Natural killer cells	Small population of lymphocytes that enhances inflammation
B-cells (B-lymphocytes)	Provide humoral immune response, interact indirectly, producing antibodies
T-cells (T lymphocytes)	Provide cellular immune response, interact directly with antigens or antigen-bearing agents to destroy them, secrete cytokines
Cytokines	Enhance cellular response to antigens
Colony-stimulating factors	Type of cytokine; stimulate bone marrow to produce lymphocytes
Interferons	Type of cytokine; block viral replication, stimulate macrophages to engulf viruses, stimulate B cells to produce antibodies, attack cancer cells
Interleukins	Type of cytokine; control lymphocyte differentiation and proliferation
Tumor necrosis factor	Type of cytokine; stops tumor growth, releases growth factors, causes fever that accompanies bacterial infection, stimulates lymphocyte differentiation
Adhesion molecules	Cell surface proteins that mediate the interaction between cells and the extracellular matrix; cell adhesion molecules, such as selectins, integrins and immunoglobulin gene adhesion receptors, mediate the different steps of leukocyte migration in inflammation

formulations, making it a flexible option for practitioners.[8]

New versions of topical drug delivery for loteprednol also adds to the appeal. Lotemax SM gel (loteprednol 0.38%, Bausch + Lomb) uses submicron particles to enhance drug dissolution in the tears, which increases the transcorneal penetration compared with Lotemax 0.5% gel drops and enables greater adherence to the ocular surface.[9] Furthermore, Lotemax SM is also preserved with a low dose of benzalkonium chloride (BAK) and at TID dosing, reducing the burden on an already compromised ocular surface. For this same reason, in addition to fewer unwanted side effects (elevated IOP and subcapsular cataracts), Lotemax SM is a good short-term anti-inflammatory for patients suffering from concomitant DED and glaucoma.

While not FDA approved for DED management, it has been used as an off-label treatment for many dry eye patients based on the consensus of peer-reviewed professional literature.

Another loteprednol option is Inveltys (loteprednol etabonate suspension 1%, Kala), which attaches loteprednol to a mucus-penetrating nanoparticle to improve penetration and concentration of the drug to ocular tissue.[10] Lastly, although not yet FDA approved, is another loteprednol option, KPI-121 0.25% (loteprednol etabonate suspension 0.25%, Kala), that would specifically be approved for signs and symptoms of DED.[11]

Outside of loteprednol, many alternative options for the treatment of ocular surface inflammation exist, including FML (fluorometholone ophthalmic suspension 0.25%, 0.1%, Allergan) or prednisolone acetate 0.12%. These are available in generic formulations and can be effective in DED management, especially as they cost less than branded counterparts.

Cyclosporine Immunomodulators

Restasis (cyclosporine A 0.05% emulsion, Allergan) is an immunomodulator that acts on T-cells in the tear film, conjunctiva and cornea, and has been available for ophthalmic use since 2003.[12] Cyclosporine A (CsA) suppresses inflammation by binding to the protein cyclophilin, which ultimately results in reducing interleukin-2 (IL-2) formulation and suppressing T-cell activation. lL-2 is secreted by T-helper cells and stimulates proliferation of cytotoxic T-cells and additional T-helper cells.[12,13]

As CsA halts further activation of T-cells, but does not target already active T-cells, there may not be an immediate improvement in DED signs. For this reason, clinicians often prescribe a short dose of corticosteroids upon the initiation of Restasis. An additional source of patient noncompliance arises from the vehicle of drug delivery. CsA 0.05% requires suspension in an emulsifying agent, such as glycerin or castor oil, as it has poor water solubility on its own. This suspension contributes to some of the side effects such as burning stinging and hyperemia.[13]

Cequa (cyclosporine 0.09% ophthalmic solution, Sun Ophthalmics) was approved by the FDA in 2018 and recently entered the ophthal-

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1 McLaurin E, Cavet ME, Gomes PJ, Ciolino JB. Brimonidine ophthalmic solution 0.025% for reduction of ocular redness: A randomized clinical trial. Optom Vis Sci. 2018;95(3):264-271

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mic market.[14] Dosing with nanomicellar technology (Ncell, Sun Ophthalmics) BID to avoid the solubility issues with CsA 0.05%, Cequa aims to provide the eye with the highest concentration of CsA on the market.[14] Nanomicelles possess both hydrophilic and hydrophobic properties, which enhances their ability to effectively penetrate the ocular epithelium with minimal irritation or drug degradation.[14] With a similar side effect profile to Restasis, it will have to be seen how patient compliance responds to the higher concentration level of cyclosporine and if concomitant steroid use will be required for effective relief of symptoms.

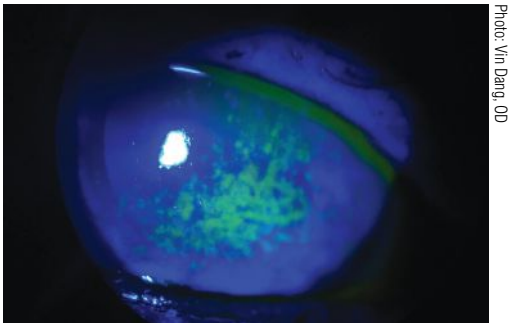

Photo: Vin Dang, OD

Sodium fluorescein staining shows this patient's corneal surface is compromised by dry eye disease.

Other Immunomodulators

Xiidra (lifitegrast ophthalmic solution 5%, Novartis) entered the market in 2016 and is currently the only approved treatment for both signs and symptoms of DED.[15] Similar to cyclosporine, Xiidra also inhibits the T-cell mediated inflammatory pathway by preventing recruitment and activation to ocular surface. However, Xiidra works by blocking the interaction between lymphocyte function-associated antigen 1 (LFA-1) and intercellular adhesion molecule 1 (ICAM-1).[16]

Blocking this interaction of LFA-1on T-cells and ICAM-1 reduces T-cell activation and migration from the blood vessel to the ocular surface, as well as the secretion of multiple pro-inflammatory cytokines (e.g., IL-1, TNF-α, IFN-Y), reducing inflammation.[17] Clinical trials of lifitegrast demonstrated statistically significant improvement in signs and symptoms of DED.[17] With the preservative-free dosing, the most common side effects are burning, blur upon instillation and dysgeusia. From a pharmacology standpoint, the inflammatory mediators are still present on the ocular surface when Xiidra is initiated, and a steroid could still be employed to enhance patient comfort. That being said, many patients are successfully managed with Xiidra as the first-line or stand-alone treatment.

Antibiotics

Oral tetracycline and tetracycline derivatives (e.g., doxycycline, minocycline) can be used to treat DED associated conditions such as rosacea, blepharitis and meibomian gland dysfunction. These broad spectrum antibiotics regulate lipids and inhibit bacterial protein synthesis in addition to having anti-inflammatory properties.2 Research shows they can decrease MMP and phospholipase A2 activity, as well as reduce the production of inflammatory mediators like IL-1 and TNF-α, resulting in reduced irritation and improved tear film stability.[2] These features make them an attractive option for short-term management of ocular inflammation or on a long-term basis at a lower dose, with minimal side effects.[2]

Another antibiotic option for management is oral azithromycin, as the anti-inflammatory properties help control both lid inflammation by inhibiting pro-inflammatory cytokines and bacterial flora.[2,18] While no universal agreement on dosing exists, a shorter course of treatment using 250mg to 500mg over five days can be efficacious in rosacea management.[18] When it comes to making a prescribing decision for an oral treatment, consider azithromycin as an initial option, unless otherwise contraindicated. A study released in 2019 demonstrated that oral azithromycin efficacy was superior to oral doxycycline for treatment of meibomian gland dysfunction when considering dosing and duration.[19]

Azasite (azithromycin ophthalmic solution 1%, Akorn) was FDA approved for bacterial conjunctivitis, but has been well-tolerated for off-label treatment of meibomian gland dysfunction and EDE. In addition to the decrease in inflammatory mediators and suppression of pro-inflammatory mediators, research shows that topical azithromycin can improve lipid behaviors of meibomian gland secretions.[20] No research yet shows any benefit to combining systemic and topical antibiotics to improve DED treatment.

As recent research shows, DED is complex and challenging. Before ODs can begin to manage it, they require a clear diagnostic understanding of the individual patient's ocular surface. This means identifying any concurrent ocular conditions as well as systemic factors that may exacerbate disease.

When determining treatment, clinicians must consider osmolarity, inflammation, treatment history and ocular surface staining. That being said, using these indicators, optometrists should prescribe dry

eye therapy that includes specific targeting along the inflammatory cascade, especially early on in disease presentation. In addition, approaching treatment and management with flexibility may serve these patients more in the long run. Although off-label options, consider TID or QID CsA dosing, prescribing concomitant anti-inflammatory treatments such as both Restasis and Xiidra, or a CsA treatment along with oral antibiotics to effectively treat and manage patient signs and symptoms. Using multiple therapies and modalities to address symptoms early on may provide that extra advantage.

Ultimately, educating patients and emphasizing the importance of lifelong management for DED is essential. There will be good days and bad days, as well as better and worse seasons of DED, and management modifications are not always setbacks in the disease course. ■

Dr. Grant is a clinical instructor at the Southern College of Optometry in Memphis, Tenn.

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PEER • REVIEWED • PRA • ARTICLE

THE DO'S & DON'TS OF ORAL MEDICATION

Understanding when and how to prescribe these essential agents will help optometrists provide optimal patient care.

By Jane Ann Grogg, OD

The use of oral therapies in optometry broadens our scope of practice and provides more comprehensive care for our patients. While topical drugs treat a wide variety of ophthalmic disorders, we need to recognize when oral medications are a better option. Familiarize yourself with clinical scenarios when orals are indicated and the proper dosing schedule.

Choosing effective medication is important to ensure prompt and adequate resolution of a patient's presenting problem. You can select medications with fewer side effects and familiarize yourself with common adverse events to help with compliance. Patients who know what to expect will be less likely to call the office with concerns or stop taking the medication because they were surprised by one of its side effects. Discuss the possibilities ahead of time to avoid these pitfalls.

Cost is always a potential issue for patients. While this cannot be a deterring factor in providing the best care for patients, less expensive generic equivalents, when available, should be considered.

Patient assessment begins with history, which is critical in shaping our differential diagnosis and management. In addition, a good medical history is critical to confirm the necessity of oral medications and to determine the risk of allergic reaction and cross-sensitivities to other medications. From the moment you begin taking the patient's history, your wheels should be turning. A good medical history will inquire about current systemic medical

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

- Describe indications of common oral medications in eye care.
- Prepare for precautions and drug interactions.
- Recognize the importance of taking a good medical history.
- Monitor patients on oral therapy for adverse events.
- Discuss proper prescribing practices.
- Identify when to refer and how to consult with other specialties.

Target Audience: This activity is intended for optometrists engaged in the care of patients requiring oral medications.

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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Disclosure Statements:

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13TH ANNUAL PHARMACEUTICALS REPORT

Oral Medications

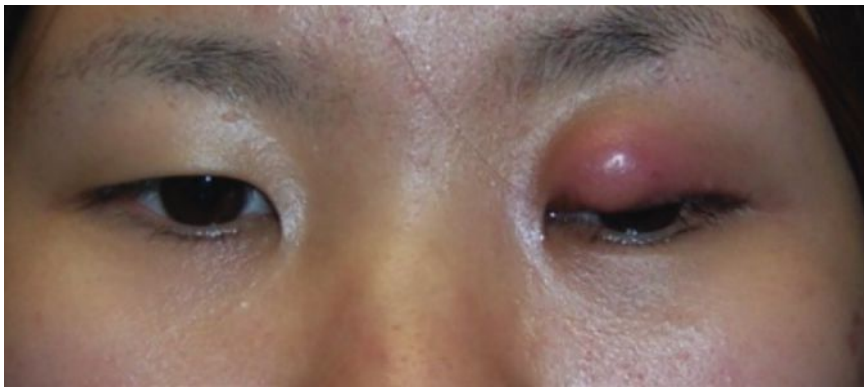

Fig. 1. This patient with preseptal cellulitis from an infected meibomian gland required oral antibiotics.

conditions that could affect liver and kidney function. Oral medications, for the most part, metabolize through either the liver or kidney, and you may have to adjust the dosing depending on the patient's creatinine clearance level or liver disease. Patients must be monitored for the potential for adverse events.

Antibiotic Therapy

When deciding on oral antibiotics, you must know how to choose between the various classes. Common classes of antibiotics include penicillinase-resistant penicillin, broad-spectrum beta lactamase inhibitor, macrolide, cephalosporin, quinolone and tetracycline. Successful use of oral antibiotics requires an option in each class of antibiotics in which you are comfortable and experienced.

A common indication for the use of oral antibiotics is for the treatment of preseptal cellulitis (*Figure 1*). This condition is often the result of infected soft tissue originating from an infected meibomian gland. Warm compresses are always an important adjunctive therapy, but oral antibiotics may be necessary to clear the infection and prevent further complications.

When treating eyelid infections, choosing a penicillinase-resistant penicillin for effective treatment is critical since bacteria produce an enzyme called penicillinase, which renders penicillin ineffective.

Dicloxacillin belongs to the class of penicillinase-resistant penicillins and is listed as a first-line therapy in the *Sanford Guide to Antimicrobial Therapy*.[1] The recommended dosage is 250mg four times a day for severe infection or 125mg four times a day for mild to moderate cases. In general, it is a well-tolerated antibiotic; however, the frequent dosing schedule may make compliance difficult.

Many clinicians prefer antibiotics that can be dosed once or twice a day to improve compliance. Gastrointestinal (GI) upset and the risk of *C. difficile* is listed as an adverse reaction and is a ubiquitous side effect with a variety of oral antibiotics.

Another common antibiotic used in the treatment of soft tissue infection is a broad-spectrum penicillin and beta lactamase inhibitor known as Augmentin (amoxicillin and clavulanic acid, GlaxoSmithKline). Because the beta lactamase inhibitor renders this antibiotic penicillinase-resistant, it can be used in eyelid infections. The dose is based on the amoxicillin component, with a typical dose being 500mg every 12 hours. For more severe infection, the dosing is every eight hours. Using amoxicillin without the clavulanic acid component may result in treatment failure due to the lack of the beta lactamase inhibitor.

Of the cephalosporins, first-generation Keflex (cephalexin, Hikma) is a potential choice for the treatment of preseptal cellulitis as well. A typical dose for a mild case would be 500mg every 12 hours. For more severe infections, this could be dosed up to three times a day.

Allergic cross-sensitivity to cephalosporin products is a listed risk in patients who are allergic to penicillin. This risk is likely overestimated and, for the most part, largely ignored unless the patient has had an anaphylactic reaction.

A true IgE-mediated anaphylactic reaction includes hypotension, laryngeal edema, wheezing, angioedema or urticaria. Sorting out the type of reaction the patient is reporting as an allergic reaction is critical. Too often patients confuse other side effects of the drug (such as stomach upset) with an allergic response. The potential for this cross-sensitivity may be related to the fact that the chemical side chain of certain cephalosporin drugs are

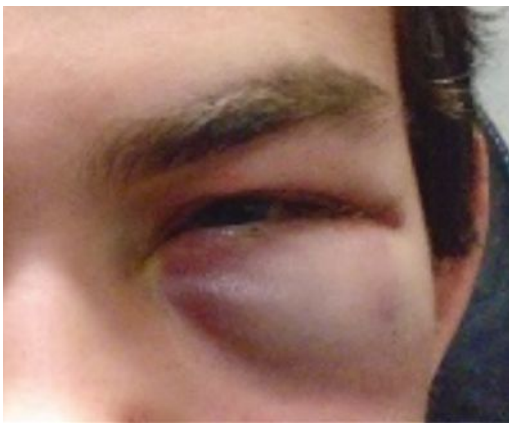

Fig. 2. In this case of an orbital blowout fracture, oral antibiotic therapy was used to prevent the possibility of orbital cellulitis.

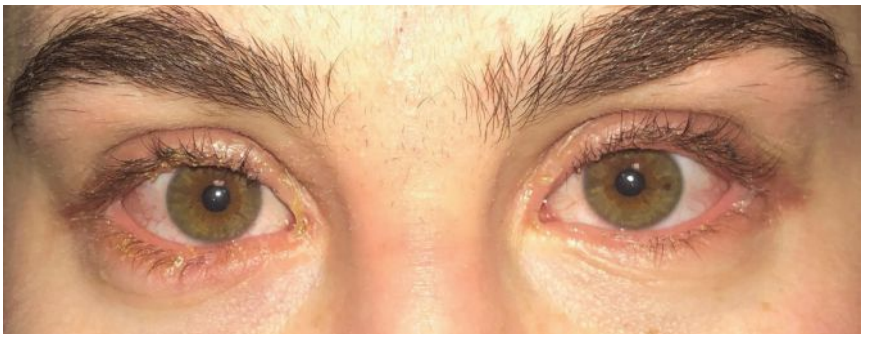

Fig. 3. Resolving right eye herpes simplex lid lesions of the upper and lower eyelid in a patient with eczema who was managed with valacyclovir.

similar to that of penicillin agents.[2]

Generally speaking, second- or third-generation cephalosporin agents with dissimilar side chains from penicillin have a low chance of cross-reactivity in IgE-mediated reactions. Commonly used cephalosporin agents with dissimilar side chains include: Ceftin (cefuroxime, GlaxoSmithKline), Vantin (cefpodoxime, Pfizer), Cefzil (cefprozil, Bristol-Myers Squibb) and Omnicef (cefdinir, AbbVie).

The recommendation is to avoid cephalosporins with similar side chains (first generation such as cephalexin and cefadroxil) if the patient has experienced an anaphylactic reaction to a penicillin. If a patient experiences a non-anaphylactic reaction, the risk of cross-sensitivity and subsequent allergic response may be quite low. However, all cephalosporin drugs will have this listed as a contraindication.

If a patient is allergic to the penicillin and you wish to avoid the cephalosporin class, a macrolide is a good alternative. A commonly prescribed macrolide is azithromycin. This antibiotic is well tolerated and generically available. It has a long half-life, which makes the dosing schedule easy. This, in turn, helps with compliance. It can be prescribed as two 250mg tablets on day one followed by 250mg daily for an additional four days. This was originally known as a "Z-pak." This long-acting antibiotic continues to provide coverage for several additional days despite its five-day short course. There is also a three-day azithromycin package which consists of three 500mg tablets.

Azithromycin is also the treatment of choice for chlamydia infections. The recommended dosage is a 1g dose given as two 500mg tablets.

For patients who have a difficult time swallowing pills, a good option is a 1g powder that can be dissolved in water or juice. A positive chlamydia culture will trigger the Health Department to follow through to ensure proper treatment of the patient as well as their sexual partners. You should also consider referring the patient for other sexually transmitted disease testing as well as safe-sex counseling.

Bactrim (trimethoprim/sulfamethoxazole, Roche) is listed as an alternative for patients with penicillin allergy and is of the sulfonamide class. It is also considered a first-line therapy for methicillin-resistant *Staphylococcus aureus* (MRSA) infections. This antibiotic interacts with oral anticoagulants, hypoglycemics, diuretics and tricyclic antidepressants, to name a few.

Additional adverse reactions include blood dyscrasias, pancreatitis and rash. You should take precautions, consider any interactions and avoid prolonged administration. The double-strength version contains 800mg of sulfamethoxazole and 160mg of trimethoprim. It is dosed as one tablet every 12 hours for a week, depending on the type of infection.

The quinolone class, such as Levaquin (levofloxacin, Janssen), offers an easy dosing schedule of 500mg once daily for seven to 10 days for uncomplicated skin and skin structure infection. However, this class of antibiotics carries a black label warning for tendinitis and tendon rupture as reported in the literature.[3] This risk is in all

Prescribing Best Practices

Proper prescribing practices include specifying the milligram dose of the medication, the number of tablets or capsules (should be equal to the number of days and frequency the patient is taking the medication) and prescription instructions for both the pharmacist and the patient.

While Latin abbreviations are still acceptable, the more common practice is to simply write (or e-prescribe) the prescription longhand to avoid medical mistakes. For instance, QD is often disallowed in the hospital setting because of the common confusion with QID.

You should consider the possibility of pregnancy and lactation in all women of childbearing age. As of 2015, the FDA replaced the assigned risk letter system (categories A, B, C, D and X) with the Pregnancy and Lactation Labeling Final Rule (PLLR) for all prescriptions. This narrative is meant to be more informative and meaningful to both the provider and individual patient. In turn, it allows for better individualized patient-directed counseling. The rule also includes a subsection called Females and Males of Reproductive Potential, which provides information about pregnancy testing, birth control and effect on fertility. Medications submitted to the FDA as of 2015 must use this format. Drugs approved on or after June 30, 2001 will be phased in gradually. Those approved prior to that are not subject to the PLLR rule but will have the letter category removed.

Oral Medications

ages, though patients older than 60 have an increased risk as do those taking corticosteroid and patients with kidney, heart or lung transplants. This class should also be avoided in patients with myasthenia gravis. Given the gravity of these complications, the quinolone class should really be limited to those who have no alternative treatment options.

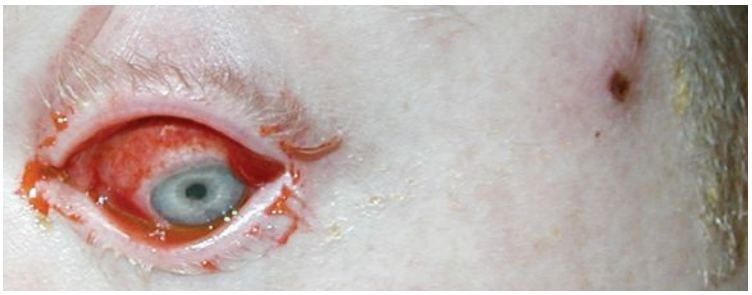

Fig. 4. This patient with culture-positive herpes simplex keratoconjunctivitis of the left eye (note herpetic lesions on temple) was treated with oral antiviral therapy and received a narcotic for pain.

The last class of antibiotics, for the purposes of this article, is the tetracycline class. Within this class, doxycycline is commonly prescribed because of the ease of dosing compared with tetracycline. Doxycycline has fewer reported adverse effects than minocycline, but prescribing may simply come down to doctor preference.[4] Keep in mind the extended release versions of these drugs, while having less GI side effects, can be more expensive.

Tetracycline is often the treatment option for chronic meibomian gland dysfunction (MGD). The available treatment options for patients suffering from chronic MGD have greatly expanded, but sometimes an oral medication for adjunctive treatment is beneficial. Generally speaking, we use doxycycline for its anti-inflammatory properties at lower doses. It may be necessary to use doxycycline for several weeks or months to treat the chronic condition of meibomianitis. Often starting with a dose of 50mg once or twice daily for a couple of weeks can then be maintained at 50mg daily for several weeks. This is typically done with adjunctive therapy such as warm compresses and lid scrubs at a minimum.

Recommending the patient take this medication with food will help ease the GI side effects. In addition, doxycycline can cause a patient to sunburn more easily and can cause esophagus reflux. Advising the patient not to lay down after taking doxycycline may help minimize this side effect.

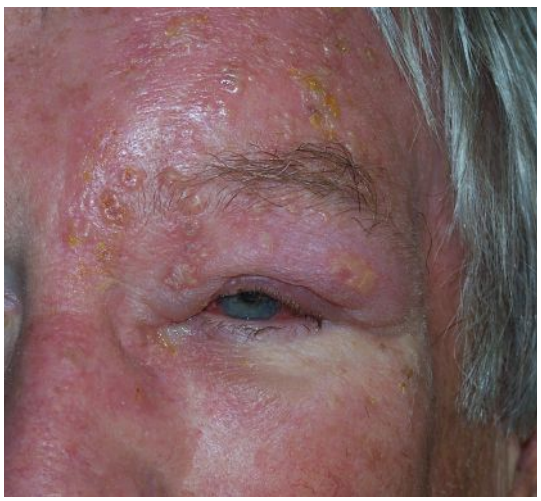

Fig. 5. This patient with herpes zoster ophthalmicus was successfully managed with antiviral therapy.

Doxycycline, in combination with topical corticosteroid, is also known to aid in the prevention of recurrent corneal erosions. Research suggests this works by inhibiting the extracellular matrix metalloproteinases, which are responsible for non-adhesion of the epithelium once traumatized.[5,6]

Current clinical practice guidelines for patients with MRSA recommend increasingly aggressive treatment with increased severity of infection.[7] The list of antibiotics known to be effective in MRSA infection includes trimethoprim-sulfamethoxazole, tetracyclines and clindamycin. Other oral agents that can be considered are oxazolidinones, delafloxacin and the tetracycline omadacycline, but cost can be substancial for these agents.

Other indications for oral antibiotics in optometric practice include dacryocystitis and prophylactic coverage in orbital blowout fractures (*Figure 2*). The latter tends to be controversial. When considering the use of an oral antibiotic prophylactically in orbital blowout fractures, consider consulting with the patient's oculoplastic surgeon.

These are just some of the recommended antibiotics and scenarios to consider when the need for oral antibiotics arises in the optometric setting. You can identify other antibiotic options through resources such as the *Monthly Prescribing Reference* or by consulting infectious causes in the *Sanford Guide to Antimicrobial Therapy*.[1] The Sanford Guide is updated yearly and is beneficial in guiding the standard of care in the medical community.

Antiviral Therapy

This plays a significant role in preventing or minimizing complications related to herpes simplex virus (HSV) and herpes zoster virus. Oral antivirals work to hasten the resolution of these conditions, reduce viral shedding and help prevent the formation of new skin lesions (*Figure 3*). They can help to decrease

both the incidence and severity of ocular complications.

When treating herpes zoster you should recommend therapeutic treatment within 72 hours of vesicles erupting. Instituting treatment within this timeframe not only reduces the duration and severity of the acute pain but may also lessen the risk of progression to post-herpetic neuralgia and other long-term complications and subsequent vision loss.[8-10] Post-herpetic neuralgia occurs as frequently as 50% of the time in patients with herpes zoster opthalmicus.[11]

Herpes simplex lesions may present on the eyelid or at the mucocutaneous border (*Figure 4*). These typically present with localized vesicular lesions that ulcerate and are painful. When the vesicles rupture, you can appreciate a shallow ragged ulcer.

Three commonly used antiviral medications are acyclovir, valacyclovir (the prodrug of acyclovir) and famciclovir. All of these are generically available and reasonably priced. The latter two offer less frequent dosing schedules and may help improve compliance.

For herpes zoster ophthalmicus, the recommended dosing is as follows: acyclovir 800mg five times a day, valacyclovir 1g three times a day and famciclovir 500mg three times a day. An easy way to remember the dosage for herpes simplex virus is to simply divide the zoster dose in half (*Figure 5*).

Oral antiviral therapy is also an effective approach for herpetic keratitis. Practitioners may choose this avenue of treatment if topical antiviral medication is not readily available or not affordable. In fact, oral antiviral therapy is the treatment of choice for children.[12]

Oral antiviral therapy is used to suppress the recurrence of herpetic disease according to the Herpetic Eye Disease study. The recurrence rate of any form of ocular HSV infection was reduced by 41%, and a 50% reduction in the rate of recurrence of stromal keratitis was observed.[13] The study used a dose of acyclovir 400mg twice a day. Valacyclovir is equally as effective at suppressing the immune system with a dose of 500mg twice a day.[14]

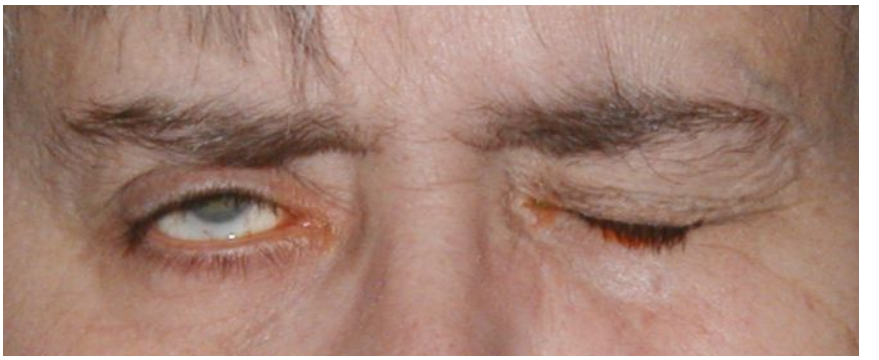

Fig. 6. This patient's cranial nerve VII palsy, presumed to be caused by herpes simplex, was managed with prednisone and antiviral therapy.

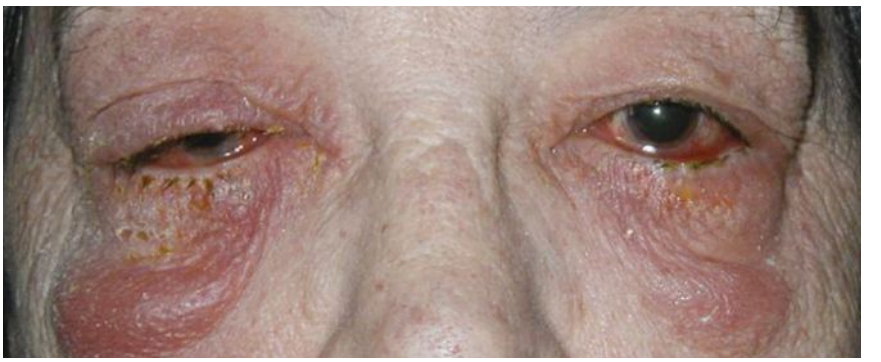

Fig. 7. Oral prednisone was used to manage this patient with contact dermatitis.

HSV has been implicated in, among others, cranial nerve VII paresis with studies indicating a likely association (*Figure 6*).[15-17] The *Sanford Guide* suggests treating Bell's palsy with oral prednisone and oral antiviral therapy if no other causative condition is suspected.[1] The addition of 60mg to 80mg of prednisone with a 20mg taper over several days is recommended if diagnosed within 72 hours of onset.

Overall, oral antiviral therapy is generally well tolerated by patients, as the drug is site-specific. Some side effects may include GI disturbance, headache, vertigo, malaise and central nervous system (CNS) disturbances (especially in the elderly).

Steroids

These play a role in the management of many ocular diseases, including contact dermatitis, eczema, temporal arteritis, orbital inflammatory pseudotumor, Bell's palsy, Graves' disease, orbital floor fractures, optic neuritis and chronic uveitis (*Figure 7*). The only true contraindications for oral steroid use are systemic fungal infection, live vaccinations and drug hypersensitivity.

However, you should be cautious when prescribing them in the presence of peptic ulcer disease, diabetes, tuberculosis, active infection, psychosis and pregnancy. Knowing what you are treating while not disseminating infection or masking pain is critical.

13TH ANNUAL PHARMACEUTICALS REPORT

Oral Medications

Side effects of oral steroids include hyperglycemia, hypokalemia, hypertension, peptic ulcer, increased intraocular pressure, cataract, benign increased intracranial hypertension, mental status changes, osteoporosis, decreased wound healing and fluid retention, to name a few. You can add a proton pump inhibitor with oral prednisone to minimize the GI side effects.

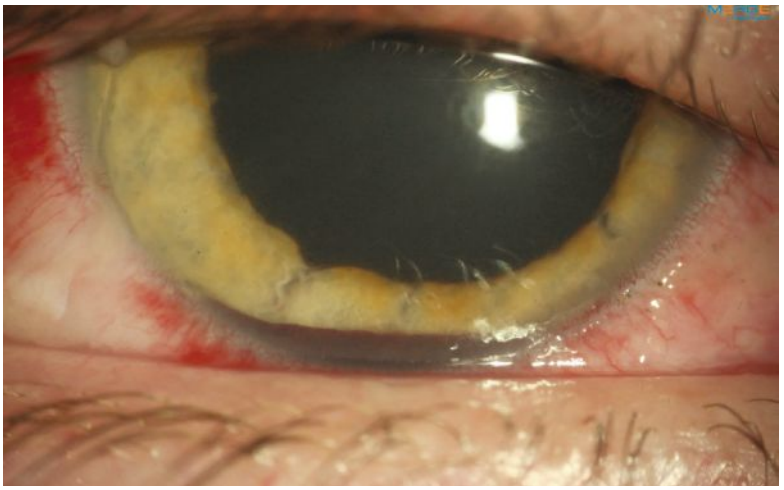

Fig. 8. This patient who had blunt trauma with hyphema, corneal and conjunctival abrasions, anterior uveitis and iris sphincter tears received an adjunctive oral narcotic for three days.

Pain Medications

Occasionally acute care management requires oral pain medications when topical therapy is not adequate. The scope of our management runs the gamut of palliative agents from artificial tears and cold compresses to prescription opioids, depending on the individual state laws. You must document the quality and extent of the patient's pain and pair that with the clinical picture to make judgement calls on their medical need. It is essential to identify the source of the pain, manage the patient effectively and treat the causative problem to avoid masking pain.

Corneal problems are the most common source of ocular pain and often the most intense because of the cornea's high density of nerves, especially compared with its neighbor, the conjunctiva. When the eye is traumatized, the patient often experiences eyelid edema, tearing and photophobia. Secondary anterior uveitis may develop in these patients as an additional source of discomfort and inflammation.

Oral pain medication options can be divided into non-narcotic and narcotic medications. Non-narcotic options include analgesics, non-steroidal anti-inflammatory drugs (NSAIDs) and COX-2 inhibitors. A common over-the-counter analgesic is acetaminophen. It is available in multiple dosages, starting at 325mg with an extra-strength version of 500mg. This can be dosed every four to six hours for a maximum of 3g daily. In 2011, the FDA asked drug manufacturers to limit the strength to 325mg tablets and to reduced the maximum recommended amount to 3g per day from the previous 4g per day.

Acetaminophen is contraindicated in liver disease, alcoholism and acetaminophen hypersensitivity. It is commonly paired with opioids to potentiate their effect, improve efficacy of the opioid and allow less narcotic to be prescribed.

Oral NSAIDs are contraindicated in aspirin allergies and should be used with precaution in active peptic ulcer or GI disease, renal or liver impairment, heart failure, edema and hypertension. Adverse reactions include gastrointestinal ulcer/bleeding/upset, headache, dizziness, fluid retention, rash, pruritus and tinnitus. Asthma patients have an increased risk of allergy to NSAIDs.

When used at lower doses, NSAIDs have pain management benefits; at higher doses they have anti-inflammatory benefits. Oral NSAIDs may be used in ocular surface injuries, moderate to severe episcleritis, mild scleritis and uveitis. Ibuprofen is dosed 200mg to 800mg every four hours for a maximum prescription dose of 3,200mg daily; however, the side effect profile is better if the maximum dose is limited to 1,600mg daily.

Naproxen, available over the counter, is given in 220mg tablets. The initial dose is two tablets, followed by one tablet every eight to 12 hours for a maximum of three tablets in 24 hours. Prescription-strength dosage is available in 250mg, 375mg and 500mg, though using the lowest effective dose for the shortest duration is advisable.

NSAIDs carry a black box warning of increased risk of serious and potentially fatal cardiovascular thrombotic events, including myocardial infarction and stroke, which may occur early in the treatment cycle and may increase with duration of use. There is also a black box warning of serious and potentially fatal GI adverse events including bleeding, ulcer and stomach or intestinal perforation.

Alternating acetaminophen with ibuprofen every two hours is often an effective and inexpensive means of managing mild to moderate pain. These medications, which employ two different pathways for pain

management, are typically readily available and most patients already have these medications in their medicine cabinets.

Narcotics in acute pain management are reserved for when topical management is not adequate or contraindicated (*Figure 8*). Narcotics range from schedule I to V (more to less addictive). Most are prescribed as a ratio of the amount of narcotic and the amount of acetaminophen in each tablet. For example, hydrocodone/acetaminophen 5/500 contains 5mg of hydrocodone bitartrate and 500mg of acetaminophen with a recommended dose of one or two tablets every four to six hours as needed for pain.

Ultram (tramadol, Johnson & Johnson) is a schedule IV narcotic with an adult dose of 50mg to 100mg every four to six hours as needed for moderate and moderate-severe pain. Alternatively, Ultracet (acetaminophen/tramadol, Janssen Pharmaceuticals) contains 325mg of acetaminophen with a slightly lower dose of tramadol (37.5mg). It is dosed two tablets every four to six hours with a maximum of eight tablets per day for up to five days.

With all centrally acting agents, precaution should be used in patients with a history of seizure disorders, head injury or respiratory depression. Adverse reactions may include dizziness, nausea, constipation, headache, somnolence, GI upset, dry mouth, itching and CNS stimulation.

Remember to warn patients of alcohol use or other CNS depressants, as CNS depression is addictive. An example is the combination of benzodiazepines and narcotics, which carry the risk of opioid-related deaths.

Given the current opioid epidemic and widespread opioid abuse, you should prescribe the least amount of narcotic possible to achieve the desired pain relief. Do not prescribe beyond the number of days the pain is severe enough to warrant opioids. Always reevaluate and adjust based on clinical findings. Be vigilant of drug-seeking behavior in patients who claim to have eye pain that is inconsistent with the clinical examination. The use of state-based prescription drug monitory programs helps identify patients at risk of addiction or overdose.

With a good medical history and a careful understanding of each medication's indications, contraindications and potential side effects, you can safely treat patients using any number of oral medications. If the patient does not respond or worsens on treatment don't be afraid to refer.

Ultimately, optometrists must embrace this therapeutic privilege. By doing so, we will be using the privilege that we had to fight to obtain, which is important in the growth of our profession. ■

Dr. Grogg is the director of the Indiana University Health Center Eye Clinic and a clinical professor of Optometry at the Indiana University School of Optometry in Bloomington, Indiana.

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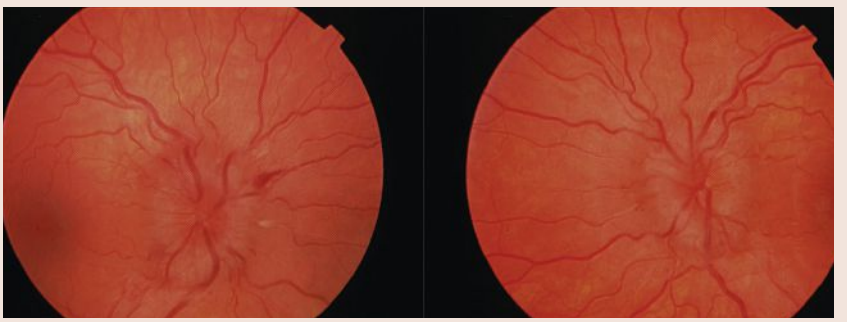
In-office Additions

Diamox (Teva Pharmaceuticals) should be kept in-office for the rare occasion someone needs additional medically lowered intraocular pressures, such as in acute angle-closure. More commonly, we use the extended release version in the management of idiopathic intracranial hypertension. The extended release version tends to be better tolerated from a side effect profile.



Papilledema secondary to idiopathic intracranial hypertension managed with Diamox sequels.

OSC QUIZ

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

You must achieve a score of 70 or higher to receive credit. Allow four weeks for processing. For each Optometric Study Center course you pass, you earn 2 hours of credit from Pennsylvania College of Optometry.

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

1. For the treatment of preseptal cellulitis associated with a hordeolum, which of the following would be an acceptable medication, assuming the patient has no medical allergies or contraindications?
a. Dicloxacillin.
b. Acyclovir.
c. Lortab.
d. Prednisone.

2. Which of these oral medications is a treatment of choice for chronic MGD?
a. Keflex.
b. Doxycycline.
c. Augmentin.
d. Levaquin.

3. Which of the following prescriptions is an appropriate treatment for a patient with acute onset of herpes zoster ophthalmicus?
a. Valacyclovir 1g TID for seven days.
b. Famciclovir 250mg 5x/day for seven days.
c. Acyclovir 400mg 5x/day for seven days.
d. Valacyclovir 500mg BID for seven days.

4. A 25-year-old male presents with a painful, swollen upper eyelid. You note an internal hordeolum with surrounding preseptal cellulitis. He has attempted warm compresses. His medical history is significant for a severe allergic reaction to penicillin as a child in which he describes shortness of breath and hives. Which of these would be an appropriate oral medication to treat this patient?
a. Augmentin.
b. Cephalexin.
c. Azithromycin.
d. Dicloxacillin.

5. Which of the following medications has the most potential for abuse and addiction?
a. Opioids.
b. Acetaminophen.
c. NSAIDs.
d. COX-2 inhibitors.

6. Reactivation of the herpes simplex virus with unilateral facial weakness and incomplete closure of the ipsilateral eyelid most likely involves which cranial nerve?
a. Cranial nerve III paresis.
b. Cranial nerve VI paresis.
c. Cranial nerve IV paresis.
d. Cranial nerve VII paresis.

7. A patient with a four-week history of "pink eye" is culture-positive for chlamydia. Which is the most appropriate treatment?
a. Azithromycin 1g PO for one day.
b. Dicloxacillin 500mg BID PO for seven days.
c. Keflex 250mg QID PO for seven days.
d. Continue topical antimicrobial therapy until resolution.

8. A common adverse reaction to many oral antibiotics is:
a. Fever.
b. Night sweats.
c. Gastrointestinal disturbances.
d. Insomnia.

9. Oral steroids commonly cause which of these adverse reactions in diabetic patients?
a. Hypoglycemia.
b. CNS depression.
c. Constipation.
d. Hyperglycemia.

10. Laryngeal edema, hypotension, wheezing, angioedema and urticaria describe:
a. Common side effects of oral steroids.
b. IgE-mediated anaphylaxis allergic reaction.
c. Chlamydia infection.
d. MRSA infection.

11. Which of these drug classifications has the potential for cross-sensitivity in patients who have a penicillin allergy?
a. Macrolide.
b. Fluoroquinolone.
c. Cephalosporin.
d. Tetracycline.

12. The risk of tendon rupture is a known adverse event for which antibiotic?
a. Fluoroquinolone.
b. Penicillin.
c. Cephalosporin.
d. Aminoglycoside.

13. According to the Herpetic Eye Disease study, the recommended dosage for reduction of recurrence of herpes simplex-related complications is:
a. Acyclovir 800mg 5x/day.
b. Valacyclovir 500mg TID.
c. Famciclovir 250mg TID.
d. Acyclovir 400mg BID.

14. Adverse reaction of narcotic agents includes which of the following?
a. Constipation.
b. CNS depression.
c. GI upset.
d. All the above.

15. Many narcotic agents have which of these medications paired with them?
a. Acetaminophen.
b. Aspirin.
c. Proton pump inhibitor.
d. Prednisone.

16. To lessen the GI side effects of prednisone, which of these can be given in conjunction with the prednisone?
a. Aspirin.
b. Acetaminophen.
c. Proton pump inhibitor.
d. Antihistamine.

17. When prescribing oral medications, which of the following is an important aspect of the medical history to consider?
a. Drug interactions.
b. Drug allergies.
c. Kidney and liver disease.
d. All the above are important considerations.

18. A patient presents with an aspirin allergy. Which of the following is contraindicated?
a. NSAIDs.
b. Prednisone.
c. Acetaminophen.
d. None of the above.

19. Opioid use is contraindicated in which of the following?
a. Diabetes.
b. Hypertension.
c. Acute intoxications.
d. Hypothyroidism.

20. A patient with severe episcleritis not responding to topical steroid eye drops may benefit from which oral medications?
a. Fluoroquinolone.
b. Acetaminophen.
c. NSAID.
d. Narcotic analgesics.

Examination Answer Sheet

The Do's & Don'ts of Oral Medication

Valid for credit through March 15, 2023

Online: This exam can be taken online at revieweducationgroup.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.

Directions: Select one answer for each question in the exam and completely darken the appropriate circle. A minimum score of 70% is required to earn credit.

Mail to: Jobson Healthcare Information, LLC, Attn.: CE Processing, 395 Hudson Street, 3rd Floor New York, New York 10014

Payment: Remit $35 with this exam. Make check payable to Jobson Healthcare Information, LLC.

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Salus University has sponsored the review and approval of this activity.

Processing: There is a four-week processing time for this exam.

Answers to CE exam:

1. Ⓐ Ⓑ Ⓒ Ⓓ
2. Ⓐ Ⓑ Ⓒ Ⓓ
3. Ⓐ Ⓑ Ⓒ Ⓓ
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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. Describe indications of common oral medications in eye care. ① ② ③ ④ ⑤
22. Prepare for precautions and drug interactions. ① ② ③ ④ ⑤
23. Recognize the importance of taking a good medical history. ① ② ③ ④ ⑤
24. Monitor patients on oral therapy for adverse events. ① ② ③ ④ ⑤
25. Discuss proper prescribing practices. ① ② ③ ④ ⑤
26. Identify when to refer and how to consult with other specialties. ① ② ③ ④ ⑤

27. 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.

28. 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): ☐

29. 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: _____

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

ⓐ Very confident ⓑ Somewhat confident ⓒ Unsure ⓓ Not confident

31. 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:

32. Additional comments on this course:

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

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

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

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

Signature _____ Date _____

Lesson 119297 RO-OSC-0320

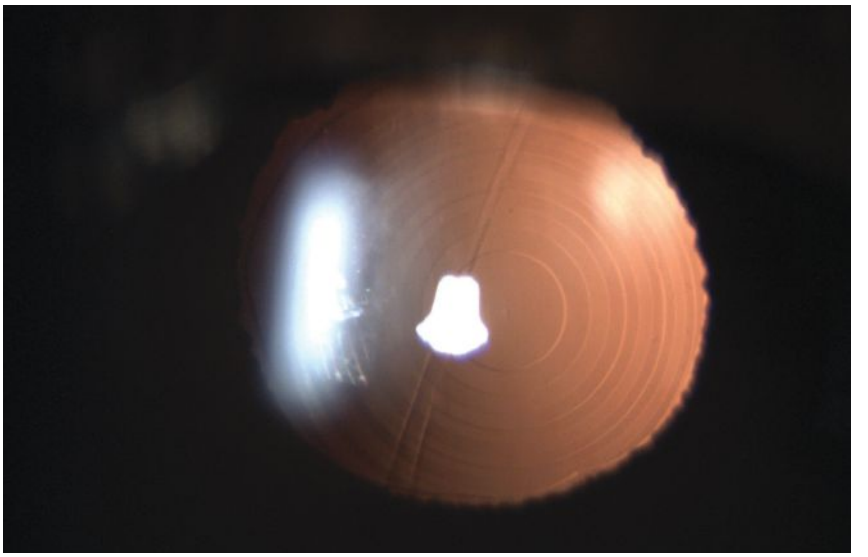
Four Steps to Make Premium IOLs Worth the Cost

Cataract patients have high demands. Here's how clinicians can use today's technologies and techniques to meet them. By Gleb Sukhovolskiy, OD, and Victoria Roan, OD

Modern cataract surgery is perceived as refractive surgery, and expectations are high. These days, patients undergoing cataract surgery live more active lifestyles, spending large amounts of time both outdoors and in front of computers or smartphones. For many, good uncorrected distance vision is not enough anymore, and complete spectacle independence is the goal.

This demand is, in part, fueled by the success stories patients hear from their friends or family members scrapping glasses and having "perfect vision" at all distances. While we don't always see these top-notch outcomes, we are fortunate enough to live in an age when many of those demands can be met.

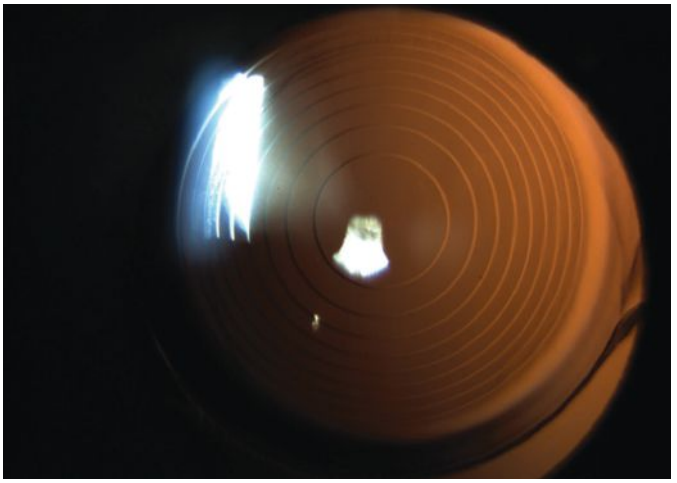
As optometrists take a larger role in postoperative care of cataract patients, it is imperative to stay up-to-date on intraocular lens (IOL) advancements, counseling techniques, pharmaceutical management of the post-op course and more. When careful patient selection and counseling are combined with good surgical skill and knowledgeable postoperative management, most patients should be able to achieve those boast-worthy outcomes that continue driving patients to keep their eyes in top shape.



PanOptix IOL (Alcon), the newest diffractive trifocal IOL, provides good unaided visual acuity at distance, intermediate and near.

Step One: Understand Your Options

It stands to reason that the doctor must have a comprehensive understanding of the technologies and techniques associated with a procedure before they can educate or even evaluate their patient for it. So, the first step to managing cataract patients is to bone up on what's available.



The Symfony IOL is the first extended depth of focus IOL on the market. Its design corrects chromatic aberration and enhances contrast sensitivity.

IOL technology is ever-evolving, with new products entering the market on a regular basis. Since 1997, multifocal IOLs have provided similarly good distance vision as monofocals, but they surpass their predecessor when it comes to intermediate and near vision. However, the multifocal lens design can result in a higher incidence of unwanted visual phenomena such as contrast sensitivity loss, glare and halos.[1-4]

Many patients have benefited greatly from multifocals, but some are extremely dissatisfied with either these unwanted visual phenomena, quality of vision, or higher-than-expected dependency on corrective lenses.[5]

All multifocal IOLs work by separating light into different foci, causing a dispersion of light energy. Different brands use different technologies to achieve this.[6] Today's multifocals function according to one of three different optic principles: refractive, diffractive or extended-depth-of focus.[7]

• ***Refractive multifocals*** use concentric zones of increasing dioptric power on the anterior lens surface with highest power in the center of the lens. These are highly dependent on the patient's pupil size, as the changes in pupil diameter affect the number of zones in use. As pupils decrease in size with the near reflex, the effective power of the lens is increased.[7-8] Early examples of this design include the Array and ReZoom (both originally from AMO, now Johnson & Johnson Vision).

• ***Diffractive multifocals*** possess concentric diffractive surfaces on the posterior portion, inducing wavefront interference. This helps reduce glare and higher-order aberrations.[8] This design does not completely eliminate stray light.[9] About 41% of incident light is allocated to distance focus, 41% to near and 18% is directed to higher-order diffraction, rendering it unusable.[10]

Both refractive and diffractive designs cause an in-focus image to be overlaid by at least one out-of-focus image. This affects contrast and exacerbates haloes and glare.[10]

Examples of diffractive bifocal IOLs include Restor (Alcon) and Tecnis multifocal IOL (Johnson & Johnson Vision).

The newest trifocal, PanOptix (Alcon), also employs a diffractive design, but improves on it to provide better unaided intermediate vision.[11-13] PanOptix uses 88% of light energy (instead of 82% with older diffractive bifocals) and reduced dependence on pupil size.[14]

• *Extended depth-of-focus (EDOF)* IOLs, such as the Tecnis Symfony (Johnson & Johnson Vision), combine a unique diffractive pattern with technology that corrects corneal chromatic aberration, resulting in an elongated depth of focus and enhanced contrast sensitivity.[7,15] An extended continuous focal point is different from two or three peaks of best visual acuity with bifocal or trifocal IOLs and helps reduce overlap of near and far images.[8] This improves visual quality and decreases glare and unwanted visual phenomena.[8,15-17]

EDOF lenses have similar photopic contrast sensitivity to monofocals, much improved from the diffractive-refractive multifocal lens design.[15] Near visual acuity with EDOF lenses may be worse than with diffractive IOLs, but intermediate visual acuity is equal or superior.[16-18] Targeting the nondominant eye for -0.50D myopia may help improve near vision without significantly sacrificing distance.

Symfony IOL provides similar uncorrected distance and intermediate vision as the PanOptix IOL with the same low rate of side effects, but PanOptix offers better uncorrected near vision.[18] There may be less halos and glare with PanOptix than Symfony, but the Symfony IOL may still be better at preserving contrast sensitivity.[14,19]

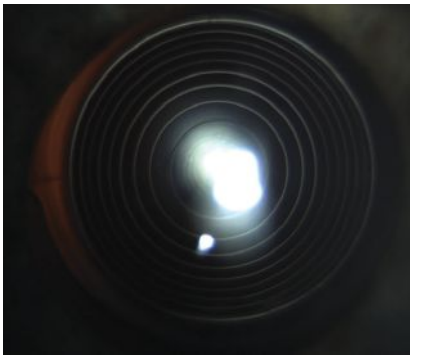
Step Two: Set the Stage

As innovative as these new IOL options are, patients should be properly educated that they aren't perfect—and may not be a perfect match for them. For starters, they'll want to know a large out-of-pocket cost is usually associated with premium IOLs. Be aware that this large price tag may actually elevate their expectations, too. The most important component of post-op success with a premium IOL is preoperative counseling. After all, it is much easier to prevent undesirable outcomes than to fix them after the fact. Implantation of multifocals without discernment or discretion may yield many disgruntled patients. Patients who elect a presbyopia-correcting IOL must be motivated to be spectacle independent. Those who place high value on the improvement in range may be more likely to accept potential small loss in contrast sensitivity or temporary dysphotopsia.

The importance of a thorough discussion with a patient prior to surgery cannot be stressed enough. The conversation should include a careful evaluation of patient's needs, lifestyle and personality. This is where the patient's primary optometrist has an edge over the surgery clinic. The optometrist may already have a relationship and know the patient well, having a better sense of the patient's expectations. Asking about lifestyle, work, hobbies will give information about the types of visual tasks patient performs. Personality assessment may help estimate the ability to neuroadapt.

Patients with unrealistic expectations or an overly critical personality are less likely to fare well with premium IOLs.

Patients' refractive error and current visual acuity should be considered. Hyperopes who have significant cataracts will gain the most



The ring pattern indicates that the patient has a multifocal IOL, though it may be difficult to identify the specific lens based on appearance.

from presbyopia-correcting IOLs, with uncorrected vision improvement at all distances. Mild myopes who rely on their near vision for specific tasks may have something to lose and could be dissatisfied with the result.

About 35% to 40% of eyes undergoing cataract surgery have astigmatism equal to or more than 1.0D and about 20% have astigmatism greater than 1.5D.[20-22] It is of great importance that most of the astigmatism is corrected. Anything more than 0.75D of postoperative cylinder will cause a significant decline in visual quality.[23] Regular corneal astigmatism with good repeatability across measurement devices is ideal and can be reliably corrected with either the implantation of a toric intraocular lens or limbal relaxing incisions.

Eyes with corneal conditions—such as keratoconus, anterior basement membrane dystrophy or corneal scars—are not good candidates for premium IOLs due to the risk of higher-order aberrations and irregular astigmatism.

Ocular surface disease (OSD) should be addressed both preoperatively and postoperatively. Even if the patient was previously asymptomatic, OSD findings may bring about symptoms after surgery. Patients with variable corneal measurements or visible dry eyes may benefit from repeat measurements, frequent artificial tears, and a treatment regimen of Restasis (cyclosporine A 0.05%, Allergan), Cequa (cyclosporine ophthalmic solution 0.09%, Sun Pharmaceuticals) or Xiidra (lifitegrast 5%, Novartis).[24]

Patients with a history of refractive surgery often prefer to maintain spectacle independence. Prior vision correction surgery presents two unique challenges. First, increased corneal aberrations after ablative procedures may create further loss of contrast sensitivity, decreased best-corrected visual acuity (BCVA) and increased dysphotopsia. Second, refractive outcomes are less predictable, with a higher incidence of residual refractive error. A second refractive procedure can be offered as an enhancement if residual refractive error is significant after cataract surgery. Patients should be thoroughly educated on risks in these cases, though typically risks are lower than that of a lens exchange.

During preoperative evaluation, pay special attention to pupil size and angle kappa. Eyes with large pupils may not get as much benefit from some pupil-dependent premium lenses. With large angle kappas, the light rays from an object fall at a greater distance from the fovea, resulting in glare or halos.[25]

Carefully evaluate eyes with any macular or optic nerve pathology before deciding if presbyopia-correcting IOLs are appropriate. Premium lenses are generally contraindicated in the severe progressive pathologies. Monofocal IOLs may be better for patients with glaucoma, macular degeneration or other diseases to preserve BCVA.

Amblyopia should also be ruled out with potential acuity testing.

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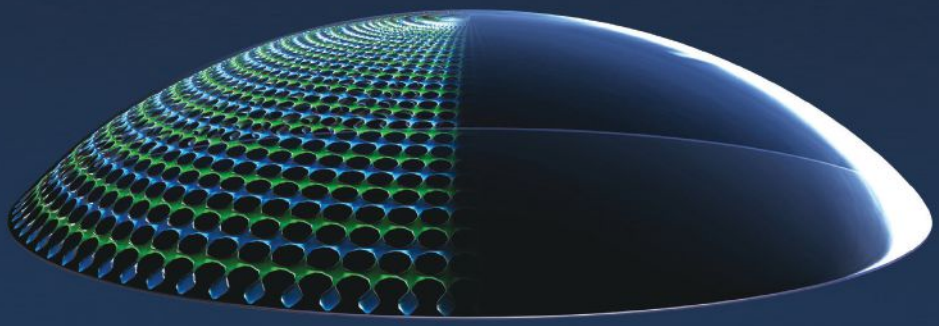

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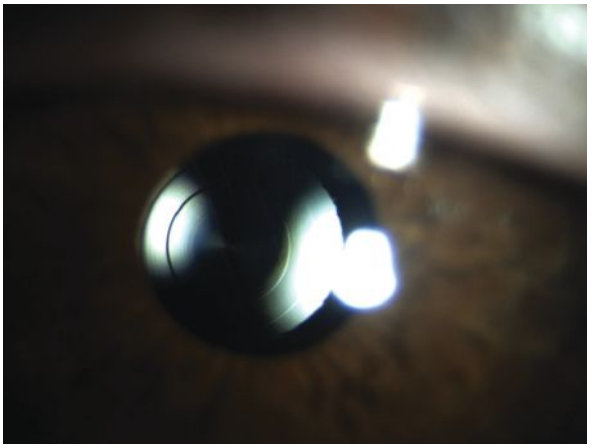

In most cases, multifocal IOL rings are visible even through an undilated pupil, as seen here.

Step Three: Anticipate Postoperative Problems

The best way to avoid premium IOL pitfalls is by predicting and preventing them prior to surgery. However, even with careful planning, patients can end up dissatisfied. The most common cause of dissatisfaction in patients with multifocal implants is residual refractive error, followed by dry eye, glare and halos.[26-28]

Residual refractive error may be addressed with laser vision correction; however, it is vital to allow for adequate healing and stabilization of corneal topography prior to any refractive surgery. Refractive surprises may occur unpredictably, but are more likely in eyes with particularly short or long axial lengths, a history of previous refractive surgery or both. Surgical practices may include laser vision correction enhancement in their premium IOL packages in case of a "refractive surprise." It may be prudent to discuss such policies with the surgical clinic to prepare your patients for such cases. Patients usually do well with premium IOLs once the residual refractive error is corrected.

If residual astigmatism is caused by rotation of toric multifocals off the intended axis, the patient should be sent back to the surgeon and the lens should be rotated into the correct position within the first few weeks after surgery.

In cases of early posterior capsular opacification, YAG capsulotomy may be needed, but it would be wise to wait at least three months to make sure the patient adapts well. If there is any chance that a lens exchange may be done, YAG capsulotomy should be delayed, as an open posterior capsule makes the exchange more difficult.

Contrast sensitivity may be significantly affected due to division of light that occurs with the multifocal design.[6] Apart from exchanging the IOL for a monofocal implant, nothing can be done to correct this.

One of the most common concerns reported by patients after multifocal implantation is nighttime dysphotopsia. This can occur even with monofocal IOL, but is more prevalent with premium lens designs.[6] The most important aid in managing these patients is time. Because multifocal IOLs divide incident light into multiple focal points, your patient's brain must adjust to process several images simultaneously. This process of neuroadaptation can take time and cause frustration, so patients must be prepared for it going in.[6]

Neuroplasticity is the ability of the brain to reorganize its function and structure in response to environmental changes.[29] Brain activity can be examined with functional magnetic resonance imaging (MRI). Using this technology, we know that patients who recently underwent multifocal IOL implantation have increased activity in their cortical areas dedicated to visual attention and effortful action, procedural learning, cognitive control and goal-oriented behavior.[30] Researchers also note a correlation between level of dysphotopsia symptoms and level of activity in the top-down attentional network in the parietal and frontal lobes as well as cingulate cortex and caudate nucleus.[30] Investigators also show that when functional MRI is repeated six months after multifocal IOL implantation, the regions of the brain associated with attentional network and effort are less activated.[31] This decrease in brain activation correlates with improved dysphotopsia symptoms at six months.[31] Since no measurable difference exists between optical parameters over the same time period, researchers believe neuroadaptation is the only explanation for such changes.[32-33]

Neuroadaptation is highly dependent on the individual. Personality traits to watch for include compulsive checking, orderliness, competence and dutifulness. Research suggests people who possess those qualities are more likely to experience glare and halos, and may fail to neuroadapt.[34]

Neuroadaptation plays an important role in multifocal outcomes, especially positive dysphotopsia. No effective treatment for these symptoms is available. Four to 12% of cases in which bothersome glare, halos or starbursts are present are due to an IOL defect.[31] In cases when patients still report bothersome dysphotopsia a month after surgery, doctors should offer

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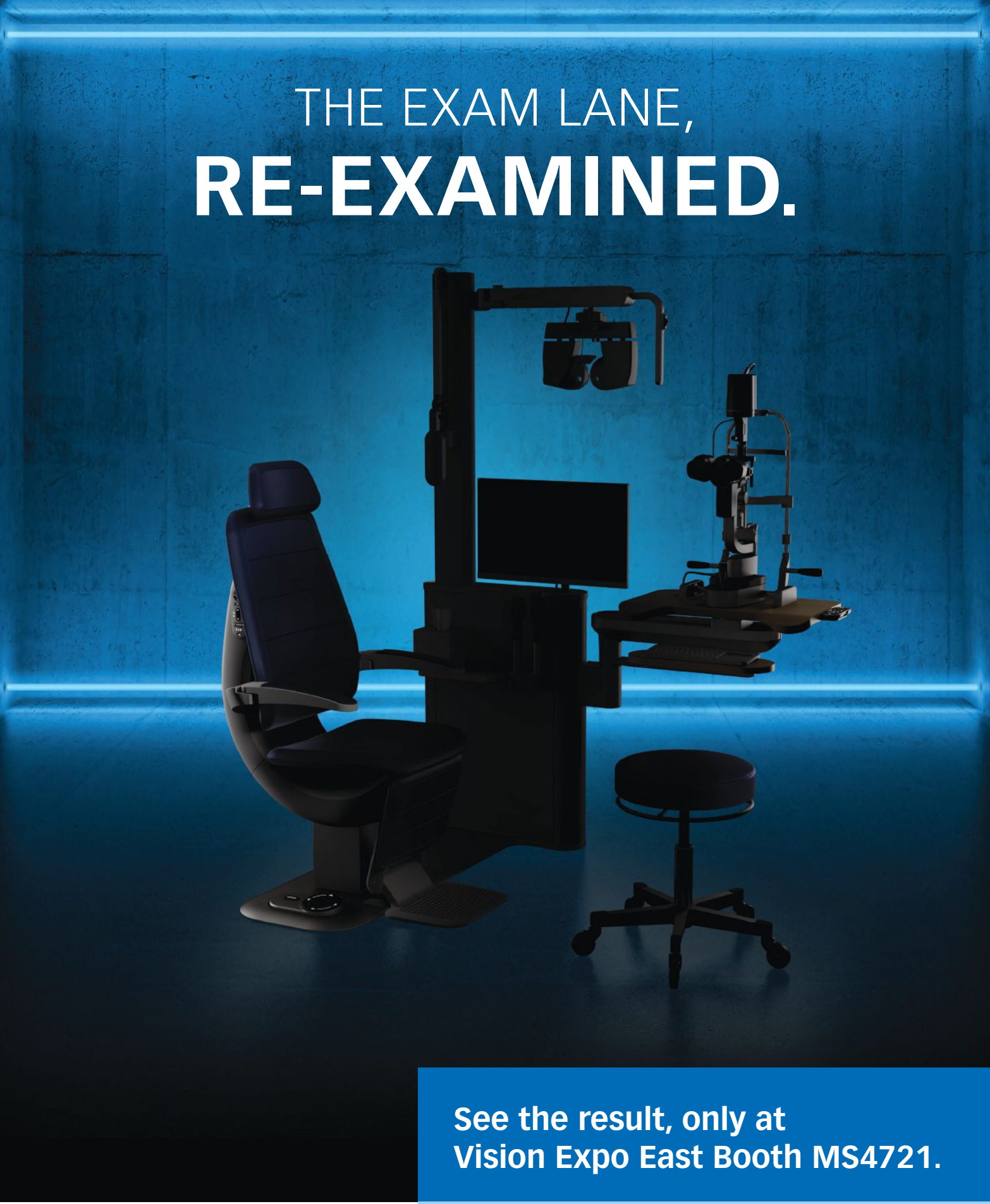

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reassurance and encouragement and allow for neuroadaptation to take over. If a patient is still bothered by dysphotopsia three to four months after surgery, or if their quality of life is significantly affected, they may consider switching out the premium lens for a monofocal implant.

Lens exchange, of course, is a last resort. Exchanging an IOL involves additional risks and costs to the patient. It helps if patients are aware of the possibility of a lens exchange prior to the initial surgery. If it does come down to this, it is best to do the exchange within the six months. Better understanding of neuroadaptive mechanism in the future may help optometrists and ophthalmologists better manage dysphotopsia and improve multifocal outcomes.

Step Four: Consider Adjustable IOLs

Given that residual refractive error is the most common reason for intraocular lens exchange, any technology that can improve accuracy is worth considering. In 2017, The RxLAL, (light adjustable lens, RxSight) was FDA approved and boasts the ability to non-surgically refine unexpected postoperative refractive error.[35,36] Corrective treatments are conducted using the company's light delivery device (LDD) and can alter up to 2D of spherical power or between 0.75D to 2D of astigmatism correction.[36]

The optics of the lens change via UV-sensitive macromers that, when stimulated, polymerize and thicken the intraocular lens where needed. For example, if a patient ended up hyperopic post-cataract surgery, the LDD would be programmed to photopolymerize the central portion of the lens, thereby thickening and increasing the power of the lens. For myopic surprises, the UV light would be concentrated in the periphery instead.

Several adjustments can be made once the patient is three weeks out of surgery to customize their vision. The flexibility of this lens offers the ability to optimize monovision endpoints and to minimize refractive surprise in those with history of laser vision correction.

Contraindications for this lens include conditions and medications that could cause sensitivity or adverse reactions to UV exposure. A careful review of the patient's systemic health should be conducted.

In the future, new lenses and devices may even allow patients to temporarily trial presbyopia-correcting optics with the ease of a UV treatment to revert back to single-focus properties if they are unable to adapt. Two companies—Perfect Lens and Clerio Vision—are currently developing such technologies. The ability to perform a noninvasive postoperative adjustment to overcome refractive surprises would be a major paradigm shift in cataract surgery. This would present more options to the patients postoperatively and greatly improve accuracy of visual outcomes.

Patients increasingly desire more spectacle independence after cataract surgery. Multifocal IOLs can provide excellent uncorrected vision across a range of distances, but may cause dysphotopsia and patient dissatisfaction. Careful selection of candidates, thorough preoperative education and high surgical skill all greatly increase chances of success with multifocals. Personality assessment should be included in the patient's regular preoperative ocular exam.

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The role of neuroadaptation cannot be underestimated, and the vast majority of post-op concerns resolve with time. Laser vision correction or IOL exchange remain an option when patients are dissatisfied with significant residual refractive error or bothersome dysphotopsia.

With adjustable IOLs beginning to enter the market, it may soon be common to improve refractive outcomes postoperatively. But for now, optometrists must perfect their preoperative examination and patient education protocol to continue increasing their surgical comanagement role. ■

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What OCT Can Offer Your Specialty Lens Fits

This technology is especially useful for customizing the design and troubleshooting any associated conditions. By Chandra Mickles, OD

Optical coherence tomography (OCT) has had a transformative impact on eye care. From detecting retinal fluid and preserving sight to monitoring glaucoma and maintaining vision, OCT has become an indispensable tool for posterior segment disease management. One of its newest iterations, anterior segment OCT (AS-OCT), is proving to be valuable for a wide variety of anterior segment applications and is becoming an integral part of specialty contact lens prescribing.

Whether they help restore sight for patients with keratoconus or relieve severe ocular discomfort for patients with dry eye disease (DED), specialty contact lenses can be life changing. However, fitting can be time-consuming and imprecise, as clinicians may need to rely on trial and error. This is where AS-OCT comes into play. Here is a comprehensive look at how AS-OCT can improve precision and proficiency in specialty contact lens eye care.

Precise, Accurate Measures

AS-OCT offers a wide array of measurements that can benefit specialty lens fitting and safeguard ocular health. The technology can provide information on the anterior eye that is particularly useful in refining specialty lens prescriptions.

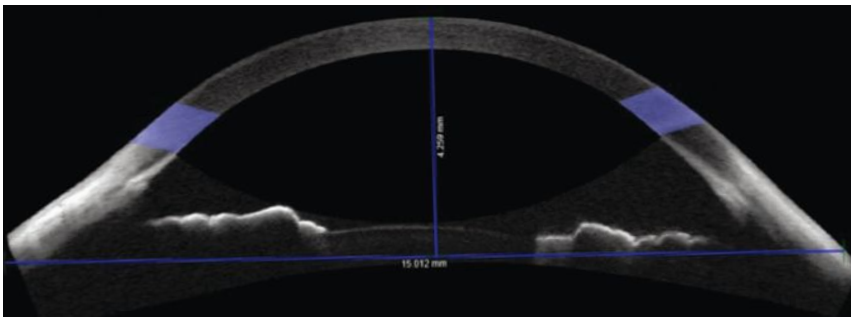


Fig. 1. Non-custom soft contact lenses didn't fit right for this patient with a sagittal height of 4,259µm. Consider custom lenses in these cases.

Sagittal height and depth. While clinicians had to estimate corneal-scleral sagittal height in the past, AS-OCT can now aid in selecting the optimal scleral lens sagittal depth. AS-OCT provides precise measurements that can increase initial lens success.[1,2]

For a lens close to 15mm in diameter, the fitter can use AS-OCT to measure the sagittal height of the anterior segment at the 15mm cord and add it to the desired initial central lens clearance. For example, if the sagittal height of the anterior segment at the 15mm chord is 4,000µm and the desired initial central lens clearance is 300µm, combining the two to find a sagittal lens depth of 4,300µm is a good starting point. The 15mm chord measurement also shows pupil size and angle kappa, which may be useful in designing multifocal lenses.

A larger sagittal depth is required for lenses larger than 15mm. Based on literature and experience, an adjustment of 400µm per each 1mm change in lens diameter, such that 4,700µm is the ideal initial sagittal depth for a 16mm diameter lens in the previous patient's case, is usually reliable.[3-6]

Conducting AS-OCT chord measurements is valuable beyond scleral lens selection. It is also beneficial

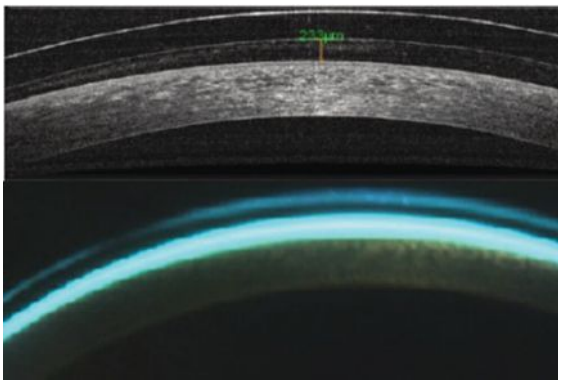


Fig. 2. AS-OCT (top) and biomicroscopy of central scleral lens clearance.

for soft lens selection. At the 15mm chord, the mean sagittal height of normal eyes is approximately 3,800µm.[3-5] Patients with sagittal height measurements less than 3,500µm or greater than 4,100µm may require a custom soft contact lens or a specialized fitting approach (*Figure 1*).[3-5]

Tear layer lens clearance. Posterior lens tear layer thickness is important when fitting scleral and hybrid lenses. AS-OCT can improve measurement accuracy, considering the clinically significant overestimation of central tear layer lens clearances of scleral lenses with slit lamps (*Figure 2*).[7-9] Central clearances for scleral and hybrid lenses designed to vault the cornea should be no lower than 100µm before lens settling and 50µm after to prevent corneal bearing (*Figure 3*).[10-13]

AS-OCT scleral lens limbal clearance measurements are also critical for a proper fit, especially as biomicroscopic assessments of limbal clearance can be challenging. While a well-fit scleral lens should clear the limbus by 10µm to 60µm, a fluorescein-stained tear layer doesn't become visible until 20µm to 30µm.[14-19] Consequently, a clinician may erroneously believe a well-fit scleral lens has limbal bearing.

Inherent inaccuracies in the subjective estimation of limbal clearance put AS-OCT measurements on an even higher pedestal. With subjective estimation, clinicians compare the post-lens tear layer thickness with the manufacturer's published center thickness. However, lens thickness varies significantly across the lens and with back vertex power (*Figure 4*).[20,21]

AS-OCT measurements are valuable when lens clearance must be assessed outside of the center of the lens, such as when the limbus is concerned or when the apex of an irregular cornea lies outside of the center of the lens in cases of keratoconus, pellucid marginal degeneration and post-graft patients (*Figure 5*). As with central and limbal areas of the cornea, excessive lens clearance or bearing is concerning at the corneal apex, a complication that AS-OCT may prevent.[11]

While advantageous, obtaining lens clearance measurements from multiple locations can be time-consuming. Fortunately, some commercial OCT instruments now generate color-coded corneal clearance maps to offer more rapid assessments of corneal clearance.[21]

Thickness and alignment. Lens thickness variance across scleral, corneal gas permeable (GP) and custom soft lenses is particularly evident with high back surface powers. Oxygen transmissibility across the surface of specialty lenses is important, so lens thickness is a crucial measurement, especially in cases of high powers. Lens thickness modification or lens wear reduction may prevent complications associated with hypoxia.

Landing zone. Scleral lenses land on the conjunctiva in a way that the lens weight should distribute evenly and the lens should neither sink into nor lift off of the conjunctiva. If the lens sinks, it could cause subtle, unwanted vessel constriction; if it rises, signs of discomfort not visible by slip lamp can be seen on OCT (*Figure 6*).

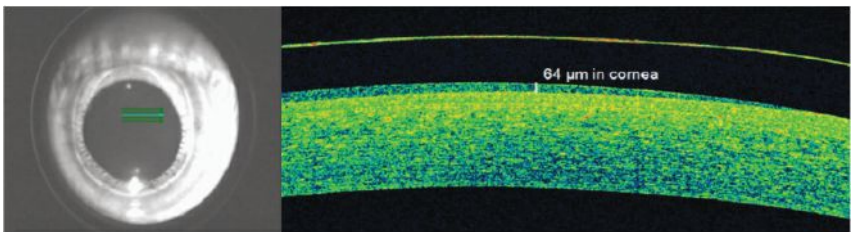


Fig. 3. OCT identified acceptable apical hybrid lens clearance on a keratoconus patient when slit lamp estimates were variable.

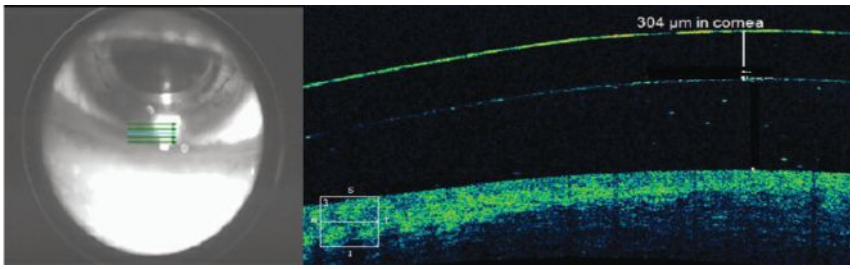


Fig. 4. The scleral lens thickness at the inferior limbus is thicker than the manufacturer-listed center thickness for this low-powered lens.

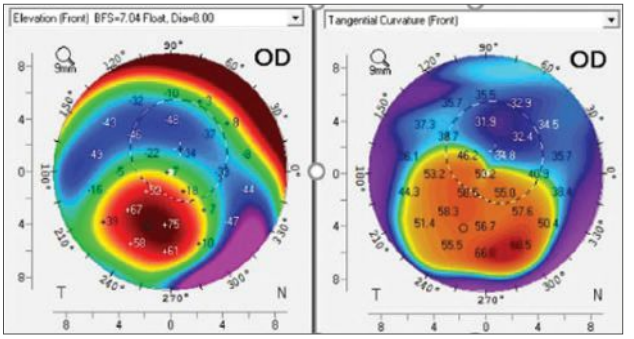


Fig. 5. Corneal topography of a post-PK patient shows the corneal apex is outside the center of the cornea.

Get to the Root of the Problem

Comfort, vision and health are essential aspects of success with contact lenses. But specialty lenses involve more than just designing lenses that can restore or enhance vision. By the nature of the conditions that require these lenses, fitters are frequently charged with unmasking and monitoring various anterior segment conditions. AS-OCT can shed light on the causes of a suboptimal wearing experience when biomicroscopy detection fails and characterizing a variety of corneal dystrophies and degenerations (*Figure 7*).[22,23] This includes delineating pathognomonic features and deposits within corneal sub-layers and monitoring corneal changes with contact lens wear.[22,23]

Discomfort. A defective lens edge on any design can cause lens discomfort. Determining if a poor-quality lens edge is the source of discomfort can be elusive with biomicroscopy. AS-OCT imaging of a lens landing zone can easily uncover if a defective lens edge is the culprit.

Blur. Visual blur with specialty lens wear can occur due to debris accumulation underneath the lens. Post-lens tear layer debris accumulation is common in scleral lens wear and creates a fog-like blur in 20% to 33% of patients (*Figure 8*).[17,21,24] AS-OCT imaging enables practitioners to observe visually impacting post-lens tear layer debris that is not always detectable at the slit lamp.

Corneal edema. Lens wear can induce corneal edema, which is especially challenging when fitting corneas prone to edema, such as post-penetrating keratoplasty and Fuchs' dystrophy and others with low endothelial cell counts. Patients should have no more than 5% of edema (*Figure 9*).[25]

Commercial OCT instruments now offer corneal thickness and pachymetry mapping to allow for efficient discovery and monitoring of edema that may be undetectable at the slit lamp. Additionally, this tool permits non-contact corneal thickness assessment with contact lens wear when conventional techniques cannot provide accurate measurements. OCT monitoring of corneal swelling in patients prone to edema is invaluable for safeguarding long-term ocular health and successful contact lens outcomes.

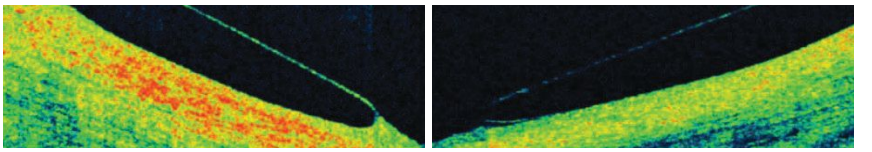


Fig. 6. AS-OCT shows a scleral lens edge with slight impingement into the conjunctiva (left) and an edge that lifts away from the conjunctiva (right).

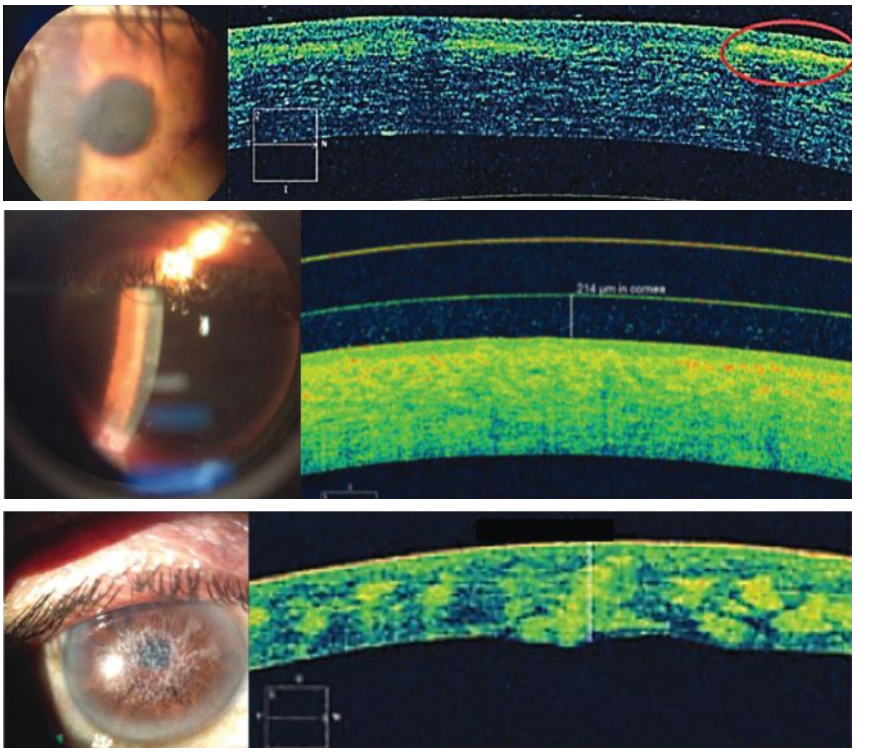
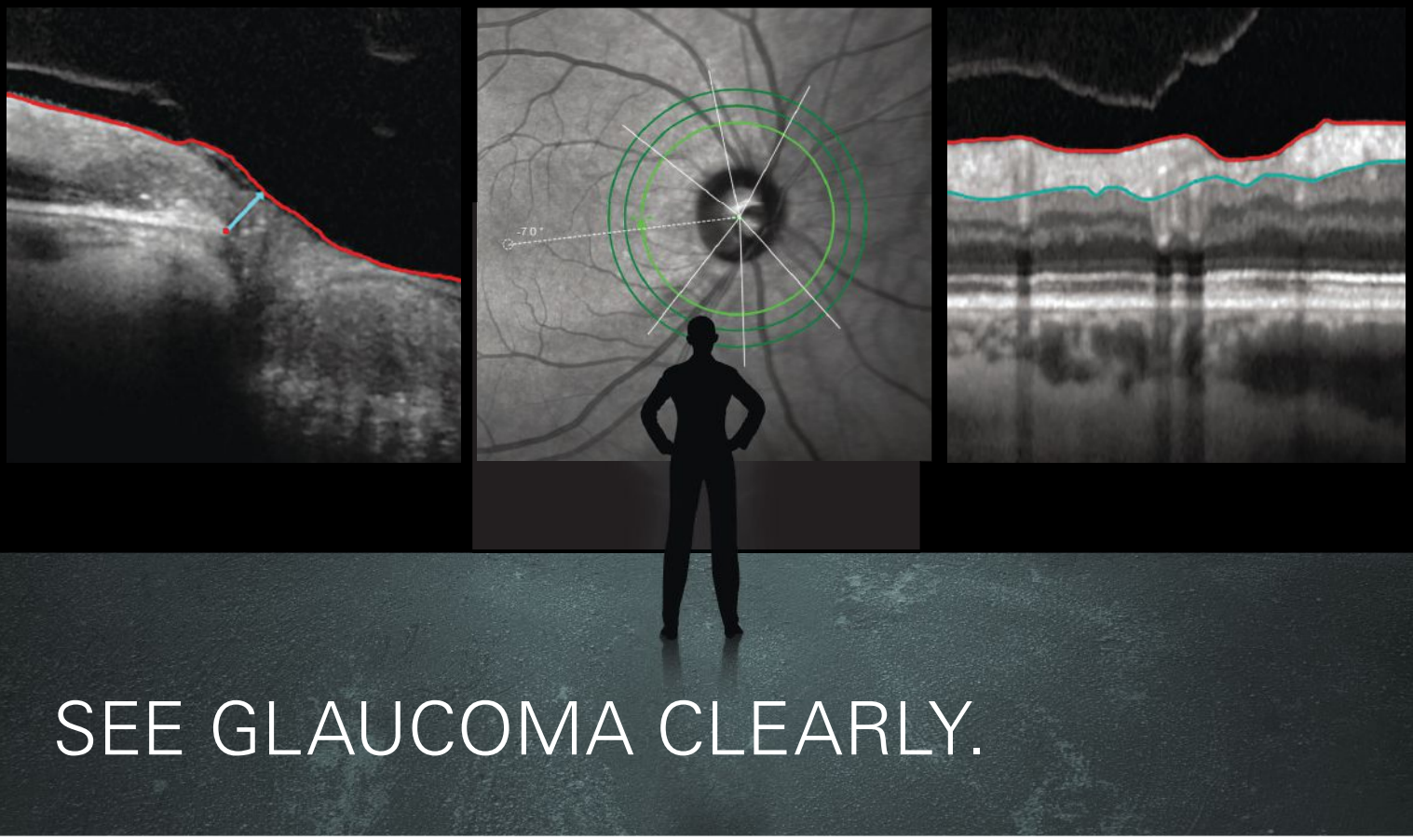


Fig. 7. From the top, Reis-Bücklers corneal dystrophy, lattice corneal dystrophy and type 1 granular corneal dystrophy with hyper-reflective deposits at Bowman's layer, in the anterior stroma and in the stroma, respectively.



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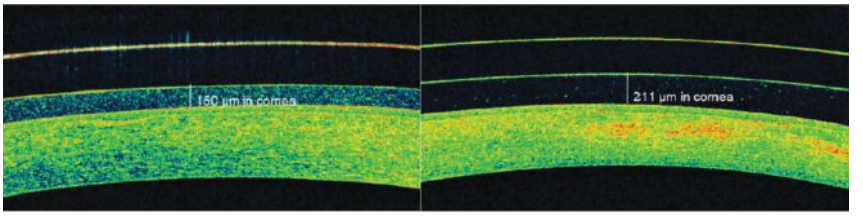

Fig. 8. AS-OCT identified significant post-scleral lens tear layer debris that was causing subtle but bothersome foggy vision.

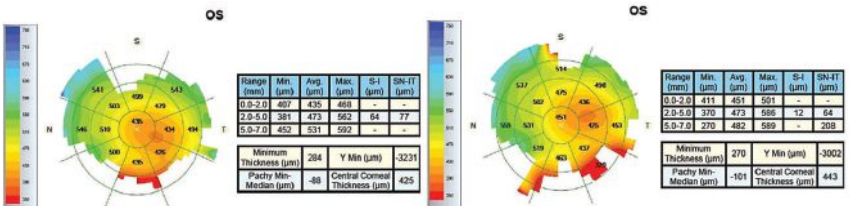

Fig. 9. Pre-scleral lens wear (left) and post-two hours of lens wear imaging (right) of an advanced keratoconus patient with a history of acute hydrops and pathological edema indicated central corneal swelling.

Keratoconus. Detecting keratoconus is crucial in preventing corneal ectasia secondary to LASIK.[26] Early detection helps delay corneal transplantation with corneal collagen crosslinking. However, the early stages of keratoconus are not easy to diagnose with conventional techniques because these eyes frequently have normal clinical findings that do not stand out on topography.[26-29] This is where OCT corneal epithelial mapping can be useful.[26,30,31]

Research shows that epithelial thickness in the thinnest corneal zone can diagnose forme fruste keratoconus.[27] In keratoconus, the locations of epithelial thinning on OCT thickness maps are also usually inferotemporal and roughly consistent with the location of corneal steepening shown on a corneal topography map.[32] While studies have investigated epithelial thickness patterns characteristic of keratoconus, keratoconus diagnosis still requires a clinician to recognize patterns on OCT pachymetry and epithelial thickness maps and correlate them with corneal topography and clinical information.[32-35]

AS-OCT may also help identify anatomic features predictive of acute corneal hydrops and future penetrating keratoplasty.[23,36] In a study involving eyes with advanced keratoconus, increased epithelial thinning, stromal thinning at the cone, anterior hyper-reflective areas in Bowman's layer and the absence of stromal scarring on AS-OCT were potential predictive factors for the development of acute corneal hydrops.[23,36] AS-OCT is also very valuable in monitoring the healing process of acute hydrops (*Figure 10*).

Inflammation and infection. While contact lenses are an effective form of vision correction, wearing them increases an individual's risk for sight-threatening infections and associated inflammation.[37] AS-OCT can be used to monitor the treatment of contact lens-associated infiltrative events. Objectively monitoring treatment response involves assessing the degree of regression of a stromal hyper-reflective band indicating infiltration of the corneal stroma and changes to corneal thickness.[23,38]

Anterior chamber cellular reaction is key in establishing inflammation severity and treatment effectiveness. AS-OCT outperforms slit lamp evaluation by enabling automated anterior chamber cell grading (for consistent inter- and intra-clinician evaluation) and the evaluation of anterior chamber inflammation in corneas with impaired clarity, such as scarred eyes with advanced keratoconus (*Figure 11*).[23,39]

Dry eye. Dry eye is common in contact lens wearers, with at least 40% of users reporting dry eye symptoms.[40] Conventional dry eye diagnostic tests, such as Schirmer's tear-wetting and ocular surface dye-staining, are invasive and subjective, which can negatively influence result accuracy. To prevent this, objective assessments of the tear film are needed.[41,42] AS-OCT can more precisely and objectively quantify markers of DED, such as tear film layer thickness and tear meniscus

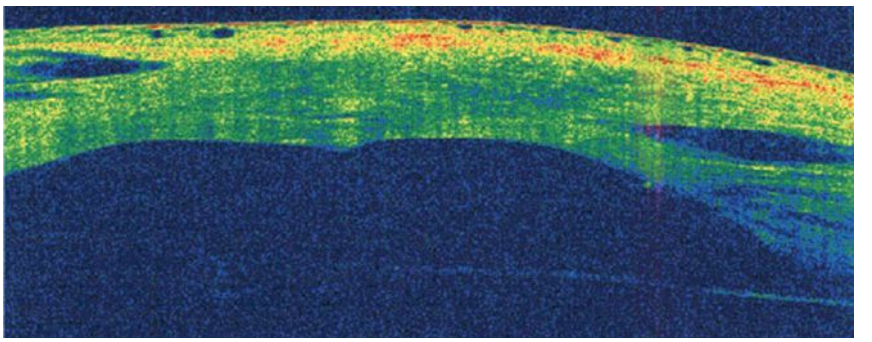

Fig. 10. AS-OCT is useful for monitoring the healing process of acute corneal hydrops.

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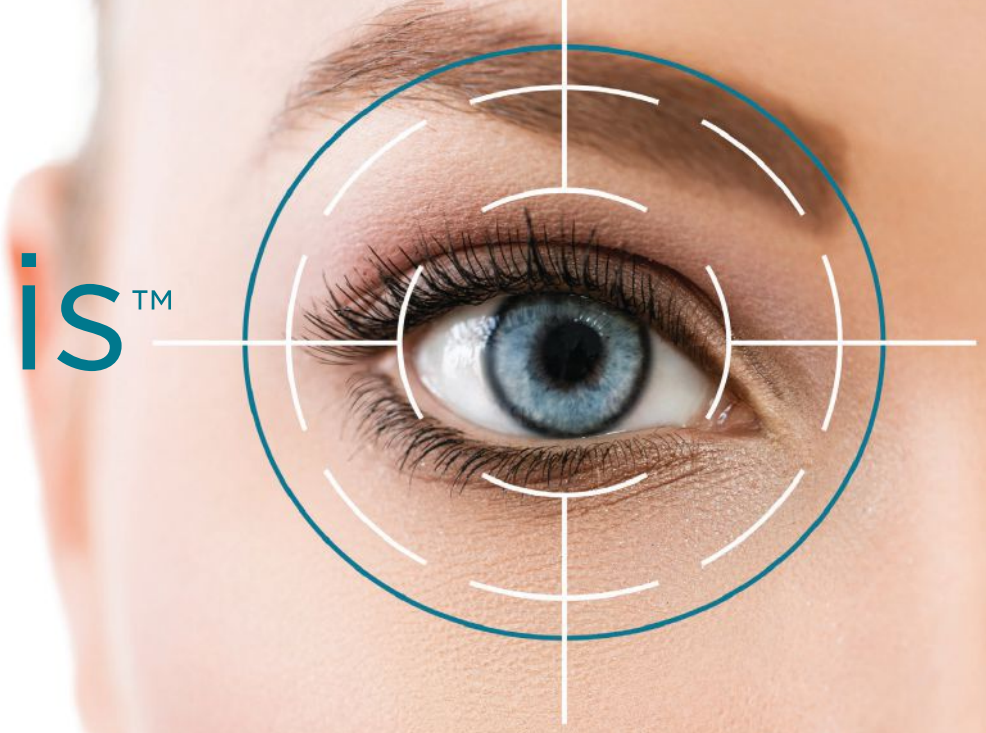

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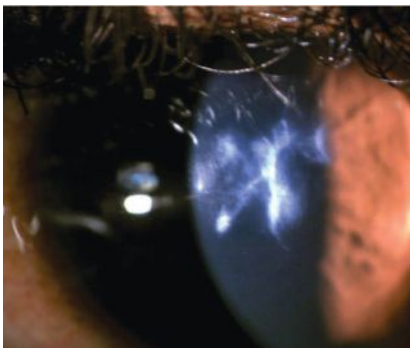

Fig. 11. Accurately assessing the anterior chamber reaction with a slit lamp would be challenging in this case of severe keratoconus with a scarred cornea.

height, than conventional means of a slit lamp beam.[43,44] With AS-OCT, the highly reflective tear film is more distinguishable from the cornea.[45] For tear meniscus height measurements, clinicians can place the AS-OCT crosshair at the lid margin and use the caliper to measure between the fornix of the globe and the palpebral conjunctiva.

While AS-OCT tear film measurements have a moderate correlation with the degree of dry eye symptoms, they are very useful for monitoring a patient's response to dry eye treatments, such as artificial tears and punctal occlusion.[46-49] AS-OCT corneal epithelial mapping can also aid in DED detection, as irregular epithelium thickness patterns are observed in dry eye.[50] In the future, OCT may become more of a simple, patient-friendly method for obtaining information on meibomian gland microscopic structural changes.[51] AS-OCT's objective and efficient role in DED identification and monitoring warrants its involvement in diagnostic protocols to improve outcomes.

OCT imaging offers far more than what the technology set out to do at its inception 30 years ago. It has advanced specialty lens practice by refining lens selection, quantifying fit aspects and troubleshooting related concerns. OCT has helped clinicians restore, enhance and maintain sight for many patients, who in turn are now able to visualize undesirable contact lens complications and better understand their corneal state and efficient wear and care techniques. OCT's role in educating patients on their condition and improving compliance with treatment regimens cannot be overstated. Here, a picture is truly worth a thousand words. ■

Dr. Mickles is an associate professor at the Nova Southeastern University College of Optometry, the coordinator of the Dry Eye Care Center and a fellow of the American Academy of Optometry and the Scleral Lens Education Society.

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Protect Your PK Patients

Scleral lenses can improve vision for these eyes if the right precautions are taken.

Edited by Joseph P. Shovlin, OD

Q I struggle with recommending scleral lenses following penetrating keratoplasty (PK); virtually all of my patients experience significant edema with prolonged wear. How can I maximize corneal oxygen flux and minimize edema and premature endothelial cell attrition with these lenses?

A "Post-PK patients are great scleral lens candidates, but their eyes are delicate and require careful consideration during the fitting process to ensure optimal corneal and graft health," according to Andrew Fischer, OD, of Professional Eyecare Associates in Indiana. "We must minimize the risks that scleral lens wear may pose to these corneas." These include corneal edema and endothelial cell attrition, which could ultimately lead to graft failure.

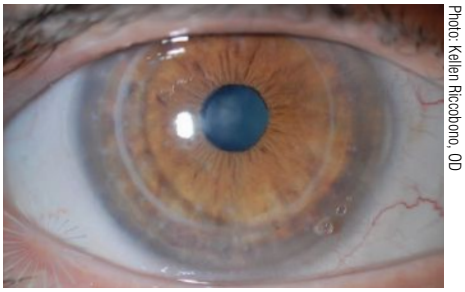

Photo: Kellen Riccobono, OD

A large-diameter scleral lens with a fenestration at 4 o'clock is fit on a post-PK patient.

Parts of the Whole

When picking a lens, Dr. Fischer suggests first considering oxygen permeability. He notes that there are many gas permeable (GP) lens materials, with some of the newest achieving ratings over 160Dk. For post-transplant corneas, his motto is the more permeable the lens, the better.

Lens thickness also plays a role in oxygen transmission to the cornea, Dr. Fischer adds. His strategy is to ask his consultants to produce the lens he needs in a more permeable material and cut it with a thinner central thickness to help with oxygen flow. Additionally, minimizing lens clearance over the cornea can help maximize corneal health. Dr. Fischer typically aims for 100µm to 150µm of clearance for post-graft eyes. Many lens designs offer quadrant-specific customization, which gives even the most irregular corneas a suitable post-lens tear lake.

The next step is choosing the lens design. Dr. Fischer has found success with larger and looser lenses, which tend to be more stable on the eye and help minimize edema. If a patient experiences irritation and/or scratching, the lens is too loose.

Especially for post-graft corneas, Dr. Fischer is mindful of ensuring the lens is not excessively tight with zero tear exchange, as this reduces oxygen to the cornea. To check oxygen supply after the lens settles on the eye, he uses a fluorescein strip to "paint" the front surface of the lens under cobalt blue light and Wratten filters. He specifies that a small amount of tear flow is ideal and indicates there is oxygen flux under the lens, not just through the GP material.

While recent innovations may make this practice unnecessary, Dr. Fischer says lens fenestrations—small holes drilled through a lens to allow surface tears to exchange with the post-lens tear layer—can help promote increased tear and oxygen transfer. He advises patients who he fits with looser edge profiles or fenestrated lenses that they may occasionally experience mid-day fogging and that removing, refilling and reinserting the lenses may be necessary for optimal clarity.

Dr. Fischer cautions not to overlook elevated intraocular pressure (IOP) as a potential stressor on endothelial cells and corneal grafts. Unfortunately, he acknowledges that obtaining accurate IOP measurements in cases of corneal transplant patients can be difficult due to severe irregularity, varying pachymetry values and the presence of corneal edema. Dr. Fischer speculates that scleral lenses may elevate IOP during wear, though it is hard to assess to what extent. For patients who experience edema with scleral lenses, he highlights IOP-lowering medications as a possible solution to decrease endothelial stress.

"Scleral lenses are ideal for most eyes after a PK; they maximize vision, minimize the risk of mechanical rubbing forces from the lens and make long-term graft success more attainable," concludes Dr. Fischer. Optimizing the fit with state-of-the-art materials and designs, and monitoring the eye, are vital to endothelial health, he emphasizes. However, in some cases, he warns that the endothelium may not be robust enough to keep a graft edema-free and a consultation for endothelial transplant may be the best option. ■

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HAAG-STREIT DIAGNOSTICS

Edited By Carlo J. Pelino, OD, and Joseph J. Pizzimenti, OD

Out of Rhythm

While amiodarone is highly effective, long-term use is associated with concerning ocular side effects. **By Sara Hyatt, OD, Scott Slagle, OD, WingYin Hui, OD, and Alicia Greene, OD**

Amiodarone is one of the most effective medications to treat ventricular and supraventricular arrhythmias. Long-term use, however, is associated with vision loss secondary to optic neuropathy.

Case Report

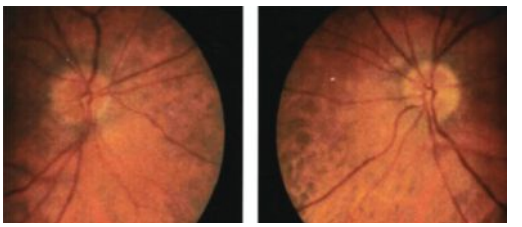
A 66-year-old Caucasian male presented with blurry vision that had gradually worsened over the past two months. He was unsure if this was a monocular or binocular phenomenon and had not found relief with glasses.

The patient's health history was positive for myocardial infarction and coronary artery stenting two years prior. He was taking amiodarone and apixaban for paroxysmal atrial fibrillation, lisinopril and metoprolol for hypertension, metformin for type 2 diabetes and atorvastatin for hyperlipidemia. He undergoes continuous positive airway pressure therapy for obstructive sleep apnea.

Clinical Examination

Corrected visual acuities were 20/20 OD and 20/25+2 OS, intraocular pressures were 16mm Hg OD and 17mm Hg OS, blood pressure was 161/94mm Hg and heart rate was 56bpm. Anterior segment exam revealed grade two whorl keratopathy OD and grade one OS. Dilated lens exam revealed grade two nuclear sclerosis OU.

The right optic nerve had a small cup-to-disc ratio (0.1/0.1) with hyperemic, blurred temporal margins. There was noticeable nerve head elevation with edema temporally and multiple small flame hemorrhages. C/D ratio OS was also small (0.15/0.15) with a crowded appearance and mild hyperemia superiorly and inferiorly. The left disc margins were distinct and without hemorrhages. Macular disease was not present OD, and only mild pigmentary changes were observed in the left macula. There was a small choroidal nevus with benign features in the posterior pole OS.



Both optic nerves are hyperemic and have blurred temporal margins, OD>OS.

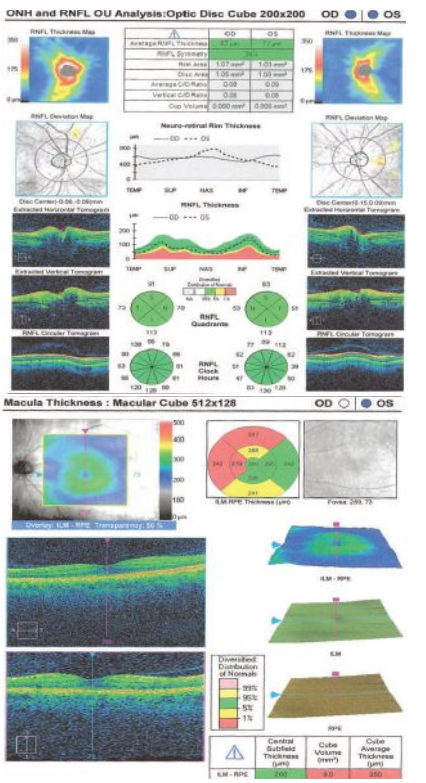
Though OCT of the retinal nerve fiber layer (RNFL) was normal at first glance, the square appearance and dark color of the thickness map raised further suspicion for disc edema OD. The map revealed asymmetric average RNFL thicknesses, with the right eye measuring 10µm greater than the left, and overall low retinal thickness most evident superior and temporal to the optic nerve, sparing the central foveal subfield.

Visual field testing OD revealed normal threshold sensitivities with a few peripheral points of decreased sensitivity that did not display clinically significant patterns. Testing of the left eye revealed an inferonasal area of low sensitivity, which may be indicative of prior optic neuropathy and influenced by age-related macular degeneration.

Patient Outcome

Serologic testing to rule out infectious and inflammatory etiologies showed normal results. Magnetic resonance imaging and venography showed no signs of abnormality.

The patient's cardiologist elected to discontinue amiodarone and continue apixaban and metoprolol. He inserted a heart monitor to detect atrial fibrillation, which did not recur.



RNFL thickness findings on OCT.

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A neuro exam revealed a superior altitudinal visual field defect using a 30mm red target (OD>OS) and a 40% decrease in red desaturation OD. Repeat visual field testing did not show any significant defects. The assessment corroborated the concern for amiodarone-associated optic neuropathy (AAON). The patient was followed closely by his team and showed slow improvement and eventual resolution of the disc edema OD over three months.

A Drug That Gives and Takes

Heart disease is the leading cause of death in the US, affecting millions of Americans.[1] Luckily, therapies such as amiodarone can help those with life-threatening cardiac arrhythmias.[1] Amiodarone is the most widely prescribed antiarrhythmic in the US.[2,3] Unfortunately, the drug's efficacy may come at a price.

Oral amiodarone is lipophilic and has a half-life of 60 to 142 days.[3,4] Because of its biphasic properties, it interferes with the movement of phospholipids across the intracellular membrane and accumulates in membrane-rich structures.[1] Its toxins can negatively affect tissues throughout the body.[5] Prolonged use is associated with pulmonary toxicity, thyroid dysfunction, peripheral neuropathy, tremor, ataxia with staggering gait, gastrointestinal disturbances and ocular manifestations.[5,6]

Ocular changes are common, with the majority developing verticillate keratopathy. Patients taking moderate to high doses may develop anterior subcapsular cataract. Maculopathy has also been reported.

Optic Neuropathy Look-alikes

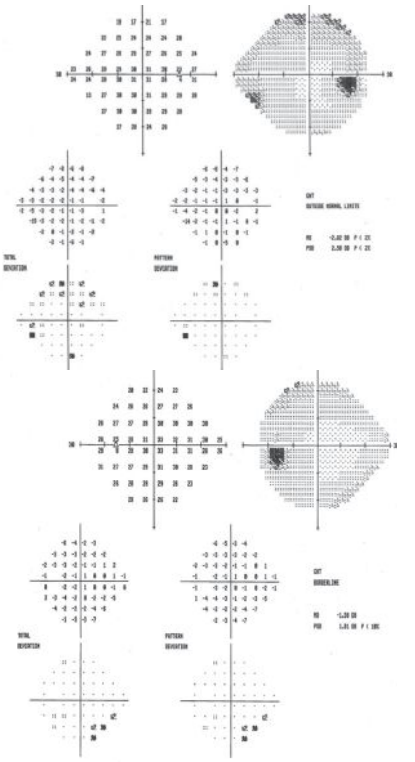
AAON's presentation is highly variable; tit may appear as unilateral or bilateral optic disc swelling with hemorrhages.[1,4] Clinical manifestation typically begins within 12 months of the start of therapy, with a median onset of four months for subtle, mild vision loss that gradually resolves over another few months.

Typical characteristics include insidious onset of visual loss and bilateral protracted disc edema over months. A wide spectrum of optic nerve involvement is possible. Some patients are asymptomatic while others have substantial visual dysfunction, with either acute or insidious vision loss, and simultaneous bilateral or sequential disc edema.[1,6,7] Conflicting evidence exists regarding acuity stabilization and visual field defect resolution in patients with AAON, but early detection and amiodarone discontinuation could make a difference in visual prognosis.[1,5,6]

Because many patients taking amiodarone often have associated vascular risk factors, clinical distinction from non-arteritic anterior ischemic optic neuropathy (NAION) can be a challenge.[7] NAION is the most common acute optic nerve disorder in patients over 50. It results from ischemic insult to the nerve head and is characterized by acute, monocular, painless vision loss with disc swelling.[8] Risk factors include previous NAION, sleep apnea, older age, HLA-A29 antigen, chlamydia, diabetes, oral fever blisters, hypertension and small, crowded optic nerves.[8]

NAION occurs equally among men and women, while AAON is more common in men. Disc edema in NAION generally resolves within weeks, while it could take months to fully clear up in AAON. Unlike with AAON, neurological signs are absent in NAION.[7,8] Mean visual acuities tend to linger around 20/60 in NAION and 20/30 in AAON.

The distinction between AAON and NAION remains controversial, as characteristics overlap. Clinicians must act on ocular changes in amiodarone users by identifying them early and considering drug discontinuation for the best outcomes. ■



Visual field testing results.

Dr. Hyatt is in private practice in San Antonio, TX. She is a Fellow of the American Academy of Optometry (AAO).

Dr. Slagle is the supervisor of the Salem VA Medical Center's optometry residency program. He is a Fellow of the AAO.

Dr. Hui is in private practice in Illinois.

Dr. Greene practices at the Salem VA Medical Center and supervises optometry interns and residents.

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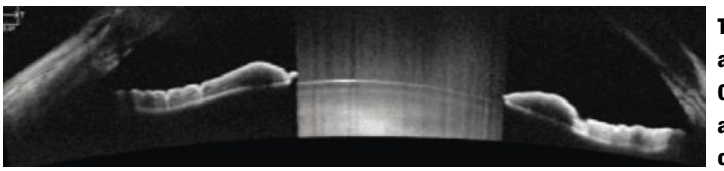
Pigmentary Glaucoma, Revisited

New pharmaceutical options may help manage secondary disease.

By Joseph W. Sowka, OD

A 61-year-old man was referred for elevated intraocular pressure (IOP). He had no visual or ocular complaints and reported no ocular trauma.

His best-corrected visual acuity was 20/20 in each eye with a moderately hyperopic correction. His anterior chambers were deep, more so in the left eye than the right. A Krukenberg spindle of pigment was noted on his left corneal endothelium. His IOPs were 15mm Hg OD and 38mm Hg OS. Central corneal thickness was slightly less than 500µm in each eye. Optical coherence tomography (OCT) showed early abnormalities in the left eye. Threshold perimetry revealed mild visual field defects nasally in his left eye. Gonioscopically, trabecular pigmentation was mild throughout the right angle and very dense throughout the left. His right iris assumed a planar configuration to the angle recess while his left iris was markedly concave.



This patient's anterior OCT shows a markedly concave iris.

Making the Call

I diagnosed this patient with unilateral pigmentary glaucoma in the left eye with pigment dispersion coming from an anomalous iris configuration that was not present in the right eye. Patients with pigment dispersion syndrome and pigmentary glaucoma demonstrate liberation of iris pigment within the anterior chamber. Often, this appears as a vertical, granular brown band along the central corneal endothelium known as a Krukenberg spindle.[1-3] Pigment accumulation may also be evident on the lens and iris.

Dense pigmentation is seen gonioscopically, often covering the trabecular meshwork (TM) for 360° with increased prominence in the inferior quadrant. When pigment accumulates on Schwalbe's line, it is referred to as Sampaolesi's line. The angle recess remains unchanged and open. Radial, spoke-like transillumination defects of the mid-peripheral iris are common, though not present in every patient.[4]

Pathophysiology

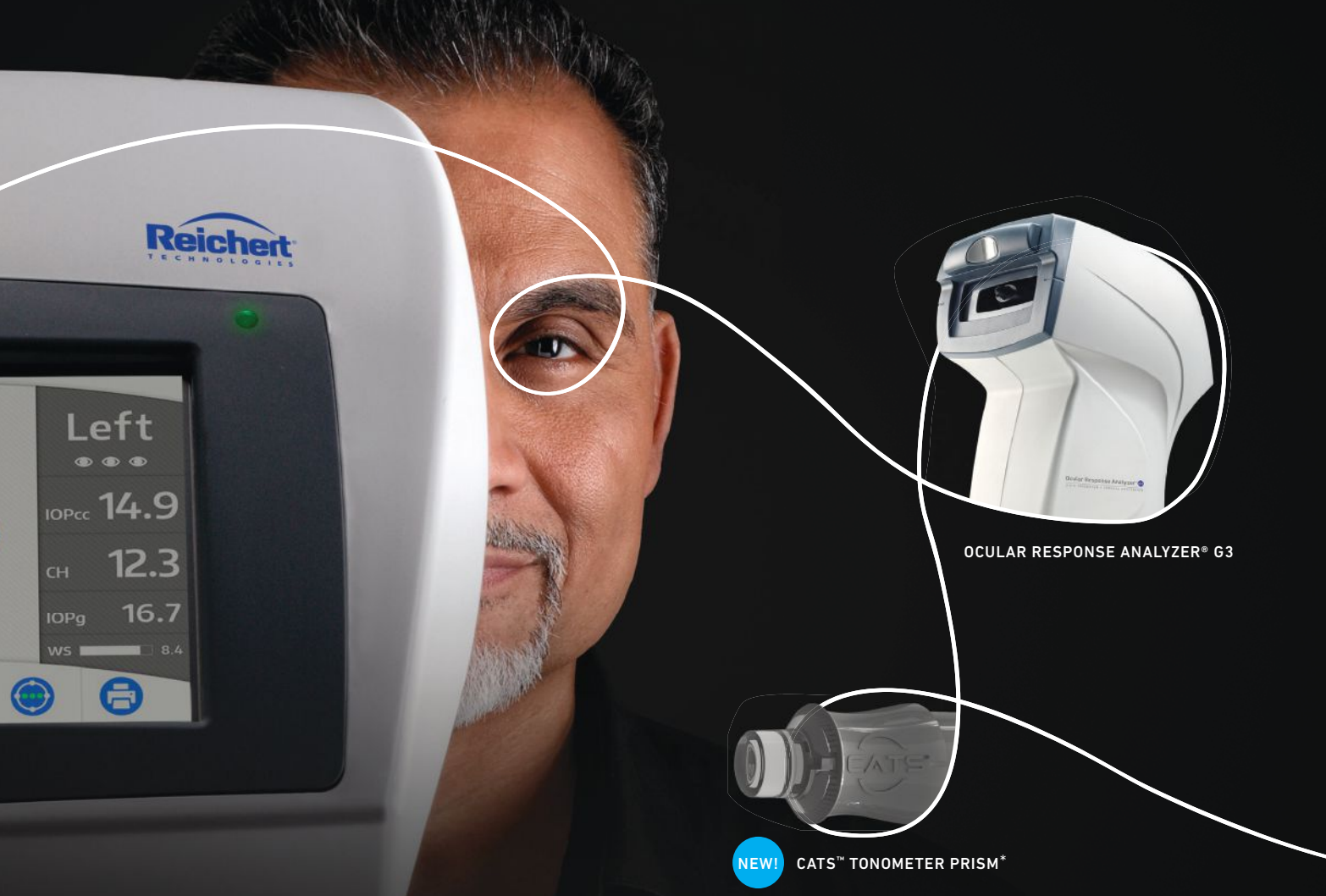
Pigment release occurs as a result of the proximity between the posterior iris pigment epithelium and the zonular fibers of the lens. The abrasive nature of this physical contact leads to mechanical disruption of the posterior iris surface and release of pigment granules into the posterior chamber, which follows the flow of the aqueous convection current into the anterior chamber angle.[5-7]

Many patients with pigment dispersion syndrome and pigmentary glaucoma demonstrate a concave approach of the iris as it inserts into the anterior chamber angle, giving the iris a "backward-bowed" appearance on gonioscopy.[7] This posterior bowing of the iris places the posterior iris into apposition with the lens zonules. As the iris responds to light, iridozonular friction results in pigment liberation from the posterior iris. Sometimes the degree of pigment loss in the mid-peripheral areas produces visible transillumination defects corresponding to packets of iris zonular fibers.[5] While the majority of these patients have a concave iris approach, others may have a flat or planar approach, making the mechanism of pigment release less clear.[6]

Researchers theorize that, in cases with a markedly concave iris insertion, the iris functions as a flap valve lying against the anterior lens surface. When a pressure gradient develops that is greater in the anterior chamber, the iris is forced backwards, closing the valve and trapping the aqueous from moving into the anterior chamber. The increased pressure forces the iris into the concave approach causing a "reverse pupillary block." This increases the irid-zonular friction and apposition, leading to pigment release from the posterior iris.[8,9] This seems to increase upon blink.[9-11]

Fallout

When excessively released pigment accumulates in the TM, there are two possible consequences. First,



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pigment may reside benignly in the trabecular meshwork. Here, IOP is unaffected and the condition remains pigment dispersion syndrome. Alternatively, when the pigment causes IOP to rise and damage occurs, the patient develops pigmentary glaucoma.[6]

Interestingly, pigment granules blocking the TM are not the likely to raise IOP long term.[12] Endothelial cells lining the trabecular beams of the TM quickly phagocytize small amounts of accumulated pigment, preserving the normal architecture of the TM.[13-15] However, in chronic cases of pigment dispersion, greater amounts of pigment are more difficult for the cells to phagocytize. When this occurs, the endothelial cells that line the TM beams disintegrate, contributing to a rise in IOP.[15]

As pigment dispersion syndrome's only impact on ocular health is potential development of pigmentary glaucoma, these patients should be treated as glaucoma suspects. Monitor these patients for IOP elevation and optic nerve changes periodically. Patients with pigment dispersion syndrome who were followed for more than 10 years without developing pigmentary glaucoma have lower risk of later development.[16] The risk of developing pigmentary glaucoma from pigment dispersion syndrome is 10% at five years and 15% at 15 years.[17] Myopic males in this age range were more likely to convert to pigmentary glaucoma, and an IOP greater than 21mm Hg at initial examination was associated with an increased risk of conversion.[17]

Therapies

Medical treatment of pigmentary glaucoma involves reduction of IOP with aqueous suppressants.[1] Beta blockers, carbonic anhydrase inhibitors and alpha-adrenergic agonists are all acceptable options.

This patient demonstrates heavy trabecular meshwork pigmentation.

Prostaglandin medications can lower IOP in eyes with pigment dispersion.[18] Researchers have not specifically reported rho-kinase inhibitors, either in isolation or in combination with latanoprost as a therapy for pigmentary glaucoma. This class of medication is approved for open-angle glaucoma (which theoretically encompasses pigmentary glaucoma) and ocular hypertension. Rho-kinase inhibitors act through trabecular relaxation and increased outflow.[19] However, it is unknown if the heavy degree of trabecular pigmentation would be a hinderance to this effect.

Procedures

Patients with pigmentary glaucoma respond well to argon laser trabeculoplasty and selective laser trabeculoplasty (SLT).[20-23] One series shows post-SLT IOP elevation was a serious adverse event.[22] Lower power settings may be necessary in patients with heavily pigmented angles.

Laser peripheral iridotomy (LPI) can convert the iris from a concave to a planar approach.[6-8] It also decreases the biomechanical factor, causing contact between the iris and zonular fibers, and may lower IOP. Nevertheless, the effects of LPI on visual field changes and progression have not been established in pigmentary glaucoma.[23,24]

My patient was treated with topical latanoprost in his left eye after detailed discussion of risks and benefits as well as possible cosmetic asymmetry from prostaglandin use. His IOP reduced to 18mm Hg in his treated eye and he's currently being followed without adverse events or progression. ■

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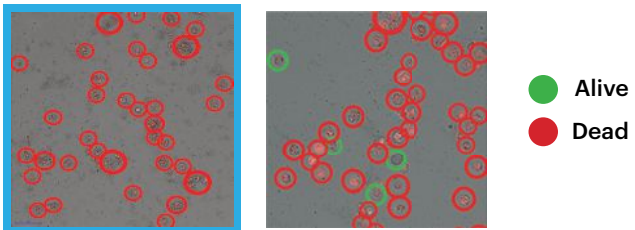
ACUVUE™ RevitaLens Multi-Purpose Disinfecting Solution offers the **all-day comfort** and convenience of a multi-purpose solution with **peroxide quality disinfection.**[1]

- Provides **broad-spectrum disinfection** against FDA/ISO standard-panel organisms, comparable to peroxide systems[1]
- Provides **greater than 99.9% kill rate against both forms of acanthamoeba** (in vitro)[1]
- **Gentle** on the eye and delivers **exceptional disinfection** against **harmful germs and bacteria**[1]
- Delivers the **all-day comfort** your patients need[1]

ACUVUE™ RevitaLens Multi-Purpose Disinfecting Solution (MPDS) killed Acanthamoeba trophozoites – delivering peroxide quality disinfection in vitro.[1]

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

3% Peroxide

* 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™[6] 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.

With Disc Edemas, Act Fast

Treatment is dependent upon the cause of the issue. It's on you to identify it.

By Saoul Mancha, OD, David Scales, MD, Joseph Pizzimenti, OD, and Richard Mangan, OD

Any decrease in vision, with or without headache, can signal consequences that go beyond the eye. These consequences can be extreme and include such dire conditions as aneurysm or tumors or can stem from infections that have systemic implications, such as Lyme disease or syphilis. These patients always warrant an urgent comprehensive work up.

When an optic disc edema is identified, optometrists need to keep their differential diagnoses broad, and consider common and uncommon etiologies.

Any appropriate serologic or radiologic testing should be ordered swiftly, as these will help identify devastating conditions.

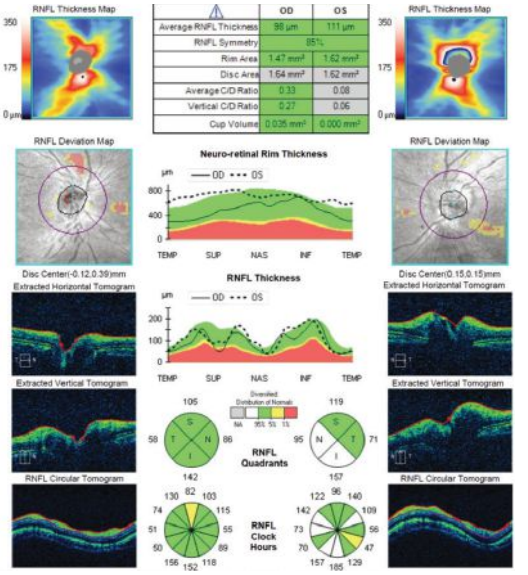


Fig. 1. These SD-OCTs of the patient's optic nerves and RNFL cube shows substantial elevation of her left eye's neuroretinal rim tissue. The extracted tomograph highlights the large elevation asymmetry between the eyes.

The Patient

A 14-year-old Hispanic female presented to the retina office regarding concerns of an edematous optic nerve, blurred vision of the left eye and headache. The blurred vision and headache were described as mild and constant since their onset 10 months prior. The patient's medical history was positive for obesity and she denied using any medications. The patient had a positive family history of diabetes and hypertension.

During the patient's initial visit, the entering visual acuities with correction were 20/25 OD and 20/40 OS (pinholed to 20/30 OS). Pupils were equal and reactive to light with no afferent pupillary defect. Extraocular muscles had smooth full range of motion with no pain and visual fields by confrontation were unremarkable. The patient's preauricular nodes were normal and non-tender on palpation. Goldmann tonometry revealed IOPs of 16mm Hg in both eyes.

Dilated fundus exam of the right eye was unremarkable; however, spectral domain optical coherence tomography (SD-OCT) revealed 2+ optic nerve swelling with blurred margins in the left eye (*Figure 1*). She also had an approximate cup-to-disc ratio of 0.2 OS, which was difficult to assess. No spontaneous venous pulsation was noted in either fundus. Macula, retinas, vessels and vitreous humor were unremarkable in either eye. Humphrey visual fields (HVF) 30-2 showed a few mildly incongruous depressed points within the superior temporal quadrants in the left eye more than the right (*Figure 2*). HVF reliability indices showed poor reliability on the left eye testing secondary to increased fixation losses.

We determined her left eye had an optic disc edema. The patient and parent were educated on the findings and the need for neuroimaging to be performed within one week. Blood work, magnetic resonance imaging (MRI) with and without gadolinium and lumbar puncture were ordered for further evaluation.

Follow-Up

At the next visit, the patient described her blurred vision as intermittent over the previous four weeks and denied any pain since the last exam. Visual acuity with correction was 20/25 OD and 20/40 OS, pin-holed to 20/25 OS. All entrance testing and anterior segment biomicroscope findings were unchanged since last exam. IOPs were 17mm Hg OD and 16mm Hg OS. Fundus findings were unchanged since previous visit. Fundus fluorescein angiography (FFA) and SD-OCT of the maculae were ordered and performed in office.

FFA results were unremarkable in the right eye, but the left showed

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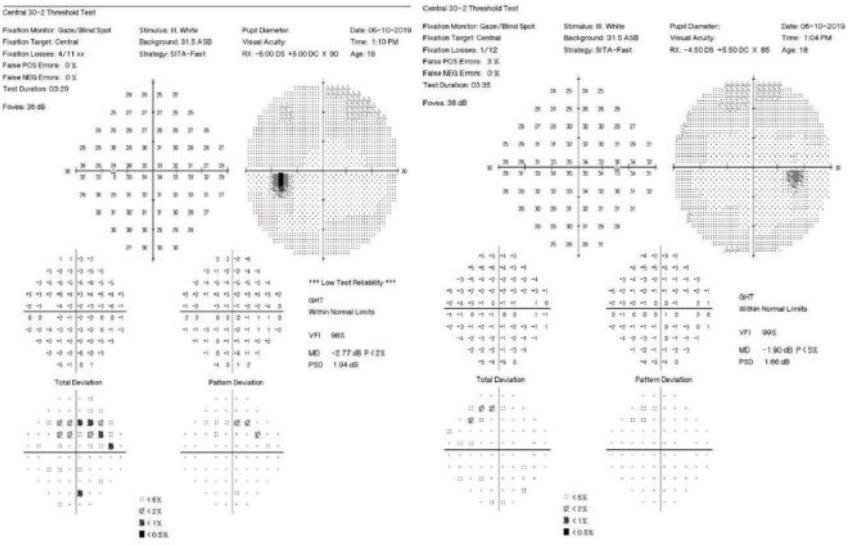
hyper-fluorescence of the superior disc during the arteriovenous phase (*Figures 3 and 4*). SD-OCT of the maculae showed no abnormal thickening or thinning and were generally symmetric between the right and left eyes (*Figure 5*). The results of the blood work revealed elevated rheumatoid arthritis factor and a slight elevation of circulating lymphocytes.

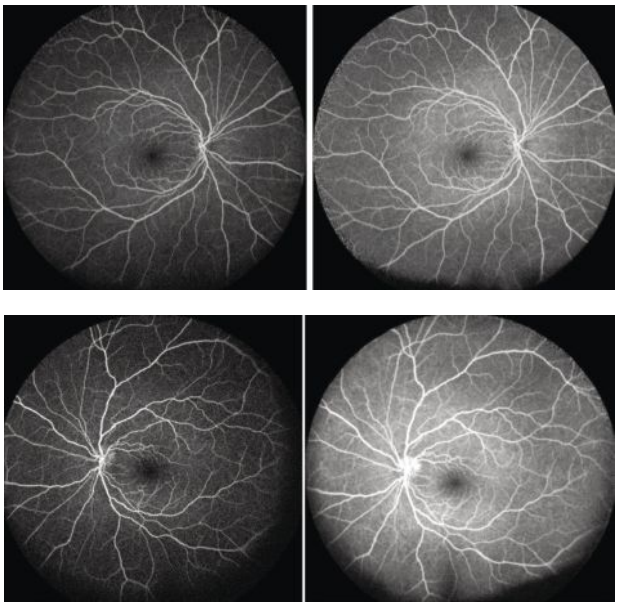
The MRI revealed a 1.4mm to 1.5mm nodular area of increased signal intensity projecting from the posterior left side of the anterior communicating artery, which is suspicious for a saccular aneurysm (*Figure 6*). Lumbar puncture was halted until further imaging, including a computed tomography angiography that could be performed to better delineate the aneurysm.

Differential Diagnoses

Unilateral disc edema is habitually bound with clinical suspicions of orbital compressive lesions, infection, inflammation and ischemia of the optic nerve.[1-6] True unilateral papilledema is a rarity among all the potential clinical causes of optic disc swelling.[2] As is the case with typical bilateral papilledema, unilateral papilledema occurs due to increased intracranial pressure.[2]

Compressive mass lesions of the optic nerves may arise in many different fashions, locations and sources. Rhabdomyosarcomas, optic nerve gliomas and optic sheath meningiomas are some of the neoplastic lesions to consider when dealing with the pediatric population. Rhabdomyosarcoma is the most common orbital malignancy in children and due to its sporadicity the cause is still unknown.[7] Optic nerve gliomas and optic nerve sheath meningiomas can both cause gradual unilateral vision loss, relative afferent pupillary defect and unilateral proptosis.[7,8]





Figs. 2-4. Above, 30-2 visual field testing shows scattered superior arcuate defect points in both eyes, but more so in the left. Center left, her right eye is normal on FA, but her left eye (bottom left) shows hyperfluorescent staining/pooling of the disc during the early and mid-phase.

Optic neuritis is an inflammatory morbidity that can manifest clinically as reduced visual acuity, with a painful swollen optic nerve.[1,9] Optic neuritis has an array of etiologies but two that stand out for this case are multiple sclerosis (MS) and rheumatoid arthritis. MS should be considered as a differential when young patients complain of diminished vision accompanied by optic disc swelling. The use of MRI with and without gadolinium will help confirm a diagnosis of MS if white matter plaques are present. Optic neuritis may be associated with elevated serum rheumatoid factor secondary to systemic arthropathies.[9]

Anterior ischemic optic neuropathy (AION) has two varieties that can cause loss of vision.[1,2] Both non-arteritic (NAION) and arteritic AION (AAION) manifest clinically as a loss of vision with a swollen nerve. NAION and AAION both typically affect patients over the age of 50 years old, the latter being

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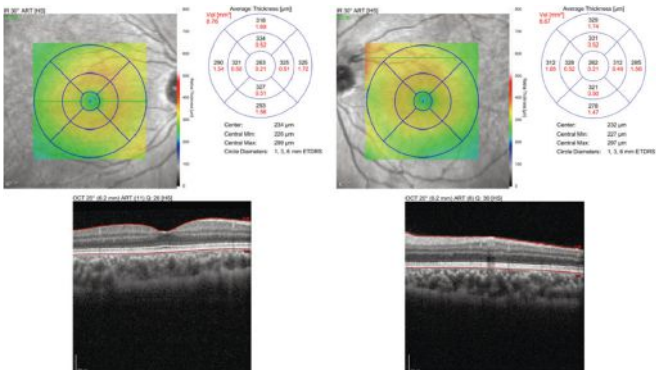

Fig. 5. OCT macular scan shows no structural abnormalities.

extremely rare in patients younger than 50 years.[1]

Infectious optic neuropathy may be caused by *Bartonella*, syphilis, herpes, tuberculosis (TB) and Lyme disease, just to name a few.[1,3] We ruled out these infectious causes with the appropriate serology including complete blood count with differential, QuantiFeron-TB Gold Plus test (Qiagen), *Treponema pallidum* antibody test and Lyme IgG/IgM test.

Diagnosis

This patient was ultimately diagnosed with left optic disc edema secondary to aneurysmal compression. Recent adult studies found that 35% of ruptured cerebral aneurysms were located at the anterior communicating artery.[10] The true prevalence of unruptured aneurysms remains unknown.[11] Patients with unruptured intracranial aneurysms may go on indefinitely with the diagnosis unbeknownst to them.

The optic nerves are susceptible to aneurysmal compression due to their proximal anatomical location to the assembly of blood vessels that make up anterior portion of the circle of Willis. This compression of the optic nerve may be perceived by patients as blurred or diminished vision unilaterally or bilaterally.[12]

The discovery of an intracranial aneurysm in a pediatric patient is uncommon. Literature reports show only 0.5% to 4.6 % of aneurysms occur in patients 18 years or younger.[13] In our case, the patient presented with signs and symptoms that may be associated with numerous etiologies. The anterior location of this patient's aneurysm also speaks to the rarity of the case. The pediatric population appears to be more prone to intracranial aneurysms within the posterior circulation.[13]

Research shows only 5% to 10% of pediatric intracranial aneurysms occur at the anterior cerebral and anterior communicating artery.[13]

Our patient is currently under close monitoring by both our retina clinic and a local neuro-ophthalmologist for consideration of surgical options for the saccular aneurysm. Treatment for intracranial aneurysms vary based on the etiology and morphology. Management options include observation, endovascular coiling therapy, and surgical clipping with or without bypass surgery.[13]

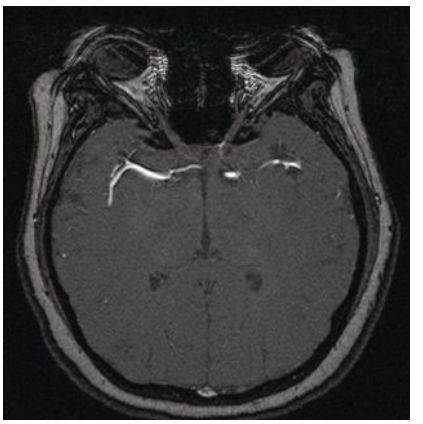

Fig. 5. An MRI exposes a 1.4mm to 1.5mm saccular aneurysm of the anterior communicating artery.

The patient and parent were educated to follow up with their pediatrician regarding the elevated serum rheumatoid factor and to return to our clinic after surgical consult to be monitored for any retinal sequalae of the compressive lesion. ■

Dr. Mancha is a primary care resident at the University of the Incarnate Word.

Dr. Scales is a board certified ophthalmologist specializing in retina and uveitis as well as a clinical professor at the University of the Incarnate Word.

Dr. Pizzimenti is a full-time faculty member at the University of the Incarnate Word in San Antonio, Texas, where he coordinates the Primary Care Residency Program.

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Living in a Purple State

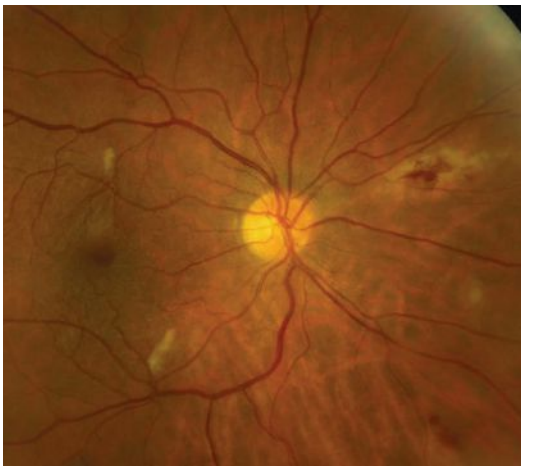
A patient with a compromised immune system has a strange visual presentation. Can her peripheral retina explain why? **By Mark T. Dunbar, OD**

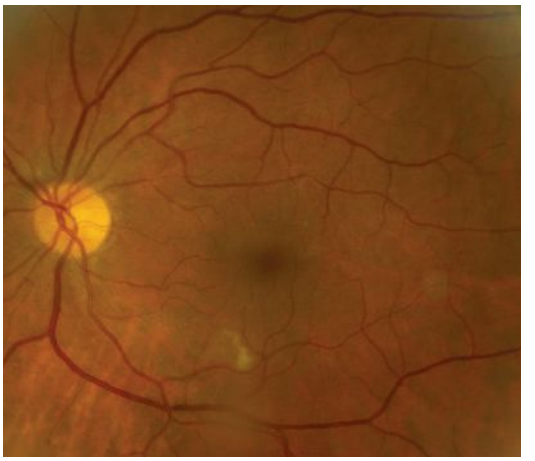
A 57-year-old Hispanic female presented with symptoms of seeing occasional purple spots in her vision for the past three weeks. She was not sure if this was limited to one eye. Beyond that, she stated her vision was good and that she only wore reading glasses.

Her medical history was significant for Type 2 diabetes and a recent HIV diagnosis. She was taking medications but did not know their names, nor did she know her CD4 count or her viral load.

Her best-corrected visual acuity was 20/20 OU. Confrontation visual fields were full-to-careful finger counting. The pupils were equally round and reactive; no afferent pupillary defect was seen. The anterior segment was unremarkable in both eyes. There were trace cells in the anterior vitreous in the right eye. The left eye was clear. Tensions by applanation measured 11mm Hg OU.

The optic nerves appeared healthy with small cups and good rim coloration and perfusion. We noted obvious retinal findings in both eyes (*Figures 1 and 2*).





Figs. 1 and 2. Note the peripheral retinal changes nasally in the right eye (above). Can you identify the finding in the posterior poles of both eyes?

Take the Retina Quiz

1. What do the peripheral retinal changes in the right eye represent?
a. Retinal vein occlusion.
b. Active chorioretinitis.
c. Active retinitis.
d. Inactive retinitis.

2. What do the white spots in the posterior pole represent?
a. Cotton wool spots.
b. Drusen.
c. Exudate.
d. Roth spots.

3. What is the diagnosis?
a. Toxoplasmosis.
b. Acute retinal necrosis.
c. Cytomegalovirus retinitis.
d. Proliferative diabetic retinopathy.

4. How should this patient be treated?
a. Observation.
b. Anti-VEGF injection.
c. Begin anti-viral therapy.
d. Begin antibiotic therapy.

For answers, see page 114.

Diagnosis

The nasal peripheral retinal changes in the right eye represents active cytomegalovirus (CMV) retinitis. CMV is one of the herpes viruses that infects most adults.[1] Almost all of us have been exposed to CMV in our lifetime but will have no symptoms of infection because our immune system is strong enough to keep the virus in check.[1] But, in people with weakened immune systems, like our HIV patient, the virus can reactivate and spread to the retina, which can lead to vision-threatening complications.[1]

CMV retinitis is the most common ocular complication seen in patients with HIV.[1] CMV retinitis develops in patients whose CD4+ lymphocyte T-Cell (CD4) counts are below 50 and usually even much lower than that. With the

advent of highly active antiretroviral therapy (HAART), CMV has virtually disappeared, with a dramatic 55% to 95% decline in the number of CMV retinitis cases.[1]

Discussion

CMV retinitis may be present anywhere in the retina.[1] Posterior pole lesions have a characteristic white, hemorrhagic appearance with retinal necrosis and edema, while the peripheral lesions are more indolent and nonhemorrhagic.[1] The active retinitis generally follows the path of the retinal vasculature centripetally.[1] In rare instances, CMV can also present as a frosted-branch angiitis in which there is a fulminant retinal vasculitis and periphlebitis, giving the fundus a frosted quality to the perivascular exudate.[1]

Vitreous cells will be present in active CMV but not nearly to the extent seen in, for example, patients with active toxoplasmosis where the vitritis can be so dense the active retinal lesion can be difficult to observe, giving it a "head lights in the fog" appearance.[1,2] In contrast, patients with CMV do not have a robust immune system so they are not able to mount a significant immune response; therefore, the vitreous inflammation is minimal and the retinitis can be easily seen.[1]

What about the white lesions in the posterior pole of each eye; is that CMV retinitis too? Luckily they're not CMV but rather cotton-wool spots (CWS), which can easily be mistaken for early CMV. The CWS are smaller than CMV lesions and vitreous inflammation will not be present. CWS and even retinal hemorrhages are commonly seen in severely immunocompromised patients with HIV.[2] In fact, CWS and retinal hemorrhages are part of the spectrum of HIV retinopathy.[2] Though the etiology is not completely understood, researchers say immunocompromised patients have increased plasma viscosity that results in immune-complex deposition.[2] This is thought to occur from a direct cytopathic effect of the virus on the vascular endothelium.[2]

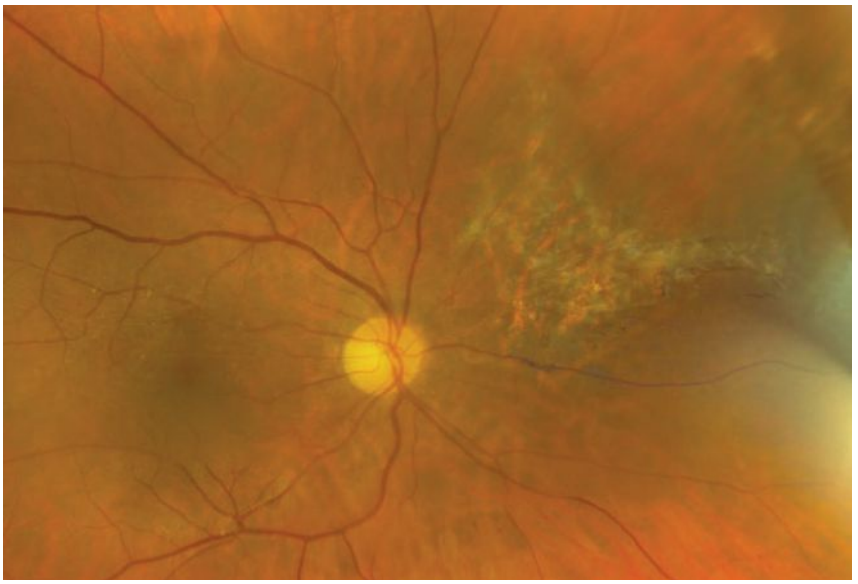


Fig. 3. Here is our patient's retina five years after initial diagnosis.

Therapeutic Options

Treatment for CMV retinitis has become fairly standardized with the advent of anti-viral medications including ganciclovir, foscarnet, valganciclovir and cidofovir. Treatment delivery methods include intravenous infusion, oral therapy, intravitreous injection and ganciclovir intraocular implant.[3] Specific anti-CMV treatment is individualized for each patient based on the location and severity of the retinitis, level of underlying immune suppression, concomitant medications and ability to comply with treatment.[3] Therapy is induced at high doses for two to three weeks or until the retinitis stabilizes followed by a maintenance dose.[3] Improved patient survival with HAART has resulted in a paradigm shift away from short-term disease suppression in the early days of treatment to now discontinuation of anti-viral therapy once immune recovery is achieved.[3]

Clearly, our patient is severely immunocompromised. She did not know her CD4 and had only been recently diagnosed after a bout with pneumonia. She had an immediate intravitreal injection of ganciclovir and subsequently had multiple ganciclovir implants over several years. Eventually, her immune system recovered enough that she was able to discontinue anti-viral therapy for the CMV. She was able to maintain excellent visual acuity in both eyes and we continue to follow her on an annual basis (*Figure 3*). ■

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Meetings + Conferences

April

■ **2-7.** *Forum on Optometry.* Marriott Hotel, Los Angeles. Hosted by: PSS EyeCare. Key faculty: Ron Melton, Randall Thomas, Jerry Sherman, Pinakin Davey. CE hours: 18. For more information, contact Sonia Kumari at 203-415-3087 or education@psseyecare.com.

■ **3-4.** *Envision Conference West 2020.* University of Texas Health Science Center at San Antonio, San Antonio, TX. Hosted by: Envision University. CE hours: Total: 32, max. per OD: 11. For more information, contact Michael Epp at michael.epp@envisionus.com or visit www.envisionconference.org.

■ **3-5.** *OAO 2020 Symposium and InfoMart.* Sheraton Centre Toronto Hotel, Toronto, Ontario, Canada. Hosted by: Ontario Association of Optometrists. Key faculty: Nathan Lighthizer, Michael Cooper, Greg Caldwell, Brad Sutton, Alan Glazier, Shirley Blanc. CE hours: Total: 52, max. per OD: 17. For more information, contact Cheryl Neave at cneave@optom.on.ca, 905-836-3522 ext. 243 or visit www.optom.on.ca/symposium.

■ **3-5.** *Sports Vision.* OEP-NEC, Timonium, MD. Hosted by: OEPF. Key faculty: Paul Harris, Geoffrey Heddle. CE hours: 21. For more information, contact Karen Ruder at karen.ruder@oepf.org, 410-561-3791 or visit www.oepf.org.

■ **3-5.** *UAB School of Optometry Primary Eye Care Update.* UAB Rosewood Hall, Birmingham, AL. hosted by: UAB School of Optometry. CE hours: 18. For more information, contact Kathryn Trammell at ktram@uab.edu, 205-934-5701 or visit uab.edu/optometry/ce.

■ **3-5.** *New Mexico Optometric Association Annual Convention.* Sandia Resort, Albuquerque, NM. Hosted by: New Mexico Optometric Association. Key faculty: Jeffry Gerson, Jordan Keith, Marc Bloomenstein, Danica Marrelli, Henry Hudson, Craig Clatnoff. CE hours: 22. For more information, contact Richard Montoya at newmexicooptometry@gmail.com, 575-751-7242 or visit www.newmexicooptometry.org.

■ **4-5.** *CE in the Southwest.* Westin Galleria Dallas. Hosted by: University of Houston College of Optometry & UIW Rosenberg School of Optometry. Key faculty: Marcus Gonzales, Sandra Fortenberry. CE hours: 16. For more information, contact optce@central.uh.edu, 713-743-1900 or visit ce.opt.uh.edu.

■ **4-5**. *UMSL Nutrition & the Eye Symposium.* UMSL Conference Center, St. Louis. Hosted by: University of Missouri St. Louis-College of Optometry and the Ocular Wellness and Nutrition Society. Key faculty: Stuart Richer, Martin Pall, Christopher Putnam, Thomas Levy, Rebecca Sieburth. CE hours: 12. For more information, contact Erin Schaeffer at schaeffere@umsl.edu or visit optometry.umsl.edu/ce courses/index.html.

■ **5.** *Opioid Continuing Education Event.* Midwestern University, St. Glendale, AZ. Hosted by: Arizona College of Optometry. Key faculty: Arizona College of Optometry faculty members. CE hours: 3. For more information, contact Christina Esposito at azcopt-ce@midwestern.edu or call 623-806-7271.

■ **11-18.** *Symposium on Ocular Disease Marriott Hotel.* Tyson's Corner, VA. Hosted by: PSS EyeCare. Key faculty: Ron Melton, Randall Thomas, Jerome Sherman, Deepak Gupta. CE hours: 20. For more information, contact Sonia Kumari at 203-415-3087, education@psseyecare.com or visit www.psseyecare.com.

■ **15-19.** *Vision by Design.* Hyatt Regency Bellevue, Bellevue, WA. Hosted by: The American Academy of Orthokeratology and Myopia Control. Key faculty: Randy Kojima, Pat Caroline, Langis Michaud, Earl Smith, Thomas Aller, April Jasper. CE hours: 30+. For more information, contact Sarah Witt at 630-659-8371, oaaast@gmail.com or visit www.orthokmeeting.com.

■ **16-19.** *New Technologies & Treatments in Eye Care/OCCRS.* Omni Barton Creek, Austin. Hosted by: Review Education Group/OCCRS. Key faculty: Paul Karpecki. CE hours: Total: 28. For more information, contact Review Meetings at reviewmeetings@jhihealth.com, 866-658-1772 or visit www.reviewofoptometry.com/events.

■ **17-18.** *FL-AAO Educational Meeting.* Mission Inn, Howey-In-The-Hills, FL. Hosted by: Florida Chapter AAO. Key faculty: Joe Pizzimenti, Julie Tyler, John McClane. CE hours: 14. For more information, contact Art Young at eyeguy4123@msn.com, 601-946-2174 or visit www.aaopt.org/membership/us-and-international-chapters/flchapter.

■ **17-19.** *Morgan-Sarver Symposium.* DoubleTree by Hilton Berkeley Marina, Berkeley, CA. Hosted by: University of Berkeley School of Optometry. Key faculty: Anthony Realini, Lisa Prokopich, Pete Kollbaum, Carl Jacobsen, Glen Ozawa. CE hours: 21. For more information, contact Lyudmila Martello at optoce@berkeley.edu, 510-642-6547 or visit optometry.berkeley.edu/continuing-education/morgan-symposium.

■ **17-19.** *Indiana Optometry's Meeting.* Embassy Suites Conference Center, Noblesville, IN. Hosted by: Indiana Optometric Association. Key faculty: Edward Bennett, Nathan Lighthizer, Elizabeth Steele, James Stringham. CE hours: 15. For more information, contact Bridget Sims at blsims@ioa.org, 317-237-3560 or visit www.ioa.org.

■ **17-19.** *SCO's Spring CE Weekend.* Southern College of Optometry Academic Complex, Memphis. Hosted by: Southern College of Optometry. CE hours: Total: 28, max. per OD: 20. For more information, contact Jeanie Snider at jsnider@sco.edu, 901-722-3397 or visit www.sco.edu/continuing-education.

■ **18-19.** *Spring Seminar.* Rosenberg School of Optometry, San Antonio, TX. Hosted by: UIW Rosenberg School of Optometry. CE hours: 16. For more information, contact Holly Greene at hfrost@uiwtx.edu, 210-283-6856 or visit optometry.uiw.edu/continuing-education/index.html.

■ **18-19.** *Miami Nice Education Seminar.* Hilton Miami Airport, Miami. Hosted by: Miami-Dade Optometric Physicians Association. Key faculty: Steven Ferrucci, David Rouse, Paul

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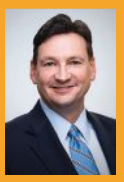

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Chous, Woods. CE hours: 16. For more information, contact Stephen Morris at steve.morris.od@gmail.com, 305-342-5473 or visit www.miami.eyes.org.

■ **18-19.** *OEC Twin Cities 2020*. Intercontinental–Saint Paul Riverfront, East Saint Paul, MN. Hosted by: Optometric Education Consultants. Key faculty: Tracy Offerdahl-McGowan, Leonid Skorin Jr., Joseph Sowka, Greg Caldwell. CE hours: 18. For more information, contact Vanessa McDonald at optoec@gmail.com, 954-612-4142 or visit www.optometricedu.com/twin-cities-2020/attendee.

■ **19.** *SUNY Breakfast & Learn*. SUNY College of Optometry, New York. Hosted by: SUNY College of Optometry. CE hours: 4. For more information, contact Betsy Torres at ce@sunyopt.edu, 212-938-5830 or visit www.sunyopt.edu/cpe.

■ **21-25.** *COVD Annual Meeting*. The Westin Harbour Castle Toronto, Toronto, Ontario, Canada. Hosted by: College of Optometrists in Vision Development. CE hours: Total: 41, maximum per OD: 30.5. For more information, contact Lauren Bissetta at info@covd.org, 330-995-0718 or visit covd2020.org.

■ **23-24**. *WOA Spring Seminar*. Marriott Madison West, Middleton, WI. Hosted by: Wisconsin Optometric Association. Key faculty: Greg Caldwell, Stuart Richer, Gerald Clark. CE hours: 14. For more information, contact Joleen Breunig at joleen@woa-eyes.org, 608-824-2200 or visit www.woa-eyes.org.

■ **23-25.** *KY Optometric Association Spring Conference*. Hyatt Regency Hotel & Lexington Convention Center, Lexington, KY. Hosted by: Kentucky Optometric Association. CE hours: 20. For more information, contact Sarah Unger at sarah@kyeyes.org, 502-875-3516 or visit www.kyeyes.org.

■ **23-26.** *ArOA Spring Convention*. Statehouse Convention Center, Little Rock, AR. Hosted by: Arkansas Optometric Assn. CE hours: 20. For more information, contact Debbie Henley at aroa@arkansasoptometric.org, 501-661-7675 or visit arkansasoptometric.org/conventions.html.

■ **24-25.** *Couer d'Alene Conference*. The Coeur d'Alene Resort, Coeur d'Alene, ID. Hosted by: Pacific U. College of Optometry. Key faculty: Justin Schweitzer, Tracy Doll, Hannah Shinoda, Tad Buckingham, Fraser Horn. CE hours: 10. For more information, contact Michelena "Miki" Buckingham at mikibuckingham@pacificu.edu, 503-352-2985 or visit www.pacificu.edu/academics/colleges/college-optometry/continuing-education.

■ **24-26.** *Arizona Optometric Association Spring Congress*. JW Marriott Tucson Starr Pass Resort, Tucson, AZ. Hosted by: Arizona Optometric Assn. For more information, contact Kate Diedrickson at kate@azoa.org or visit www.azoa.org/connect.

To list your meeting, please send the details to:
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Finding the Nerve

A patient with CN VII damage can't get her lid to cover her cornea in one eye.

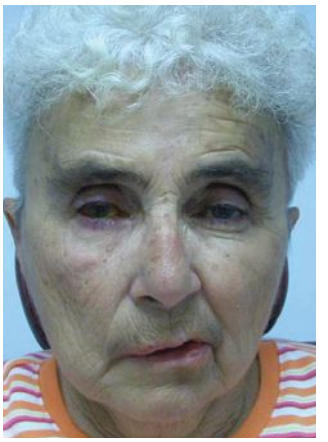
By Andrew S. Gurwood, OD

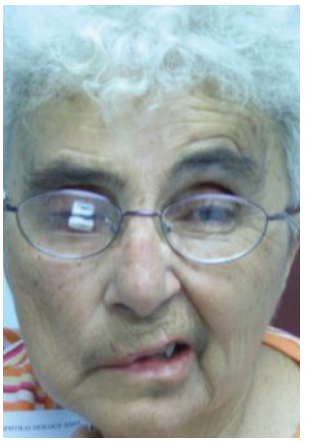
History

A 77-year-old Caucasian female was referred to the clinic for a corneal evaluation secondary to worsening lagophthalmos produced by cranial nerve (CN) VII damage in her left eye. The issue occurred as a result of the removal of an acoustic neuroma. Her systemic history was remarkable for acoustic neuroma which was removed surgically approximately two weeks prior.

Her previous ocular history was contributory as the lagophthalmos was aggravated by an old cosmetic ptosis repair to her left eye three years earlier.

In response to the inability of the lid to cover the cornea, she was referred to an ear, nose and throat specialist who placed gold weights into the superior lid of her left eye. When the desired effect was inadequate, the ophthalmology department was consulted. She denied allergies of any kind.





An external exam shows our patient both with eyes opened (at left) and closed. Note the inability to fully close her left eye. Can these images help identify her underlying condition?

Diagnostic Data

Her best corrected entering visual acuities were 20/20, OU at distance and near. An external examination uncovered poor facial nerve function of her left eye with an inability to voluntarily close it or adequately cover the cornea using the Bell's phenomenon. No evidence of afferent pupillary defect was noted. The pertinent findings are demonstrated in the photographs. Goldmann applanation tonometry measured 15mm Hg OU.

The dilated fundus findings were normal peripherally and centrally with normal nerves and maculae.

Your Diagnosis

Does the case presented require any additional tests? What would be your diagnosis? What's the most likely prognosis? To find out, visit www.reviewofoptometry.com. ■

Retina Quiz Answers *(from page 100):* 1) c; 2) a; 3) c; 4) c.

Next Month in the Mag

Coming in April, *Review of Optometry* will present its annual Cornea Report.

Topics include:

- *Put The Breaks on Contact Lens Dropout*
- *Corneal Crosslinking in Optometry: What it Can Do and Who Can Do it?*
- *Gear Up for Corneal Foreign Body Removal*
- *Understanding Corneal Nerve Function—and Dysfunction* (Earn 2 CE Credits)

Also in this issue:

- *Incorporating Glaucoma Treatment into Any OD Practice*
- *How to Work Up Lid Ptosis*

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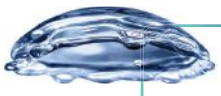

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