Glaucoma Grand Rounds: When a Surgery–and Surgeon–Fails, *P. 86*

REVIEW of OPTOMETRY®

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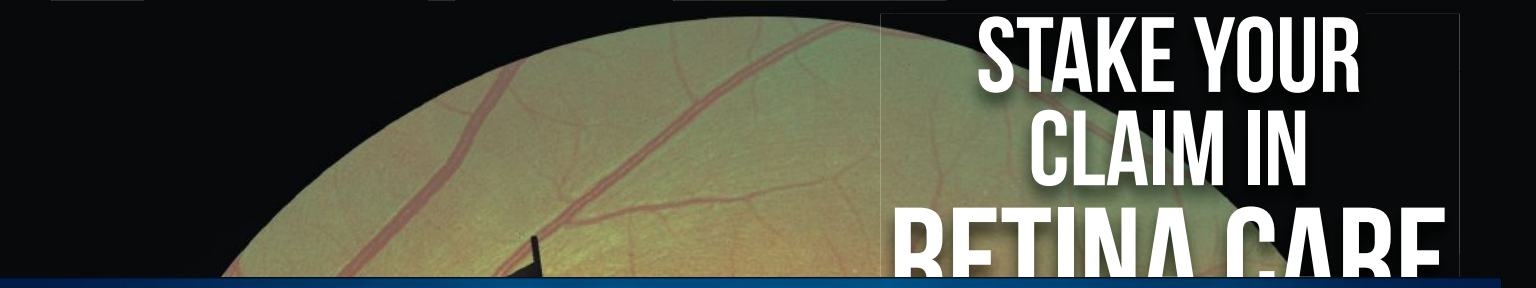
Leadership in clinical care

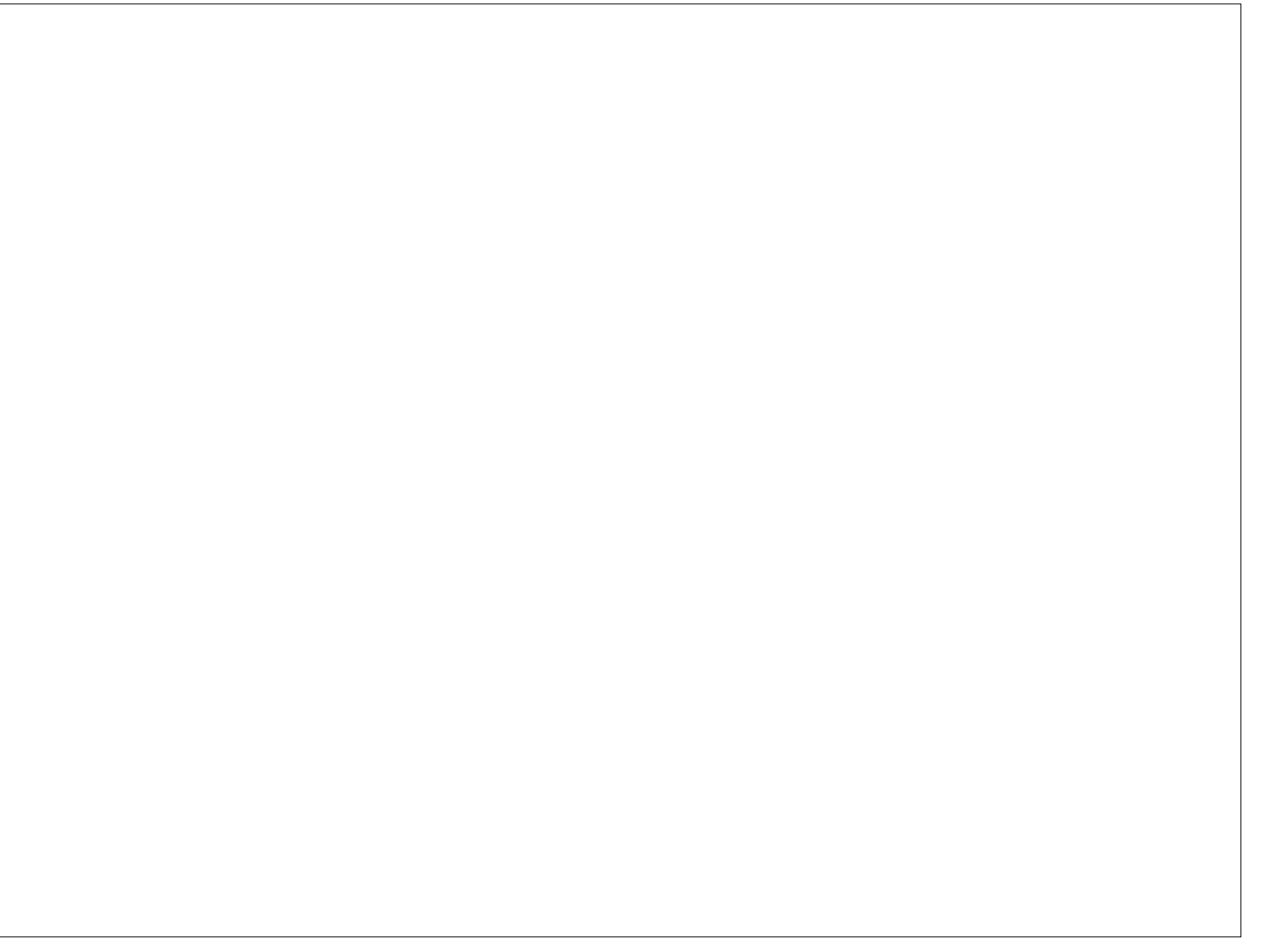
UNDERSTANDING TODAY'S PROGRESSIVES

Help patients choose and use these lenses with fewer hassles and greater success.

PAGE 34



STAKE YOUR CLAIM IN RETINA CARE



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STAKE YOUR CLAIM IN RETINA CARE

With more patients in need than ophthalmologists can handle, optometry needs to gain ground for the good of all.

12th Annual Retina Report

VYZULTA®
(latanoprostene bunod ophthalmic solution), 0.024%

THE HORSEPOWER YOU NEED TO LOWER IOP

Powerful IOP reduction with excellent tolerability[1,2]

VYZULTA delivered **up to 9.1 mmHg mean IOP reduction** from baseline in pivotal trials.[1,2*]

TAKE A TEST RIDE AT VYZULTAHCP.COM

*Pivotal study designs: Two Phase 3, randomized, multicenter, parallel-group studies, APOLLO and LUNAR, evaluating noninferiority of once-daily VYZULTA vs twice-daily timolol maleate 0.5% in patients with open-angle glaucoma or ocular hypertension. Primary endpoint was IOP measured at 9 assessment time points in study eye. APOLLO (VYZULTA, n=284; timolol, n=133) and LUNAR (VYZULTA, n=278; timolol, n=136).[2,3]

INDICATION

VYZULTA® (latanoprostene bunod ophthalmic solution), 0.024% is indicated for the reduction of intraocular pressure (IOP) in patients with open-angle glaucoma or ocular hypertension.

IMPORTANT SAFETY INFORMATION

- Increased pigmentation of the iris and periorbital tissue (eyelid) can occur. Iris pigmentation is likely to be permanent
- Gradual changes to eyelashes, including increased length, increased thickness, and number of eyelashes, may occur. These changes are usually reversible upon treatment discontinuation
- Use with caution in patients with a history of intraocular inflammation (iritis/uveitis). VYZULTA should generally not be used in patients with active intraocular inflammation
- Macular edema, including cystoid macular edema, has been reported during treatment with prostaglandin analogs. Use with caution in aphakic patients, in pseudophakic patients with a torn posterior lens capsule, or in patients with known risk factors for macular edema
- There have been reports of bacterial keratitis associated with the use of multiple-dose containers of topical ophthalmic products that were inadvertently contaminated by patients
- Contact lenses should be removed prior to the administration of VYZULTA and may be reinserted 15 minutes after administration
- Most common ocular adverse reactions with incidence ≥2% are conjunctival hyperemia (6%), eye irritation (4%), eye pain (3%), and instillation site pain (2%)

For more information, please see Brief Summary of full Prescribing Information on adjacent page.

References: 1. VYZULTA Prescribing Information. Bausch & Lomb Incorporated. **2.** Weinreb RN, Scassellati Sforzolini B, Vittitow J, Liebmann J. Latanoprostene bunod 0.024% versus timolol maleate 0.5% in subjects with open-angle glaucoma or ocular hypertension: the APOLLO study. *Ophthalmology.* 2016;123(5):965-973. **3.** Medeiros FA, Martin KR, Peace J, Scassellati Sforzolini B, Vittitow JL, Weinreb RN. Comparison of latanoprostene bunod 0.024% and timolol maleate 0.5% in open-angle glaucoma or ocular hypertension: the LUNAR study. *Am J Ophthalmol.* 2016;168:250-259.

BAUSCH+LOMB

BRIEF SUMMARY OF PRESCRIBING INFORMATION

This Brief Summary does not include all the information needed to use VYZULTA safely and effectively. See full Prescribing Information for VYZULTA.

VYZULTA® (latanoprostene bunod ophthalmic solution), 0.024%, for topical ophthalmic use.

Initial U.S. Approval: 2017

1 INDICATIONS AND USAGE

VYZULTA® (latanoprostene bunod ophthalmic solution) 0.024% is indicated for the reduction of intraocular pressure (IOP) in patients with open-angle glaucoma or ocular hypertension.

4 CONTRAINDICATIONS

None

5 WARNINGS AND PRECAUTIONS

5.1 Pigmentation

VYZULTA® (latanoprostene bunod ophthalmic solution), 0.024% may cause changes to pigmented tissues. The most frequently reported changes with prostaglandin analogs have been increased pigmentation of the iris and periorbital tissue (eyelid).

Pigmentation is expected to increase as long as latanoprostene bunod ophthalmic solution is administered. The pigmentation change is due to increased melanin content in the melanocytes rather than to an increase in the number of melanocytes. After discontinuation of VYZULTA, pigmentation of the iris is likely to be permanent, while pigmentation of the periorbital tissue and eyelash changes are likely to be reversible in most patients. Patients who receive prostaglandin analogs, including VYZULTA, should be informed of the possibility of increased pigmentation, including permanent changes. The long-term effects of increased pigmentation are not known.

Iris color change may not be noticeable for several months to years. Typically, the brown pigmentation around the pupil spreads concentrically towards the periphery of the iris and the entire iris or parts of the iris become more brownish. Neither nevi nor freckles of the iris appear to be affected by treatment. While treatment with VYZULTA® (latanoprostene bunod ophthalmic solution), 0.024% can be continued in patients who develop noticeably increased iris pigmentation, these patients should be examined regularly *[see Patient Counseling Information (17) in full Prescribing Information]*.

5.2 Eyelash Changes

VYZULTA may gradually change eyelashes and vellus hair in the treated eye. These changes include increased length, thickness, and the number of lashes or hairs. Eyelash changes are usually reversible upon discontinuation of treatment.

5.3 Intraocular Inflammation

VYZULTA should be used with caution in patients with a history of intraocular inflammation (iritis/uveitis) and should generally not be used in patients with active intraocular inflammation as it may exacerbate this condition.

5.4 Macular Edema

Macular edema, including cystoid macular edema, has been reported during treatment with prostaglandin analogs. VYZULTA should be used with caution in aphakic patients, in pseudophakic patients with a torn posterior lens capsule, or in patients with known risk factors for macular edema.

5.5 Bacterial Keratitis

There have been reports of bacterial keratitis associated with the use of multiple-dose containers of topical ophthalmic products. These containers had been inadvertently contaminated by patients who, in most cases, had a concurrent corneal disease or a disruption of the ocular epithelial surface.

5.6 Use with Contact Lens

Contact lenses should be removed prior to the administration of VYZULTA because this product contains benzalkonium chloride. Lenses may be reinserted 15 minutes after administration.

6 ADVERSE REACTIONS

The following adverse reactions are described in the Warnings and Precautions section: pigmentation (5.1), eyelash changes (5.2), intraocular inflammation (5.3), macular edema (5.4), bacterial keratitis (5.5), use with contact lens (5.6).

6.1 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.

VYZULTA was evaluated in 811 patients in 2 controlled clinical trials of up to 12 months duration. The most common ocular adverse reactions observed in patients treated with latanoprostene bunod were: conjunctival hyperemia (6%), eye irritation (4%), eye pain (3%), and instillation site pain (2%). Approximately 0.6% of patients discontinued therapy due to ocular adverse reactions including ocular hyperemia, conjunctival irritation, eye irritation, eye pain, conjunctival edema, vision blurred, punctate keratitis and foreign body sensation.

8 USE IN SPECIFIC POPULATIONS

8.1 Pregnancy

Risk Summary

There are no available human data for the use of VYZULTA during pregnancy to inform any drug associated risks.

Latanoprostene bunod has caused miscarriages, abortion, and fetal harm in rabbits. Latanoprostene bunod was shown to be abortifacient and teratogenic when administered intravenously (IV) to pregnant rabbits at exposures ≥ 0.28 times the clinical dose. Doses ≥ 20 µg/kg/day (23 times the clinical dose) produced 100% embryofetal lethality. Structural abnormalities observed in rabbit fetuses included anomalies of the great vessels and aortic arch vessels, domed head, sternebral and vertebral skeletal anomalies, limb hyperextension and malrotation, abdominal distension and edema. Latanoprostene bunod was not teratogenic in the rat when administered IV at 150 mcg/kg/day (87 times the clinical dose) *[see Data]*.

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 latanoprostene bunod daily by intravenous injection on gestation days 7 through 19, to target the period of organogenesis. The doses administered ranged from 0.24 to 80 mcg/kg/day. Abortion occurred at doses ≥ 0.24 mcg/kg/day latanoprostene bunod (0.28 times the clinical dose, on a body surface area basis, assuming 100% absorption). Embryofetal lethality (resorption) was increased in latanoprostene bunod treatment groups, as evidenced by increases in early resorptions at doses ≥ 0.24 mcg/kg/day and late resorptions at doses ≥ 6 mcg/kg/day (approximately 7 times the clinical dose). No fetuses survived in any rabbit pregnancy at doses of 20 mcg/kg/day (23 times the clinical dose) or greater. Latanoprostene bunod produced structural abnormalities at doses ≥ 0.24 mcg/kg/day (0.28 times the clinical dose). Malformations included anomalies of sternum, coarctation of the aorta with pulmonary trunk dilation, retroesophageal subclavian artery with absent brachiocephalic artery, domed head, forepaw hyperextension and hindlimb malrotation, abdominal distention/edema, and missing/fused caudal vertebrae.

An embryofetal study was conducted in pregnant rats administered latanoprostene bunod daily by intravenous injection on gestation days 7 through 17, to target the period of organogenesis. The doses administered ranged from 150 to 1500 mcg/kg/day. Maternal toxicity was produced at 1500 mcg/kg/day (870 times the clinical dose, on a body surface area basis, assuming 100% absorption), as evidenced by reduced maternal weight gain. Embryofetal lethality (resorption and fetal death) and structural anomalies were produced at doses ≥ 300 mcg/kg/day (174 times the clinical dose). Malformations included anomalies of the sternum, domed head, forepaw hyperextension and hindlimb malrotation, vertebral anomalies and delayed ossification of distal limb bones. A no observed adverse effect level (NOAEL) was established at 150 mcg/kg/day (87 times the clinical dose) in this study.

8.2 Lactation

Risk Summary

There are no data on the presence of VYZULTA 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 VYZULTA, and any potential adverse effects on the breastfed infant from VYZULTA.

8.4 Pediatric Use

Use in pediatric patients aged 16 years and younger is not recommended because of potential safety concerns related to increased pigmentation following long-term chronic use.

8.5 Geriatric Use

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

13 NONCLINICAL TOXICOLOGY

13.1 Carcinogenesis, Mutagenesis, Impairment of Fertility

Latanoprostene bunod was not mutagenic in bacteria and did not induce micronuclei formation in the *in vivo* rat bone marrow micronucleus assay. Chromosomal aberrations were observed *in vitro* with human lymphocytes in the absence of metabolic activation.

Latanoprostene bunod has not been tested for carcinogenic activity in long-term animal studies. Latanoprost acid is a main metabolite of latanoprostene bunod. Exposure of rats and mice to latanoprost acid, resulting from oral dosing with latanoprost in lifetime rodent bioassays, was not carcinogenic.

Fertility studies have not been conducted with latanoprostene bunod. The potential to impact fertility can be partially characterized by exposure to latanoprost acid, a common metabolite of both latanoprostene bunod and latanoprost. Latanoprost acid has not been found to have any effect on male or female fertility in animal studies.

13.2 Animal Toxicology and/or Pharmacology

A 9-month toxicology study administered topical ocular doses of latanoprostene bunod to one eye of cynomolgus monkeys: control (vehicle only), one drop of 0.024% bid, one drop of 0.04% bid and two drops of 0.04% per dose, bid. The systemic exposures are equivalent to 4.2-fold, 7.9-fold, and 13.5-fold the clinical dose, respectively, on a body surface area basis (assuming 100% absorption). Microscopic evaluation of the lungs after 9 months observed pleural/subpleural chronic fibrosis/inflammation in the 0.04% dose male groups, with increasing incidence and severity compared to controls. Lung toxicity was not observed at the 0.024% dose.

U.S. Patent Numbers: 7,273,946; 7,629,345; 7,910,767; 8,058,467.

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ODs Fire Back at MIPS Report's So-So Marks

Advocates call out the 2017 data from the first year of the CMS program as skewed.

A recent analysis suggesting ophthalmologists who participated in the Merit-based Incentive Payment System (MIPS) in 2017 earned higher scores than other physicians—including optometrists—is drawing fire among the profession and its advocates.

The paper stated that ophthalmologists had significantly higher MIPS scores in all categories compared with ODs and other physicians.[1] Specifically, the paper reports that mean final MIPS scores for ophthalmologists in 2017 were 10 to 20 points higher than optometry and other physician specialties in both group and individual reporting. The authors also noted that ophthalmologists are more likely to perform at a higher level in MIPS.

Citing the AOA's 2018 American Eye-Q Survey, optometrist Jeffrey Michaels suggests that ophthalmology knows optometry is the preferred source for eye care in America and that optometrists are trusted more than any other source for ocular health exams.

"Ophthalmology is grasping at any conclusion they can to prove their worth, and the article contradicts the viewpoint of their own most experienced ophthalmologists," he says. "As optometrists continue to expand scope of practice across the country, the opposition begins to show desperation."

Advocates say the data, which came out in 2017 when the Centers for Medicare & Medicaid Services rolled out the program, should be taken with a grain of salt.

That year, CMS granted widespread flexibilities to help ease providers into the MIPS program, making available a "pick your pace" participation that allowed three reporting options for clinicians, including test, partial and full-year reporting.[2] The first option allowed providers to report "some data" to avoid a negative adjustment and gain familiarity with the program, while the latter options stepped up data requirements, incentives and penalties.[2]

> **"Ophthalmology is grasping at any conclusion they can to prove their worth, and the article contradicts the viewpoint of their own most experienced ophthalmologists."**
>
> **—Jeffrey Michaels, OD**

Additionally, the overall performance threshold for MIPS was established at a relatively low level of three points out of a possible 100, and ODs earned far above that with an average of 55 points.[2] The AOA affirms nearly every OD who participated in MIPS during 2017 earned a passing score.[2]

Although performance for MIPS began broadly in 2017, CMS gradually rolled out the program with that year acting as a transitional period, but also narrowed criteria for eligible providers based on strong feedback.[2]

The group reporting database included 6,776 ophthalmologists, 12,206 optometrists and 231,285 other physicians. The individual clinician database included 8,595 ophthalmologists, 15,193 optometrists and 293,210 other physicians.

"MIPS is still relatively new and not well understood by most doctors of optometry," says Chris Wroten, OD. "As is true for all health care providers, assessing quality of care based on whether a box in an EHR was checked or not doesn't ensure the accuracy of the care reported, nor does it ensure the quality of care provided."

Ophthalmology has a built-in advantage in its exclusive IRIS registry, which streamlines reporting for MIPS and enhances scoring, Dr. Wroten adds. Additionally, several eye care quality measures developed through the IRIS registry and then approved by CMS exclude optometry's participation and compel ODs to use other topped-out quality measures with maximum scores that are capped at 70% or less than the maximum available for other measures.

"Further, CMS has acknowledged from the start of MIPS that it favors providers in large practices that have more resources to assist with compliance and reporting requirements. A much higher percentage of MIPS-eligible ophthalmologists practice within hospital systems, multispecialty groups and large eye care clinics compared with doctors of optometry, further explaining any potential discrepancy in MIPS scores," Dr. Wroten says.

1. Sheth N, French DD, Tanna AP. Merit-based incentive payment system scores in ophthalmology and optometry. Ophthalmology. 2021;128(5):793-5.

2. AOA faults ophthalmology journal analysis of optometrist, ophthalmologist MIPS scores. American Optometric Association. www.aoa.org/news/practice-management/perfect-your-practice/aoa-faults-ophthalmology-journal-mips-study?sso=y. May 13, 2021. Accessed May 17, 2021.

TIME IS RUNNING OUT!

UPGRADE CURRENT AIR OPTIX® WEARERS WITH NO REFIT REQUIRED![1,2]

THE TRIPLE UPGRADE

HYDRAGLYDE® MOISTURE MATRIX

Longer-lasting lens surface moisture[3-5*]

MORE LUBRICATION

Increased surface wetting and lubricating effects[6-7**]

IMPROVED MARKINGS

Better scribe mark visibility and reduced markings

Bring more lens surface moisture[3-5*] and outstanding comfort[8,9†] to their AIR OPTIX® performance now!

*vs. AIR OPTIX® AQUA contact lenses.
**AIR OPTIX® plus HydraGlyde® sphere lenses compared to AIR OPTIX® AQUA and O2 OPTIX® sphere lenses.
†Based on clinical studies with AIR OPTIX® AQUA, AIR OPTIX® AQUA Multifocal and AIR OPTIX® for Astigmatism contact lenses.

Important information for AIR OPTIX® plus HydraGlyde® (lotrafilcon B) contact lenses: For daily wear or extended wear up to 6 nights for near/far-sightedness. Risk of serious eye problems (i.e., corneal ulcer) is greater for extended wear. In rare cases, loss of vision may result. Side effects like discomfort, mild burning or stinging may occur.
Important information for AIR OPTIX® plus HydraGlyde® Multifocal (lotrafilcon B) contact lenses: For daily wear or extended wear up to 6 nights for near/far-sightedness and/or presbyopia. Risk of serious eye problems (i.e., corneal ulcer) is greater for extended wear. In rare cases, loss of vision may result. Side effects like discomfort, mild burning or stinging may occur.
Important information for AIR OPTIX® plus HydraGlyde® for Astigmatism (lotrafilcon B) contact lenses: For daily wear or extended wear up to 6 nights for near/far sightedness and astigmatism. Risk of serious eye problems (i.e., corneal ulcer) is greater for extended wear. In rare cases, loss of vision may result. Side effects like discomfort, mild burning or stinging may occur.

References: 1. Alcon data on file, 2017. **2.** Alcon data on file, 2018. **3.** Marx S, Sickenberger W. Wettability of different silicone hydrogel lens materials and blister solutions measured using non-invasive keratographic drying up time (NIK-DUT). *Optom Vis Sci.* 2016;93:E-abstract 165113. **4.** Tucker R, Lemp J, Guillon M. In vitro and on eye wettability of lotrafilcon B lenses packaged with a substantive wetting agent. *Invest Ophthalmol Vis Sci.* 2017;58: ARVO E-Abstract 3070. **5.** Lemp J, Muya L, Driver-Scott A, Alvord L. A comparison of two methods for assessing wetting substantivity. Poster presented at: 2016 Global Specialty Lens Symposium (GSLS); January 21-24, 2016; Las Vegas, NV. **6.** Alcon data on file, 2017. **7.** Lemp J, Zhao X, Perry S. Retention of packing solution comfort agents. Poster presented at: 2017 American Academy of Optometry Annual Meeting; October 11-14, 2017; Chicago, IL. **8.** Eiden SB, Davis R, Bergenske P. Prospective study of lotrafilcon B lenses comparing 2 versus 4 weeks of wear for objective and subjective measures of health, comfort, and vision. *Eye Contact Lens.* 2013;39(4):290-294. **9.** Lemp J, Kern J. A comparison of real time and recall comfort assessments. *Optom Vis Sci.* 2016;93:E-abstract 165256.

See product instructions for complete wear, care and safety information. Rx only

Alcon

COVID Vaccines and Corneal Graft Rejection

A recent paper reveals that clinicians and patients should be aware of the potential of corneal graft rejection associated with COVID-19 vaccine administration and consider vaccination in advance of planned non-urgent keratoplasties. The researchers believe that their study is the first report of temporal association between corneal transplant rejection following immunization against COVID-19 and the first report of DMEK rejection following any immunization. They hypothesize that the allogeneic response may have been initiated by the host antibody response following vaccination.

In one case, DMEK was performed around the time of vaccination, but it had taken place years earlier in the other. In both cases, one unilateral and the other bilateral, the transplant rejection was treated successfully with topical corticosteroids.

In the first case, a 66-year-old woman underwent an uneventful DMEK in her right eye. Her history was notable for HIV infection that was well controlled (undetectable viral load). Two weeks after DMEK, she received the first dose of Pfizer's COVID vaccine. She presented a week later with acute-onset blurred vision, redness and photophobia in her right eye. Clinical examination found indications typical of acute endothelial graft rejection. The frequency of topical steroid (dexamethasone 0.1%) was increased from four times daily to every hour. Signs and symptoms began to resolve after three days, and by four weeks after the rejection onset, visual acuity was good and there was no active inflammation.

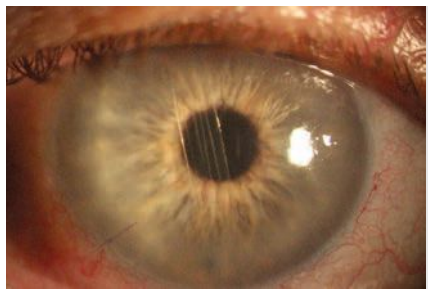

Photo: Mitch Ibach, OD, Scott Hauswirth, OD

Failed graft patients were successfully treated with topical corticosteroids.

In the second case, an 83-year-old woman had undergone DMEK in her right eye six years earlier and in her left eye three years earlier. She presented with symptoms of rejection two months after receiving her first COVID vaccine dose and three weeks after the second dose. Bilateral, simultaneous acute endothelial graft rejection was diagnosed, and she was put on hourly steroid drops. Seven days later, signs of inflammation were reduced and both grafts were functioning well, at which time the frequency of topical dexamethasone was reduced.[1]

In an email to Reuters Health, the authors speculated that, "The patient's antibody response triggered by vaccination caused immunological injury to the internal (endothelial) surface of the transplanted donor cornea."[2]

Nevertheless, their report states, "Patients with corneal transplants and their clinicians should not be deterred from COVID-19 vaccination."

1. Phylactou M, Li JPO, Larkin DFP. Characteristics of endothelial corneal transplant rejection following immunisation with SARS-CoV-2 messenger RNA vaccine. Br J Ophthalmol. April 28, 2021. [Epub ahead of print].

2. Baltic S. SARS-CoV-2 mRNA vaccine might trigger corneal-transplant rejection. Medscape. www.medscape.com/viewarticle/950985. May 13, 2021. Accessed May 26, 2021.

Gland Expression Improves Cataract Surgery Results

Upon investigating the effects of administering meibomian gland warming and expression prior to cataract surgery, researchers recently found that preoperative treatment may be a safe and effective intervention for relieving MGD and DED induced by surgery.

This prospective, randomized controlled study assessed 124 eyes scheduled for cataract surgery. Participants were randomly allocated to control and treatment groups, with the treatment cohort receiving therapy three weeks prior to surgery. The team evaluated meibomian gland atrophy, gland expressibility and gland secretion quality at baseline and one and three months postoperatively. They also took tear film break-up time (TBUT), Oxford corneal staining score and tear film lipid layer thickness (LLT) measurements at each visit, in addition to Ocular Surface Disease Index (OSDI) and Dry Eye Questionnaire (DEQ) scores.

The investigators reported a significant decrease in meibomian gland expressibility, meibum quality, LLT, corneal staining and dry eye symptoms in controls following cataract surgery. Conversely, the treatment group showed significantly improved meibomian gland patency, meibum quality, TBUT and corneal staining. This group also reported better subjective outcomes on both OSDI and DEQ.

The improvement of each parameter in the treatment group showed a linear correlation with baseline MGD grade. Patients without baseline MGD also showed improvement in DED induced by surgery from preoperative gland expression.

"[This treatment] might be recommended not only for the patients with preoperative MGD, but also for those without baseline MGD to prevent the development of MGD and dry eye induced by ocular surgeries," the study authors concluded in their paper.

Park J, Yoo YS, Shin K, et al. Effects of Lipiflow treatment prior to cataract surgery: a prospective, randomized, controlled study. Am J Ophthalmol. May 13, 2021. [Epub ahead of print].

IN THE BATTLEGROUND OF DRY EYE...

When Dry Eye Flares strike, fight back first with fast.

- EYSUVIS is ***THE FIRST AND ONLY FDA APPROVED SHORT TERM (up to two weeks) RX TREATMENT*** for the signs and symptoms of Dry Eye Disease
- EYSUVIS ***RAPIDLY REDUCED**** Dry Eye signs and symptoms in the largest clinical development program in Dry Eye (N=2871)[1]
- EYSUVIS ***TARGETS OCULAR SURFACE INFLAMMATION***, an underlying pathology of Dry Eye
- EYSUVIS is formulated with AMPPLIFY® Drug Delivery Technology, designed to ***ENHANCE OCULAR SURFACE TISSUE DISTRIBUTION AND PENETRATION***[2,3]
- EYSUVIS had a ***LOW INCIDENCE OF INTRAOCULAR PRESSURE ELEVATION*** (similar to vehicle) and was well-tolerated in clinical trials[4] —Please see Warning on Intraocular Pressure Increase below

*The safety and efficacy of EYSUVIS was assessed in 4 multicentered, randomized, double-masked, placebo-controlled trials in 2871 patients with documented Dry Eye. Patients received either EYSUVIS or vehicle 4 times a day for at least 2 weeks. Patients taking EYSUVIS showed significant reduction in the symptoms of Dry Eye (ocular discomfort) as early as Day 4 after starting treatment (versus vehicle). Symptoms continued to improve up to the end of the treatment period (Day 15). Patients taking EYSUVIS also showed significant reduction in signs of Dry Eye (conjunctival hyperemia) at Day 15 versus vehicle.

EYSUVIS®
(loteprednol etabonate ophthalmic suspension) 0.25%
THE FAST FLARE FIGHTER

INDICATION

EYSUVIS is a corticosteroid indicated for the short-term (up to two weeks) treatment of the signs and symptoms of dry eye disease.

IMPORTANT SAFETY INFORMATION

Contraindication:

EYSUVIS, 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.

Warnings and Precautions:

Delayed Healing and Corneal Perforation: Topical corticosteroids have been known to delay healing and cause corneal and scleral thinning. Use of topical corticosteroids in the presence of thin corneal or scleral tissue may lead to perforation. The initial prescription and each 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.

Intraocular Pressure (IOP) Increase: Prolonged use of corticosteroids may result in glaucoma with damage to the optic nerve, as well as defects in visual acuity and fields of vision. Corticosteroids should be used with caution in the presence of glaucoma. Renewal of the medication order should be made by a physician only after examination of the patient and evaluation of the IOP

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

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

Viral Infections: Use of a corticosteroid medication in the treatment of patients with a history of herpes simplex requires great caution. Use of ocular corticosteroids 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 corticosteroid application. Fungus invasion must be considered in any persistent corneal ulceration where a corticosteroid has been used or is in use.

Adverse Reactions:

The most common adverse drug reaction following the use of EYSUVIS for two weeks was instillation site pain, which was reported in 5% of patients.

Please see Brief Summary of Prescribing Information for EYSUVIS on the next page.

References: 1. Holland E, Nichols K, Foulks G, et al. Safety and efficacy of KPI-121 ophthalmic suspension 0.25% for dry eye disease in four randomized controlled trials. Presented at: AAO 2020: November 13-15, 2020; virtual meeting. **2.** Schopf L, Enlow E, Popov A, et al. Ocular pharmacokinetics of a novel loteprednol etabonate 0.4% ophthalmic formulation. *Ophthalmol Ther.* 2014;3(1-2):63-72. **3.** Popov A. Mucus-penetrating particles and the role of ocular mucus as a barrier to micro- and nanosuspensions. *J Ocul Pharmacol Ther.* 2020;36(6): 366-375. **4.** Korenfeld M, Nichols KK, Goldberg D, et al. Safety of KPI-121 ophthalmic suspension 0.25% in patients with dry eye disease: a pooled analysis of 4 multicenter, randomized, vehicle-controlled studies. *Cornea.* 2020. In press.

kala PHARMACEUTICALS

US-EYS-2100022 www.EYSUVIS.com

EYSUVIS (loteprednol etabonate ophthalmic suspension) 0.25%, for topical ophthalmic use

BRIEF SUMMARY OF FULL PRESCRIBING INFORMATION

INDICATIONS AND USAGE

EYSUVIS is a corticosteroid indicated for the short-term (up to two weeks) treatment of the signs and symptoms of dry eye disease.

CONTRAINDICATIONS

EYSUVIS, 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.

WARNINGS AND PRECAUTIONS

Delayed Healing and Corneal Perforation—Topical corticosteroids have been known to delay healing and cause corneal and scleral thinning. Use of topical corticosteroids in the presence of thin corneal or scleral tissue may lead to perforation. The initial prescription and each 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.

Intraocular Pressure (IOP) Increase—Prolonged use of corticosteroids may result in glaucoma with damage to the optic nerve, as well as defects in visual acuity and fields of vision. Corticosteroids should be used with caution in the presence of glaucoma. Renewal of the medication order should be made by a physician only after examination of the patient and evaluation of the IOP.

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

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

Viral Infections—Use of corticosteroid medication in the treatment of patients with a history of herpes simplex requires great caution. Use of ocular corticosteroids 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 corticosteroid application. Fungus invasion must be considered in any persistent corneal ulceration where a corticosteroid has been used or is in use. Fungal cultures should be taken when appropriate.

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

Contact Lens Wear—The preservative in EYSUVIS may be absorbed by soft contact lenses. Contact lenses should be removed prior to instillation of EYSUVIS and may be reinserted 15 minutes following administration.

ADVERSE REACTIONS

Adverse reactions associated with ophthalmic corticosteroids 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.

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.

The most common adverse reaction observed in clinical trials with EYSUVIS was instillation site pain, which was reported in 5% of patients.

USE IN SPECIFIC 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 1.4 times the recommended human ophthalmic dose (RHOD) and to pregnant rats at doses 34 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 3.4 times the RHOD. Maternal toxicity was observed in rats at doses 347 times the RHOD, and a maternal no observed adverse effect level (NOAEL) was established at 34 times the RHOD.

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 (1.4 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 (5.6 times the RHOD). At 3 mg/kg (41 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 (83 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 (34 times the RHOD); and cleft palate, agnathia, cardiovascular defects, umbilical hernia, decreased fetal body weight and decreased skeletal ossification at 50 mg/kg (347 times the RHOD). Embryofetal lethality (resorption) was observed at 100 mg/kg (695 times the RHOD). The NOAEL for developmental toxicity in rats was 0.5 mg/kg (3.4 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 (3.4 times the clinical dose), reduced survival was observed in live-born offspring. Doses ≥ 5 mg/kg (34 times the RHOD) caused umbilical hernia/incomplete gastrointestinal tract. Doses ≥ 50 mg/kg (347 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 EYSUVIS and any potential adverse effects on the breastfed infant from EYSUVIS.

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

Geriatric Use—No overall differences in safety and effectiveness have been observed between elderly and younger adult 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 thymidine kinase (tk) assay, in a chromosome aberration test in human lymphocytes, or *in vivo* in the single dose mouse micronucleus assay. Treatment of male and female rats with 25 mg/kg/day of loteprednol etabonate (174 times the RHOD based on body surface area, assuming 100% absorption) prior to and during mating caused pre-implantation loss and decreased the number of live fetuses/live births. The NOAEL for fertility in rats was 5 mg/kg/day (34 times the RHOD).

For a copy of the Full Prescribing Information, please visit www.EYSUVIS.com.

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Recent Myopia Demographics Examined

At the 2021 ARVO virtual meeting, presenters discussed new research on myopia prevalence. One found that girls were more prone to the condition in a study of kids vs. adults. In the latter group, men had a higher prevalence.[1] In another, researchers found that home confinement during the COVID-19 epidemic increased the burden in kids between the ages of six and eight, who are more sensitive to environmental changes than older patients.[2]

Gender Shift

Rising rates of myopia worldwide are prompting researchers and clinicians to find ways to understand its course and halt its progression. This study suggests gender may play a role in (or at least be correlated with) development of this condition, which was found to be more common in younger girls of the present generation of children, yet, in adults, had a greater prevalence among males.[1]

The research team from the Netherlands and Poland explored gender differences in myopia development based on two prospective, population-based cohorts from different generations. The first investigation, Generation R, enrolled roughly 7,000 participants and tracked them from birth until young adulthood. The second, the Rotterdam Study I-III, included about 9,000 participants who were older than 45.

Cycloplegic refraction was measured in the children at ages six, nine and 13, while automated refraction was evaluated in the adults and axial length and height were calculated in both groups. Myopia was defined as a spherical equivalent less than -0.5D in at least one eye.

Children responded to questionnaires about their lifestyle, including near work and outdoor exposure, and adults were additionally queried about their education levels. Change in height was also taken into account.

Photo: Breanne McGhee, OD

Gender may be correlated with trends in myopia development.

In children, the rate of myopia steadily increased from 2.5% to 11.5% and then nearly doubled to 22.5% at ages six, nine and 13, respectively. The prevalence was higher in adults at roughly 31%.

Female gender was linked to childhood myopia, but this finding was the opposite in adults. Whether a child developed myopia or not appeared to be influenced by several factors including outdoor exposure, height growth, sports participation, reading time and the number of books read per month.

Controlling for these factors collectively lessened the gender effect by roughly 35%. In adults, education was the most important mediator and lessened gender's influence by about 90%.

These findings provide compelling evidence that lifestyle factors and education are strong drivers of myopia and that girls in particular should be guided to adhere to behaviors that lessen their myopia risk, the researchers said.[1]

Younger Shift During COVID-19

Lockdowns that were enacted during the COVID pandemic have meant less time spent outdoors for millions of children, which raised concerns about whether home confinement worsened myopia burden. A school-based cross-sectional study in China investigated the refractive change and prevalence of myopia for school-aged kids.[2]

A total of 123,535 children ages six to 13 were screened between 2015 and 2020, which included a total of 194,904 tests from 389,808 eyes. Non-cycloplegic refraction and spherical equivalent refraction were recorded for each child and the prevalence of myopia for each age group in each year was calculated. The mean spherical equivalent and prevalence of myopia were compared between 2020 (after home confinement) and the previous five years for each age group.

A substantial myopic shift (around -0.3D) was found in the 2020 school-based screenings when compared with previous years for school-aged children at ages six (-0.32D), seven (-0.28D) and eight (-0.29D). The prevalence of myopia in the 2020 tests was much higher than the highest rates from 2015 to 2019 for children at ages six (21.5% vs. 5.7%), seven (26.2% vs. 16.2%) and eight (37.2% vs. 27.7%).

The differences in spherical equivalent and prevalence of myopia between 2020 and previous years were minimal in children ages nine to 13.

"Home confinement due to COVID-19 was associated with a significant myopic shift for children," the authors concluded in their presentation. "Younger children (ages six to eight) are more sensitive to environmental changes to develop myopia than older children, given that the younger children are in a critical period for the development of myopia."[2]

"This very interesting study looking at home confinement due to COVID-19 confirms many of our clinical impressions related to myopic progression in this vulnerable age group," Joseph Shovlin, OD, noted. "Home confinement likely is associated with more close work, fewer outdoor activities and ultimately seems to lead to more myopic progression."

1. Enthoven C, Haarman AEG, Swierkowska J, et al. Gender predisposition to myopia shifts to girls in the young generation. ARVO 2021 Annual Meeting.

2. Qian X, Li Y, Musch DC, et al. The critical period of myopia, insight from the myopic shift in school age children after COVID-19 home confinement. ARVO 2021 Annual Meeting.

Black Patients May Have Stiffer Scleras

Primary open-angle glaucoma (POAG) is more prevalent and more severe in Black populations compared with white subjects. The reason for this is unknown, but researchers believe differences in ocular biomechanics may play a role. Joshua Canavan, an OD candidate at Ohio State University, presented his group's findings on this topic in a poster session recently at the ARVO 2021 virtual meeting. The researchers investigated corneal and scleral stiffness responses in healthy eyes of sub-Saharan African, European and mixed-race individuals to gather baseline data and found scleral stiffness may contribute to POAG.

Subjects included in the study had no history of ocular disease. They were categorized by self-reported ancestry: sub-Saharan African (n=40 eyes, mean age 37, 65% female), European (n=84 eyes, mean age 35, 60% female) and mixed-race (n=36 eyes, mean age 32, 61% female). The researchers measured corneal hysteresis, central corneal thickness and biomechanically corrected intraocular pressure (IOP) to derive *in vivo* parameters of corneal and scleral (SP-HC) stiffness.

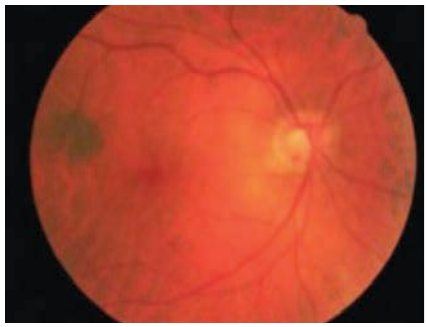


Photo: Rim Makhlouf, OD

Stiffer scleras might contribute to POAG's pathophysiology.

In the question-and-answer virtual chat, Mr. Canavan explained that scleral stiffness was derived from the Corvis ST (Oculus) and calculated as the "difference between corneal deflection amplitude at maximum capacity and at first applanation." He noted, "It also considers the load imposed on the cornea by the device. The sclera limits the maximum deformation of the cornea in response to the air puff, so SP-HC is a biomarker for scleral stiffness."

His team found no significant difference between the three ancestry cohorts in corneal hysteresis, corneal stiffness, Goldmann applanation tonometry, IOP, Pascal dynamic contour tonometry, or central corneal thickness. Mean scleral stiffness was as follows:

- 14.37mm Hg/mm in the sub-Saharan African cohort
- 13.86mm Hg/mm in the European cohort
- 14.72mm Hg/mm in the mixed-race cohort

The researchers noted that when adjusted for central corneal thickness and higher IOP in the mixed-race cohort, mean scleral stiffness was significantly higher among sub-Saharan African individuals than the other two cohorts.

They concluded that individuals of sub-Saharan African descent may have greater scleral stiffness independent of other ocular biomechanical parameters. They believe this may contribute to the pathophysiology of POAG. "The ramifications of this finding warrant further investigation," said Mr. Canavan.

Canavan J, Koons A, Mahmoud A, et al. Corneal and scleral biomechanical differences among individuals of Sub-Saharan African, European and mixed-race descent. ARVO 2021 Meeting.

10-2 Test a Better Predictor of Central Progression

Researchers recently compared the variability and ability to detect visual field (VF) progression of the 24-2, central 24-2 and 10-2 tests in eyes with abnormal VFs. The team determined that the time to detect central VF progression was reduced with 10-2 mean deviation (MD) due to the test's lower variability.[1]

"Identifying glaucoma progression perimetrically can be challenging due to variability in testing and patient responses," noted Joseph Sowka, an attending optometric physician at Center for Sight in Sarasota, FL, in his commentary on the study for *Practice Update*. "Additionally, the most used perimetric program in glaucoma, the 24-2 pattern, can miss small changes due to the degree of spacing of tested points."[2]

The study included a total of 52,806 24-2 and 11,966 10-2 VF tests from 7,307 eyes from the Glaucoma Research Network database. Only eyes with five or more visits and two or more years of follow-up were included.[1]

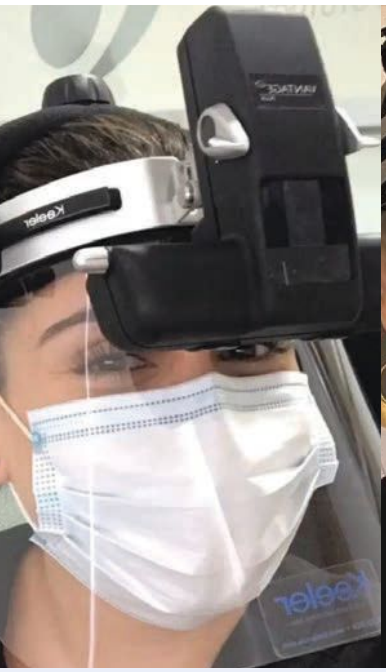
Upon evaluating all three patterns, MD variability was highest within the -5dB to -20dB range and consistently lower with the 10-2 compared with the 24-2 and central 24-2. Overall, time to detect confirmed significant progression at 80% power was the lowest with the 10-2 test, with a decrease of 14.6% to 18.5% when compared with the 24-2 and a decrease of 22.9% to 26.5% when compared with the central 24-2.[1]

The researchers believe these findings contribute to current evidence of the potential value of 10-2 testing in glaucoma patient management and in clinical trial design. Using 10-2 tests could result in a moderate increase in the ability to detect progression centrally, without compromising the clinician's assessment of non-central regions.[1]

1. Susanna FN, Melchior B, Paula JS, et al. Variability and power to detect progression of different visual field patterns. Ophthalmology. April 20, 2021. [Epub ahead of print].

2. Freeman KF, Sowka J. Progression detection with different visual field patterns. Practice Update. www.practiceupdate.com/c/117552/2/5. May 10, 2021. Accessed May 19, 2021.

Helping Heroes See Clear And Stay Safe

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ORS Resident Case Report Contest Winners

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

The two cases shown here, selected by the ORS Awards Committee, were co-winners of the fifth annual Larry Alexander Resident Case Report Contest. The contest is sponsored by Zeiss, Heidelberg and Optos.

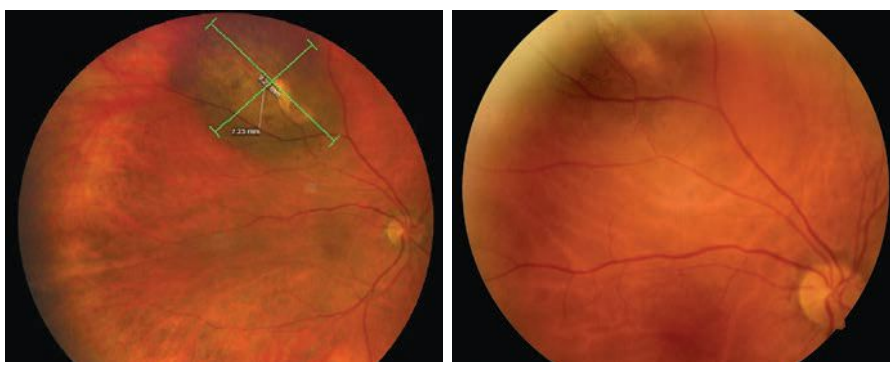
"When reviewing submissions, we look for manuscripts that are well written, include high-quality images and provide new and innovative information to the community," says Julie Rodman, OD, professor and chief of the Fort Lauderdale (Broward) Eye Care Institute at Nova Southeastern University in Florida, and ORS treasurer. "This year, our winning manuscripts focused on timely topics and provided insight into the diagnosis and management of entities that are often challenging to diagnose," she notes.

The full text and images of both case reports are available online at www.reviewofoptometry.com.

Case 1: Choroidal Melanoma

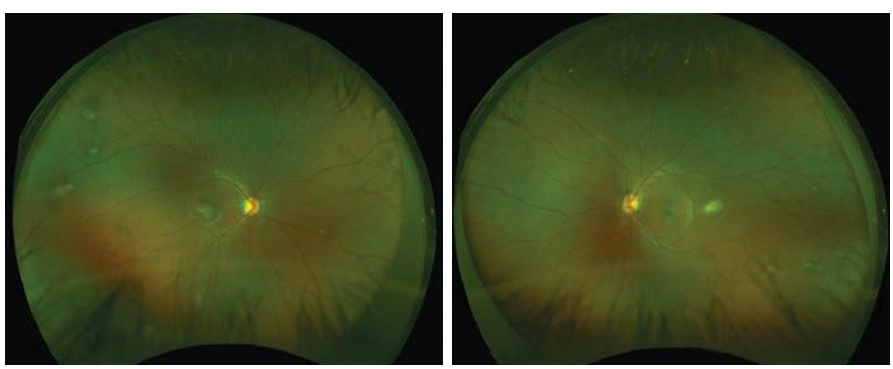
This case study, presented by Amy Bade, OD, a low vision and ocular disease resident at the Northeastern State University College of Optometry, reviewed characteristic risk factors for choroidal melanoma and the use of multimodal imaging to assist in the detection of these key features.

Photos: Amy Bade, OD



Case 1. Comparing images from 2020 (left) and 2018 (right) of the pigmented choroidal tumor of the right eye demonstrates obvious expansion of the choroidal melanoma.

Photos: Isis Topete, OD



Case 2. Fundus images of the right and left eyes reveal that CHRRPE lesions often appear elevated and pigmented (usually grey, white or brown, but may be orange, yellow or green).

The report detailed the management of a Native American woman diagnosed with a choroidal melanoma and discussed the findings and clinical utility of supplementary multimodal imaging. According to Dr. Bade, early and accurate detection of choroidal melanoma has greatly improved with the use of contemporary multimodal imaging technology including enhanced-depth imaging OCT, fundus autofluorescence and widefield fundus photography.

Contemporary management of choroidal melanoma most often includes plaque radiotherapy, and newer, innovative methods are promising. Although the condition is uncommon, Dr. Bade believes it is highly critical that eye care providers be familiar with and able to recognize high-risk features suggestive of small choroidal melanomas by using multimodal imaging.

Case 2: CHRRPE in a Pediatric Patient

Isis Topete, OD, a pediatric optometry resident at the Duke University Eye Center, presented a case of bilateral, combined hamartoma of the retina and retinal pigment epithelium (CHRRPE) leading to a suspected diagnosis of neurofibromatosis type 2 (NF2). CHRRPE are rare intraocular tumors characterized by the malformation of the neurosensory retina, retinal pigment epithelium and adjacent vitreous. NF2 is a multiple neoplasia syndrome that predisposes patients to the development of tumors in the nervous system, eyes and skin.

In her case report, Dr. Topete discussed the clinical characteristics of CHRRPE, as well as other potential ocular manifestations of neurofibromatosis type 2 and the importance of early diagnosis. She noted that it is important for eye care practitioners to recognize that children with NF2 more commonly present with ocular, dermatological and/or neurological signs and require careful ophthalmic examination.

Helping Parents Find Confidence in Myopia Management Using Clinical Data

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MYOPIA MANAGEMENT PROGRAM
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Clinical studies and data prove contact lenses are safe for children to use, but many parents are hesitant to say "yes" to a contact lens program at the start due to concerns about ocular side effects such as redness or infections, and the maturity of the child to handle the lenses appropriately. Here's how Dr. Roxanne Achong-Coan from Coan Eye Care & Optical Boutique uses clinical data in her myopia management conversations with parents.

Roxanne Achong-Coan, OD, FAAO, FIAOMC, FSLS, Coan Eye Care & Optical Boutique, Ocoee, Florida

When discussing contact lens options for myopia management with parents, what is your primary recommendation?

I always lead with CooperVision's Brilliant Futures™ Myopia Management Program with MiSight® 1 day because it is the only FDA-approved* soft contact lens to slow the progression of myopia in children, aged 8 – 12 years at the initiation of treatment. The supporting clinical data is also incredibly strong. The first three years of data conclusions showed the lens slowed myopia progression by 59% in refractive error and 52% in axial length on average.[1†] Over a 6-year period, one-in-four children wearing MiSight® 1 day did not change in prescription[2†‡]. Early identification of myopia risk and early intervention is key. I am confident in communicating these clinically proven outcomes to parents. For parents, they are confident because it's the only option that is FDA approved.

Which studies do you find are most informational for parents to help them overcome their hesitations regarding their child wearing contact lenses?

Choosing studies to share depends on the specific parent concern:

- Ocular side effects. I share a study that concludes the incident of corneal infiltrative events in children 8 – 12 years old is no higher than it is in adults, and in some cases is markedly lower.[3]
- Ocular health. I share a new study that followed children wearing MiSight® 1 day* daily disposable lenses over six years and found their ocular health was similar to when they started wearing lenses, proving there is a minimal impact on the ocular surface and that lenses are very safe.[4]
- Applying and removing contact lenses. I talk about the CLIP study that concludes it only takes younger children about 15 minutes longer to learn how to apply and remove their lenses.[5]
- Switching from glasses to contact lenses. I share the ACHIEVE study that found children and teens had significantly improved satisfaction with their vision correction, and contact lenses also improved how they felt about themselves, their appearance, self-esteem and ability to perform activities.[6]

Does sharing clinical data help parents in deciding to pursue myopia management for their child?

Sharing data is a great way to show parents that these medical devices have been studied intently over time and having so many studies with positive outcomes proves that contact lenses are safe for their young child, so that is helpful in the decision-making process. However, discussing clinical data is only one part of the conversation. Parents ultimately decide to pursue myopia management because we educate them on what myopia is, why and when it progresses and how it can affect their child's eye health long-term. Once a parent learns that myopia progresses quickly at a young age, and we use data to show what could happen to their vision and ocular health long-term, then parents truly understand the importance of managing myopia early rather than waiting until their child is older.

What tips do you have for ECPs who are just starting to have conversations with parents about myopia management and want to cite clinical data?

Know the studies you want to share thoroughly. In conversation, you should simply summarize studies and mention a few of the best results because hearing all the data could overwhelm a parent. Most parents won't press further into the data, but if a parent does, you'll want to know your numbers and details of the study. Your confidence translates into a parents' confidence. Parents look for your clinical expertise to prescribe the best option for their child. It's also a best practice to provide trifold brochures or a summary sheet that parents can take home to review with their spouse.

*** Indications for use:** MiSight® 1 day (omafilcon A) soft (hydrophilic) contact lenses for daily wear are indicated for the correction of myopic ametropia and for slowing the progression of myopia in children with non-diseased eyes, who at the initiation of treatment are 8–12 years of age and have a refraction of -0.75 to -4.00 diopters (spherical equivalent) with ≤ 0.75 diopters of astigmatism. The lens is to be discarded after each removal.

† Compared to a single vision 1 day lens over a 3 year period.

‡ No clinically meaningful change in refractive error -0.25D or less from baseline.

References:

[1] Chamberlain P, et al. A 3-year randomized clinical trial of MiSight® lenses for myopia control. *Optom Vis Sci.* 2019; 96(8):556-567. Compared to a single vision 1 day lens over a 3 year period.

[2] Chamberlain P, Arumugam B, Jones D et al. Myopia Progression in Children wearing Dual-Focus Contact Lenses: 6-year findings. *Optom Vis Sci.* 2020;97(E-abstract): 200038.

[3] Bullimore, M. The Safety of Soft Contact Lenses in Children. *Optom Vis Sci.* 2017 Jun; 94(6):638-646.

[4] Woods, J., et al. Ocular Health of Children Wearing Daily Disposable Contact Lenses Over a 6-Year Period. *Contact Lens Anterior Eye.* 2021.

[5] Walline JJ, Jones LA, Rah MJ, et al. Contact lenses in pediatrics (CLIP) study: chair time and ocular health. *Optom Vis Sci.* 2007;84: 896-902.

[6] Walline JJ, et al. The Adolescent and Child Health Initiative to Encourage Vision Empowerment (ACHIEVE) study design and baseline data. *Optom Vis Sci.* 2006 Jan;83(1):37-45.

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Slows Myopia Progression
on average[1†]

52%
Axial Length Elongation Reduction
on average[1†]

Child Friendly[1]
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*__Indications for use:__ MiSight® 1 day (omafilcon A) soft (hydrophilic) contact lenses for daily wear are indicated for the correction of myopic ametropia and for slowing the progression of myopia in children with non-diseased eyes, who at the initiation of treatment are 8-12 years of age and have a refraction of -0.75 to -4.00 diopters(spherical equivalent) with ≤ 0.75 diopters of astigmatism. The lens is to be discarded after each removal.

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[1]Chamberlain P, et al. A 3-year randomized clinical trial of MiSight® lenses for myopia control. *Optom Vis Sci.* 2019; 96(8):556-567.

FEATURES

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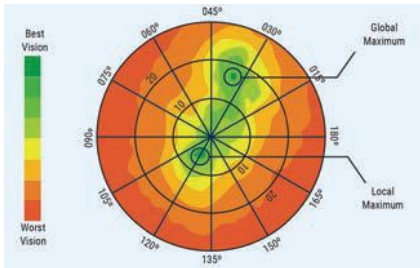

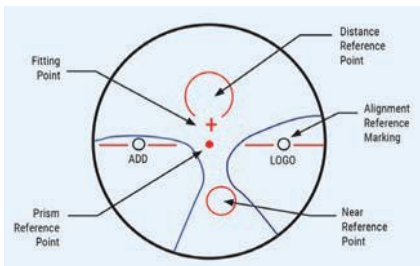

We Welcome Your Comments

Feedback from the community provides important insights about clinical practice. If you would like to share your thoughts on the topics discussed in this issue—or the wider field of optometry at large—please write to:

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INDICATION

Upneeq® (oxymetazoline hydrochloride ophthalmic solution), 0.1% is indicated for the treatment of acquired blepharoptosis in adults.

IMPORTANT SAFETY INFORMATION

WARNINGS AND PRECAUTIONS

- Alpha-adrenergic agonists as a class may impact blood pressure. Advise Upneeq patients with cardiovascular disease, orthostatic hypotension, and/or uncontrolled hypertension or hypotension to seek medical care if their condition worsens.
- Use Upneeq with caution in patients with cerebral or coronary insufficiency or Sjögren's syndrome. Advise patients to seek medical care if signs and symptoms of potentiation of vascular insufficiency develop.
- Upneeq may increase the risk of angle closure glaucoma in patients with untreated narrow-angle glaucoma. Advise patients to seek immediate medical care if signs and symptoms of acute narrow-angle glaucoma develop.
- Patients should not touch the tip of the single patient-use container to their eye or to any surface, in order to avoid eye injury or contamination of the solution.

ADVERSE REACTIONS

Adverse reactions that occurred in 1-5% of subjects treated with Upneeq were punctate keratitis, conjunctival hyperemia, dry eye, blurred vision, instillation site pain, eye irritation, and headache.

DRUG INTERACTIONS

- Alpha-adrenergic agonists, as a class, may impact blood pressure. Caution in using drugs such as beta blockers, anti-hypertensives, and/or cardiac glycosides is advised. Caution should also be exercised in patients receiving alpha adrenergic receptor antagonists such as in the treatment of cardiovascular disease, or benign prostatic hypertrophy.
- Caution is advised in patients taking monoamine oxidase inhibitors which can affect the metabolism and uptake of circulating amines.

To report SUSPECTED ADVERSE REACTIONS or product complaints, contact RVL Pharmaceuticals at 1-877-482-3788. You may also report SUSPECTED ADVERSE REACTIONS to the FDA at 1-800-FDA-1088 or www.fda.gov/medwatch.

Please see next page for Brief Summary of full Prescribing Information.

Reference: 1. Upneeq® (oxymetazoline hydrochloride ophthalmic solution), 0.1%. [Prescribing Information].

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BRIEF SUMMARY: The following is a brief summary only; see full Prescribing Information at https://www.upneeq.com/Upneeq-PI.pdf for complete information.

1 INDICATIONS AND USAGE

UPNEEQ is indicated for the treatment of acquired blepharoptosis in adults.

2 DOSAGE AND ADMINISTRATION

Contact lenses should be removed prior to instillation of UPNEEQ and may be reinserted 15 minutes following its administration.

If more than one topical ophthalmic drug is being used, the drugs should be administered at least 15 minutes between applications.

4 CONTRAINDICATIONS

None.

5 WARNINGS AND PRECAUTIONS

5.1 Potential Impacts on Cardiovascular Disease

Alpha-adrenergic agonists may impact blood pressure. UPNEEQ should be used with caution in patients with severe or unstable cardiovascular disease, orthostatic hypotension, and uncontrolled hypertension or hypotension. Advise patients with cardiovascular disease, orthostatic hypotension, and/or uncontrolled hypertension/hypotension to seek immediate medical care if their condition worsens.

5.2 Potentiation of Vascular Insufficiency

UPNEEQ should be used with caution in patients with cerebral or coronary insufficiency, or Sjögren's syndrome. Advise patients to seek immediate medical care if signs and symptoms of potentiation of vascular insufficiency develop.

5.3 Risk of Angle Closure Glaucoma

UPNEEQ may increase the risk of angle closure glaucoma in patients with untreated narrow-angle glaucoma. Advise patients to seek immediate medical care if signs and symptoms of acute angle closure glaucoma develop.

5.4 Risk of Contamination

Patients should not touch the tip of the single patient-use container to their eye or to any surface, in order to avoid eye injury or contamination of the solution.

6 ADVERSE REACTIONS

6.1 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.

A total of 360 subjects with acquired blepharoptosis were treated with UPNEEQ once daily in each eye for at least 6 weeks in three controlled Phase 3 clinical trials, including 203 subjects treated with UPNEEQ for 6 weeks and 157 subjects treated with UPNEEQ for 12 weeks. Adverse reactions that occurred in 1-5% of subjects treated with UPNEEQ were punctate keratitis, conjunctival hyperemia, dry eye, blurred vision, instillation site pain, eye irritation, and headache.

7 DRUG INTERACTIONS

7.1 Anti-hypertensives/Cardiac Glycosides

Alpha-adrenergic agonists, as a class, may impact blood pressure. Caution in using drugs such as beta-blockers, anti-hypertensives, and/or cardiac glycosides is advised.

Caution should also be exercised in patients receiving alpha adrenergic receptor antagonists such as in the treatment of cardiovascular disease, or benign prostatic hypertrophy.

7.2 Monoamine Oxidase Inhibitors

Caution is advised in patients taking MAO inhibitors which can affect the metabolism and uptake of circulating amines.

8 USE IN SPECIFIC POPULATIONS

8.1 Pregnancy

Risk Summary

There are no available data on UPNEEQ use in pregnant women to inform a drug-associated risk for major birth defects and miscarriage. In animal reproduction studies, there were no adverse developmental effects observed after oral administration of oxymetazoline hydrochloride in pregnant rats and rabbits at systemic exposures up to 7 and 278 times the maximum recommended human ophthalmic dose (MRHOD), respectively, based on dose comparison. [see Data]. The estimated background risks of major birth defects and miscarriage for the indicated population are unknown. All pregnancies have a background risk of birth defect, loss, or other adverse outcomes. In the U.S. general population, the estimated background risk of major birth defects and miscarriage in clinically recognized pregnancies is 2-4% and 15-20%, respectively.

Data

Animal Data

Effects on embryo-fetal development were evaluated in rats and rabbits following oral administration of oxymetazoline hydrochloride during the period of organogenesis. Oxymetazoline hydrochloride did not cause adverse effects to the fetus at oral doses up to 0.2 mg/kg/day in pregnant rats during the period of organogenesis (28 times the MRHOD, on a dose comparison basis). Oxymetazoline hydrochloride did not cause adverse effects to the fetus at oral doses up to 1 mg/kg/day in pregnant rabbits during the period of organogenesis (278 times the MRHOD, on a dose comparison basis). Maternal toxicity, including decreased maternal body weight, was produced at the high dose of 1 mg/kg/day in pregnant rabbits and was associated with findings of delayed skeletal ossification.

In a rat prenatal and postnatal development study, oxymetazoline hydrochloride was orally administered to pregnant rats once daily from gestation day 6 through lactation day 20. Maternal toxicity was produced at the high dose of 0.2 mg/kg/day (28 times the MRHOD, on a dose comparison basis) in pregnant rats and was associated with an increase in pup mortality and reduced pup body weights. Delayed sexual maturation was noted at 0.1 mg/kg/day (14 times the MRHOD, on a dose comparison basis). Oxymetazoline hydrochloride did not have any adverse effects on fetal development at a dose of 0.05 mg/kg/day (7 times the MRHOD, on a dose comparison basis).

8.2 Lactation

Risk Summary

No clinical data are available to assess the effects of oxymetazoline on the quantity or rate of breast milk production, or to establish the level of oxymetazoline present in human breast milk post-dose. Oxymetazoline was detected in the milk of lactating rats. The developmental and health benefits of breastfeeding should be considered along with the mother's clinical need for UPNEEQ and any potential adverse effects on the breastfed child from UPNEEQ.

8.4 Pediatric Use

Safety and effectiveness of UPNEEQ have not been established in pediatric patients under 13 years of age.

8.5 Geriatric Use

Three hundred and fifteen subjects aged 65 years and older received treatment with UPNEEQ (n = 216) or vehicle (n = 99) in clinical trials. No overall differences in safety or effectiveness were observed between subjects 65 years of age and older and younger subjects.

10 OVERDOSAGE

Accidental oral ingestion of topical intended solutions (including ophthalmic solutions and nasal sprays) containing imidazoline derivatives (e.g., oxymetazoline) in children has resulted in serious adverse events requiring hospitalization, including nausea, vomiting, lethargy, tachycardia, decreased respiration, bradycardia, hypotension, hypertension, sedation, somnolence, mydriasis, stupor, hypothermia, drooling, and coma. Keep UPNEEQ out of reach of children.

PATIENT COUNSELING INFORMATION

Advise the patient to read the FDA-approved patient labeling (Instructions for Use).

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BY JACK PERSICO
EDITOR-IN-CHIEF
OUTLOOK

Profit and Loss

Who wins and who loses as optometrists choose employment over entrepreneurship?

Optometry is one of the few remaining professions with a still-vibrant culture of single-doctor private practices, giving its practitioners a chance to live out the classic "hang a shingle" mode of making a living. Or at least it used to be a thriving part of optometry. More ODs are turning away from private practice ownership and opting instead to choose a paycheck over a P&L statement.

A recent study dissecting the profession's workforce has much to say about the state of optometry now and in the near future. Age, gender and ethnicity characteristics, patient volume, hours worked, income levels and a host of other factors all get rigorous attention. There's much about the study, undertaken by the AOA, worth digging into; you can read our full summary of it online in our news feed now and next month in the print edition.

One of the most interesting statistics was the report's finding that the ranks of employed optometrists jumped 15% (from 29% to 44%) in five years, from 2012 to 2017. This period, mind you, is just before a wave of private equity acquisitions gobbled up optometry practices. Surely, the number of employed ODs today is even higher.

Is this a good or a bad thing? I doubt there's a simple answer, as there are so many different stakeholders in the delivery of optometric care: ODs of course, their patients and employees, medical equipment and pharma companies, health insurers and lots more.

Some bemoan the decline of solo practice, out of a romantic attachment to the idea. But with a few exceptions, it's hard to see overwhelming losses from the trend toward employment.

The AOA's workforce study found that employed and self-employed optometrists worked about the same number of hours and were equally productive and satisfied in their roles. Interestingly, the employed ODs saw more patients per week than those who were self-employed (58 vs. 54) "This is despite the number of hours worked per week being similar," the study points out, likely given the added hassles of practice administration that fall to a self-employed doctor.

One setback for employed ODs is income, as those who earned the most tended to be self-employed. Another seems to be fewer self-employment opportunities for women, as only 43% of female ODs in the study practiced that way, compared with 66% of their male counterparts. The report didn't address underlying reasons for the distinction, but one popular explanation is that the added burden of childcare that women disproportionately shoulder makes self-employment a harder proposition for them. (Does that ring true? Please write to us at editor@reviewofoptometry.com with your thoughts!)

Who's doing the employing? That's not delineated either, but anecdotally I encounter more and more ODs who hail from within ophthalmology's private practices and teaching institutions among the doctors who write for *Review*. This is obviously not a representative sample, but perhaps it is a bellwether. New grads enter the field with abundant clinical skill in medical eye care and eagerness to use it. If ophthalmologists are seeing the value of adding an OD or two to their offices, it validates the strength of optometry's institutions—and its very calling. ■

BY PAUL M. KARPECKI, OD
CHIEF CLINICAL EDITOR

Rescuing the Retina

Innovations are expanding optometry's role in care as specialists find themselves overburdened and demand piles up.

This month I talked all about the retina with one of the best, John Kitchens, MD, a previous chief surgical fellow at Bascom Palmer who practices at Retina Associates of Kentucky. Here are some highlights of our talk.

Central Serous Retinopathy

Probably the biggest current breakthrough is in imaging, specifically enhanced-depth visualization using spectral domain or swept-source OCT. These technologies have helped us understand the critical role of the choroid, which is important because the pathogenesis of central serous retinopathy (CSR) is often related to a thickened choroid (i.e., pachychoroid). While there have been reports of systemic therapies for CSR such as mineralocorticoid receptor antagonists, aldosterone antagonists and even melatonin, the mainstay of treatment is observation for early occurrences and laser or PDT therapy for chronic cases or cases where rapid visual improvement is necessary (e.g., pilots).

In the Pipeline

The retinal disease drug pipeline is also exciting. First, we should have faricimab (Genentech) approved in early 2022. This is a bispecific antibody, meaning it binds two different targets: VEGF and ANG-2. The latter is felt to play a role in inflammation and vascular destabilization. Phase III studies for wet AMD and DME showed this drug could be used in a treat and extend approach with dosing extending to three months in over 70% of patients.

The port delivery system (PDS, Genentech) should also be approved in 2022. This is tremendously exciting as it is the first sustained-release drug delivery system for anti-VEGF.

> **The retina is one of the great opportunities for our profession, with exciting developments improving our ability to help patients with sight-threatening pathologies.**

Patients in the trial benefited from six-month dosing and sustainability of treatment effect. Although the Phase III trials involved six-month refills, the Phase II LADDER study (more open-ended, with as-needed refills) showed the average refill time to be over a year (median 15 months).

Exciting Developments

There will be sustained efforts to identify patients with diabetic eye disease earlier, as current therapies show improvements in retinopathy and a decrease in vision-threatening complications such as PDR and DME. The NEI's 10-year follow-on study of AREDS2 reaffirms that we can significantly slow progression of intermediate to advanced dry AMD with AREDS2 formulations like Pre-serVision and many others. Patients with a family history of AMD, low carotenoid levels or earlier stages of AMD may be best served with a carotenoid supplement such as MacuHealth or Ocuvite. MacuHealth's new micromicellar formulation showed greater bioavailability with a six times higher serum response and 1.5x retinal response over a six-month supplementation period.[2]

The introduction of a head-mounted dark adaptometer (AdaptDx Pro, MacuLogix) is helping us improve our ability to accurately diagnose and manage AMD patients. This functional test can be administered anywhere there is a comfortable chair and with minimal technician time because the system's voice guidance system is consistently administering adaptive feedback and instructions to the patient. This device allows us to make dark adaptation testing our standard of care for every at-risk patient age 50 and older.

Monitoring carotenoid levels in the serum, which have been shown to correlate with macular pigment, can be easily performed with a biophotonic hand scanner in less than 30 seconds. Now more than ever, patients understand the importance of overall health; carotenoid levels have been shown to not only affect AMD but improve health and cognitive ability in Alzheimer's patients.[1] Lastly, at-home monitoring (Notal Vision) can help optometry identify wet AMD earlier and improve patients' prognosis.

The retina is one of the great opportunities for our profession, with exciting developments improving our ability to help patients with sight-threatening pathologies. ■

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2. Green-Gomez M, Prado-Cabrero A, Moran R, et al. The impact of formulation on lutein, ze-axanthin, and meso-zeaxanthin bioavailability: A Randomised Double-Blind Placebo-Controlled Study. Antioxidants (Basel). 2020 Aug 18;9(8):767.

About Dr. Karpecki **Dr. Karpecki** is medical director for Keplr Vision and the Dry Eye Institutes of Kentucky and Indiana. He is the Chief Clinical Editor for *Review of Optometry* and chair of the New Technologies & Treatments conferences. A fixture in optometric clinical education, he consults for a wide array of ophthalmic clients, including ones discussed in this article. Dr. Karpecki's full disclosure list can be found in the online version of this article at www.reviewofoptometry.com.

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BY MONTGOMERY VICKERS, OD

CHAIRSIDE

The New New

More and more people are getting vaccinated—you know what that means.

So, the country is slowly but surely evolving from the hide-in-your-attic, the-sky-is-falling, why-should-I-pay-for-a-year's-supply-of-contact-lenses-when-we-are-all-going-to-die mentality.

Have you had your vaccine? I have. Has this changed my day-to-day existence? Yes; I no longer have an excuse not to dog- and babysit every weekend. (Hey, prior to the vaccine, I sat around doing nothing out of love for my family. I am just naturally a giving person.)

In the office, we still wear masks. Our patients still wear masks. Guess we'll have to start brushing our teeth again soon, but that can wait a little longer.

My wife and I have gained the courage to get back to the gym. I for one was surprised that I had not declined in my ability to bench press 12 pounds and then spend the next hour shooting the breeze with the lifeguards while my wife actually exercised.

One thing that didn't change was eating out. I did get more particular about ordering foods I can detect sneeze remnants on, though. Also, before COVID, I would eat anything. Now, I eat everything but sea urchin. Getting picky.

In large part, optometry was not permanently destroyed—just a little bruised. We're recovering and now able to ponder the many things that can help us in the next pandemic, which will probably come out of Congress from what I can tell.

First, the fallout from COVID did not improve anyone's eyesight. Years ago, in a smoky room in Philadelphia, a few of us barely-passing optometry students came up with something people would have to use every day that would screw up their eyes. This is how the idea of the sandpaper contact lens came to be. Since we were only dreaming, someone suggested we call a college dropout and get him to use all his newly-acquired free time to create this monster. To protect the innocent, I'll just call him "Gill Bates." Unfortunately, Gill moved on to his other project, which eventually still screwed up everyone's eyes, before this one could take off. All's well that ends well, I guess.

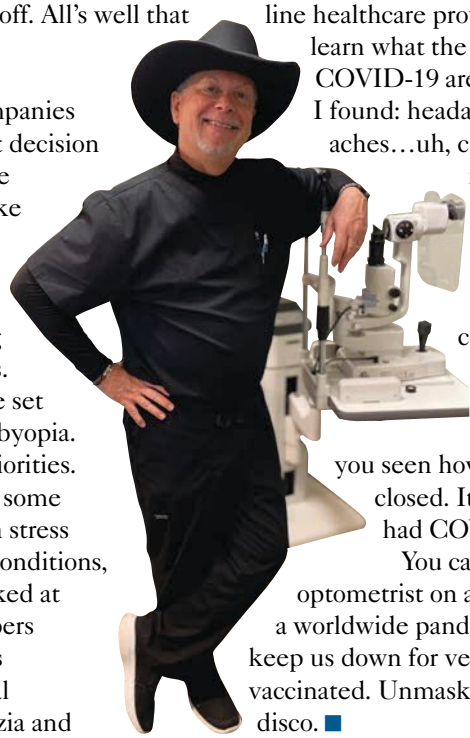
I also recall when pharmaceutical companies made the important decision to quit trying to cure heart disease to make room for the more important research on eye drops to treat itchy eyes. Big feather in their caps. Now their sights are set on eliminating presbyopia. Cancer can wait. Priorities.

I knew there was some correlation between stress and various ocular conditions, but I am truly shocked at the increased numbers of posterior vitreous detachments, retinal detachments, chalazia and formerly very happy glasses wearers who are now so sick of mask-induced foggy glasses that they want contact lenses for the first time. Too bad they are all 53 years old, their prescription is plano -3.25x137 with a +2.50 add and they just took a new job working on computers all day. Darn that Gill Bates! It would be much easier if they all just stuck with the retinal detachments.

And my staffers? God bless 'em. They are all pregnant. Well, not Bill. Not yet.

Now that I am vaccinated, I do sense that my patients, overall, are less scared to come in and are quicker to pull their masks off their noses when the phoropter fogs up. I guess they think that my vaccination protects them. I'm sorry, did I miss something?

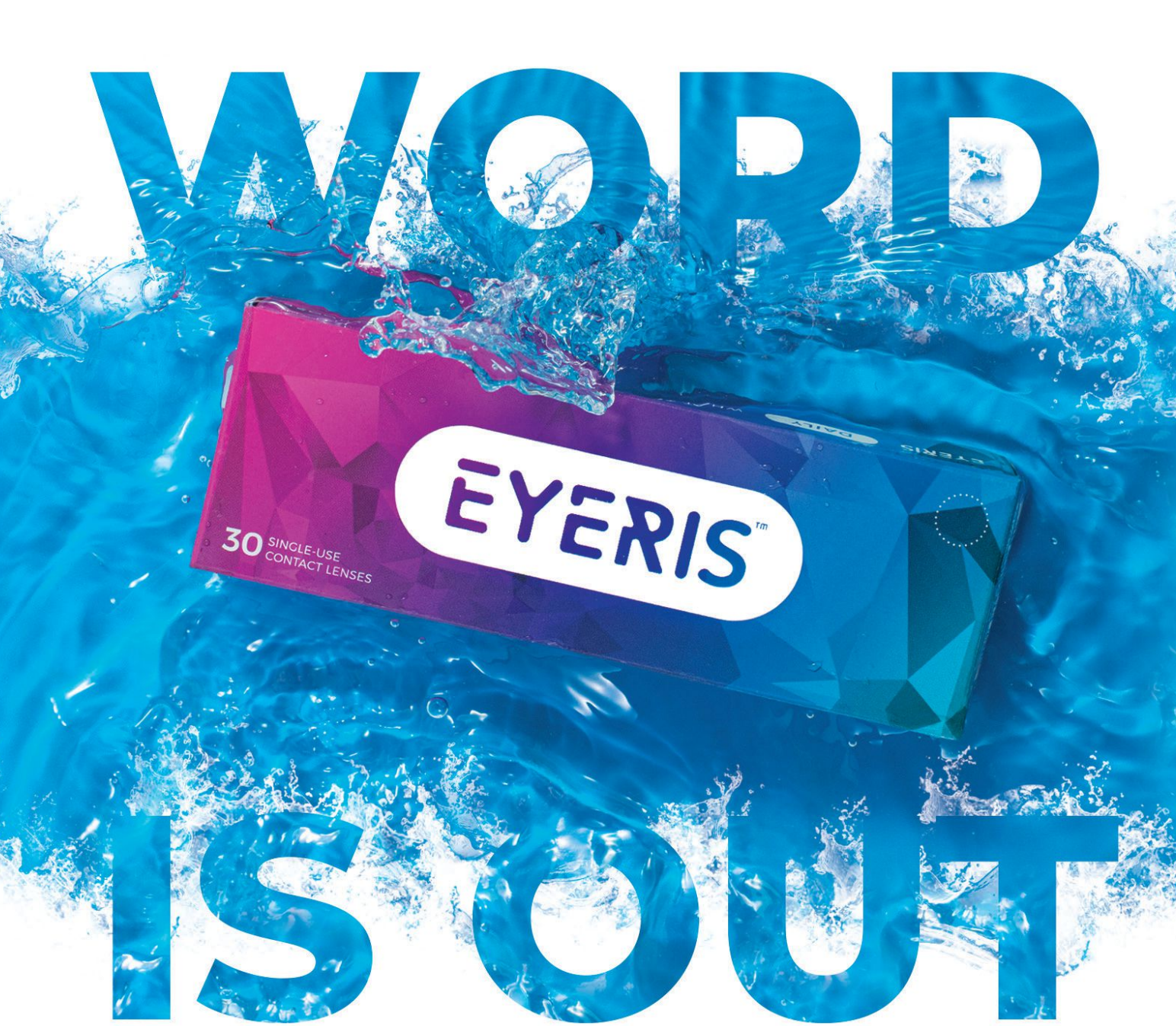
On my end, I decided, now that the pandemic has been going on for more than a year, maybe, as a first-line healthcare provider, I should learn what the symptoms of COVID-19 are. Here's what I found: headaches, body aches…uh, check! Did I lose my sense of smell? Well, it was certainly damaged during my all-male college experience (self defense). And my sense of taste? Have you seen how I dress? Case closed. It turns out I have had COVID since 1992.



You cannot defeat an optometrist on a mission. Even a worldwide pandemic cannot keep us down for very long. Get vaccinated. Unmask. See you at the disco. ■

About Dr. Vickers **Dr. Vickers** received his optometry degree from the Pennsylvania College of Optometry in 1979 and was clinical director at Vision Associates in St. Albans, WV, for 36 years. He is now in private practice in Dallas, where he continues to practice full-scope optometry. He has no financial interests to disclose.

WORD



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Burn, Baby, Burn

It is essential to determine the nature of a chemical injury when faced with this ocular emergency.

Q A patient presented with a history of accidentally getting eucalyptus oil in both eyes, with subsequent corneal abrasions. What is a good protocol for handling chemical burns?

A Chemical burns to the eye are an emergency all optometrists should feel comfortable treating and managing, says Trennda Rittenbach, OD, staff doctor at the Palo Alto Medical Foundation. It is critical to train your front office and phone staff to get these patients in immediately when they call. The first and most important step is copious irrigation of the eye with any brand of sterile irrigating solution, something you should stock in every exam lane. "Evert the upper and lower lids to irrigate all parts of the eye and adnexa, as chemical could remain trapped in the lower cul-de-sac or under the upper lid," she advises. Use litmus paper five to 10 minutes after irrigation in order to determine the pH. It is imperative to continue irrigation until the pH is neutralized.

Dr. Rittenbach cautions that a careful corneal exam, along with the conjunctiva and anterior chamber, is critical in order to not miss any collateral damage. "I commonly see oil splash burns from cooking that result in first- and sometimes second-degree burns to the eyelids, so also carefully examine the adnexa," she advises. Pay attention to intraocular pressure (IOP) readings, as alkaline chemical burns may cause immediate or delayed rises. Alkaline chemicals can penetrate the eye more easily due to their lipophilic nature, therefore can also damage the trabecular meshwork, ciliary body and possibly the lens.[1]

Investigate the Agent

This particular patient was rubbing eucalyptus oil onto his forehead for religious reasons, and it ended up in both eyes. This resulted in severe pain from bilateral epithelial defects centrally extending to the periphery OU. "After doing some quick research, I was able to find that an alcohol-based agent is sometimes used in non-pure forms of essential oils," Dr. Rittenbach notes.

Acidic chemicals that commonly cause ocular injuries are battery acid, acetic acid such as nail polish remover or vinegar, toilet bowl cleaners and some swimming pool agents. A very common injury optometrists see is from hydrogen peroxide–based contact lens solutions. Some common alkaline substances that can cause serious ocular injuries are oven cleaner, drain cleaner, chlorine bleach, lime found in plaster, cement and mortar, and ammonia products usually found in cleaning products and fertilizer. Get a detailed history and ask the patient to bring in the product they got into their eye(s).

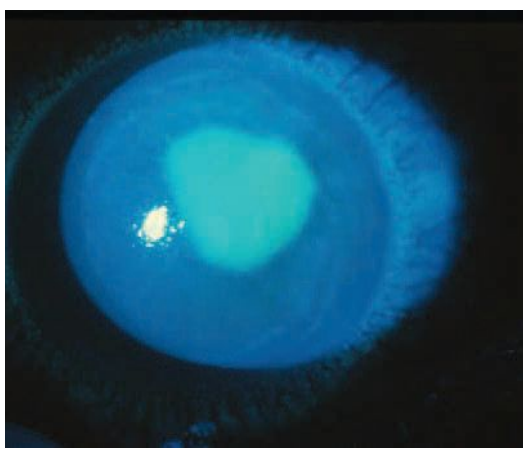


Photo: Brian Den Beste, OD

Chemical burns can create painful epithelial defects.

Treatment will depend on clinical signs, and ranges from frequent lubrication and topical steroids to amniotic membranes. Autologous serum tears may be necessary if there has been a loss of limbal stem cells. If the IOP is elevated, be sure to manage that as well.

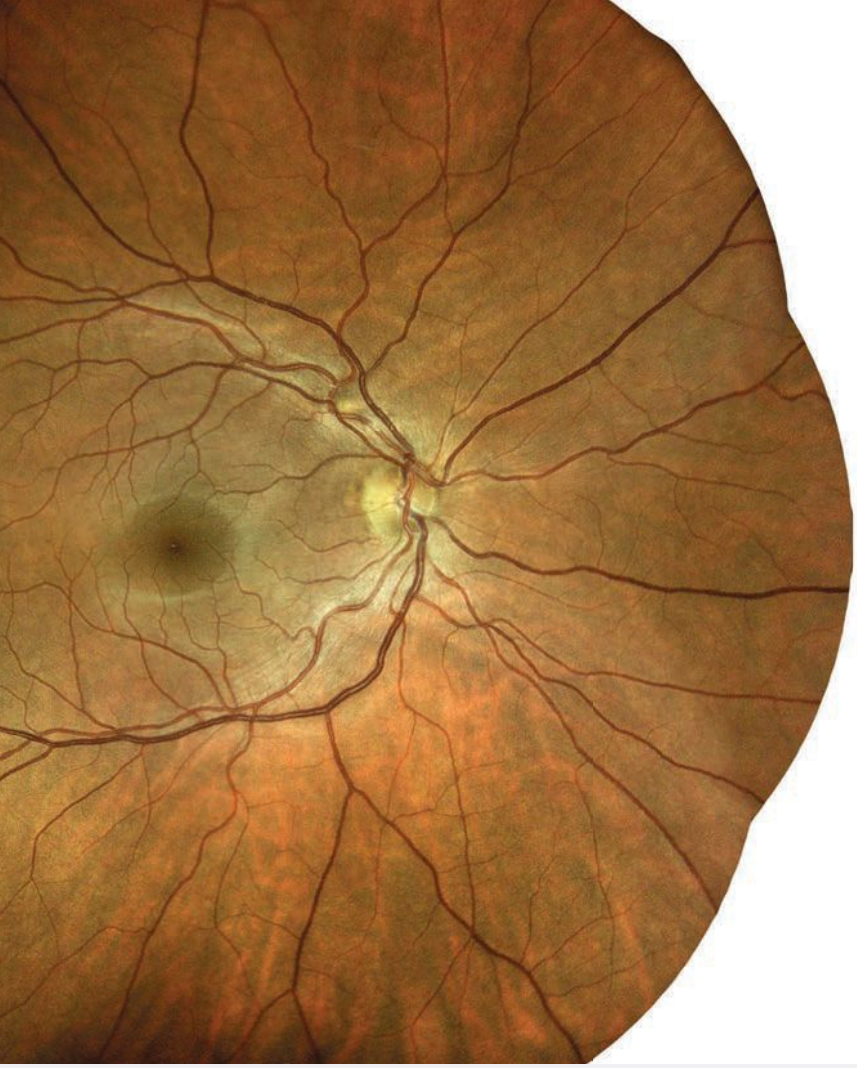
The patient did well with erythromycin ointment every four hours and Refresh Celluvisc (carboxymethylcellulose sodium 1% gel, Allergan) six to eight times per day, according to Dr. Rittenbach. The next day, slit lamp exam showed immense improvement with only a small central area of epithelial loss. By day six, the epithelium was completely healed.

It is vital to assess the presence and degree of limbal ischemia as well as any opacification of the cornea. An injected conjunctiva puts one a little more at ease. Blanching can indicate ischemia, usually associated with an alkali burn, that can lead to limbal stem cell loss and tissue necrosis. These patients may have a poor outcome with corneal scarring, loss of vision and life-long dry eye and may possibly need surgical intervention such as a corneal transplant.

A white, blanched eye or a cornea that is not healing quickly warrants a referral to an anterior segment or cornea specialist. ■

1. Baradaran-Rafii A, Eslani M, Haq Z, et al. Current and upcoming therapies for ocular surface chemical injuries. Ocul Surf. 2017;15(1):48-64.

About Dr. Ajamian **Dr. Ajamian** is the center director of Omni Eye Services of Atlanta. He currently serves as general chairman of the education committee for SECO International. He has no financial interests to disclose.



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BY MARC B. TAUB, OD, MS, AND PAUL HARRIS, OD

Space: the Final Frontier

Where appropriate, work cheiroscopic tracing into your workup to get a better handle of the case in your chair.

The June 2017 installment of this column, "Putting Pen to Paper," included a cheiroscopic tracing to show size perception differences in how a patient with aniseikonia saw the world. Cheiroscopic tracings are not a routine part of optometric practice, but they come in handy as a problem-solver in some of the more difficult cases. In this month's column, we will reacquaint you with the procedure and, more importantly, help you understand which types of cases it is best suited for.

The Process

A typical example of a stereoscope can be seen in *Figure 1*. The optics can vary, but most have +5.00 sphere lenses, which set the working distance at 20.00cm, simulating distance demand for accommodation. Additionally, they have base-out prism in front of each eye, allowing the two pictures to be separated in space, even though they appear to be overlapping to the viewer with little vergence demand. In one form or another, each also has a septum, which prevents the right eye from seeing the left target and the left eye from seeing the right target.

We now have the two channels isolated, but the sense is that they are unified. *Figure 2* shows a patient completing a tracing. The standard paper used is 11.50in wide by 4.25in tall with the reference figure printed in the center (*Figure 3a*).

When performing the test, first the printed target is placed before one eye in the stereoscope. Then, the patient is given a pencil and asked to trace the figure. When the figure is in front of the left eye, the right hand performs the tracing, and vice versa. This gives us two ways to see the variations between the spatial percepts as formed through each eye. This helps with size differences (aniseikonia), cyclodeviations (where one eye relative to the other sees an altered scene) and vertical deviations (where one tracing is above the reference picture and the other is below).

Fig. 1. The Wolff Standup Cheiroscope (Bernell).

There is a misconception that aniseikonia is nearly always present when different refractive powers exist between the right and left eyes. However, this is not always the case. We have seen cases with large differences in refraction between the right and left eyes with no size perception differences and cases where it is present.

> **There is a misconception that aniseikonia is nearly always present when different refractive powers exist between the right and left eyes. However, this is not always the case.**

We have also seen our fair share of cases with nearly equal refractive powers on each side, in which a clinically significant size perception difference existed. Without testing for it, we would be searching in the dark for something that is rather easy to identify and if present, is rather easy to prescribe for. As the late Robert A. Kraskin, OD, used to say, "It's not what a lens does to a person, but what a person does with a lens" that really matters.

More Than Meets the Eye

Figure 3b shows a size perception difference between a pair of eyes of about 10%. When the reference figure is in front of the right eye, the left hand traces a larger figure than is referenced. While tracing, the point of the pencil looks like it follows the outline of the reference figure, and when done, the two figures look as if they are one solid figure—both

About Drs. Taub and Harris

Dr. Taub is a professor, chief of the Vision Therapy and Rehabilitation service and co-supervisor of the Vision Therapy and Pediatrics residency at Southern College of Optometry (SCO) in Memphis. He specializes in vision therapy, pediatrics and brain injury. **Dr. Harris** is also a professor at SCO. Previously, he was in private practice in Baltimore for 30 years. His interests are in behavioral vision care, vision therapy, pediatrics, brain injury and electrodiagnostics. They have no financial interests to disclose.

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appearing to be the same size. The spatial transformation is the result of optics plus perception and is not secondary to only the optics.

When the reference figure is in front of the left eye, the right hand traces a smaller figure by the same amount on the right side of the paper, which, again, appears to be the same size as the target. It is important to have both representations to compare the size differences between the two figures. The more similar the size transformations are, the higher the likelihood those measures are clinically significant.

Figure 3c shows a similar set of tracings where no size difference is present but the perception of what is vertical and horizontal is clearly off with respect to how the world is viewed through each eye. In this instance, we can see 10° of outward cyclorotation. We always label the direction of the rotation relative to the top and whether it is shifted inwards toward the nose or outwards toward the ear.

The Case

An older male patient was referred due to sudden-onset diplopia. In a person aged 65+, this is always a red flag. His history included acute sinus infections, which required medical treatment but no surgery.

Fig. 2. A modified Brewster Stereoscope (Keystone). This version is affixed to the wall and can be adjusted to meet the needs of most people.

Cover testing in primary gaze at distance revealed a 10 prism-diopter exophoria with a 2 prism-diopter vertical deviation. At near, the amount of exo increased, but the patient did not show a tropia until we did some eye movements.

When looking in any direction 20° to 25° away from straight ahead, he saw double vision with a horizontal and vertical component. However, he also mentioned that every time his vision doubled, one image was twisted, or, as he said, "vertical wasn't vertical in one of the images."

We performed a double Maddox rod test and measured 10° of cyclodeviation in primary gaze. This same cyclo was seen on a cheiroscopic tracing, similar to *Figure 3c*.

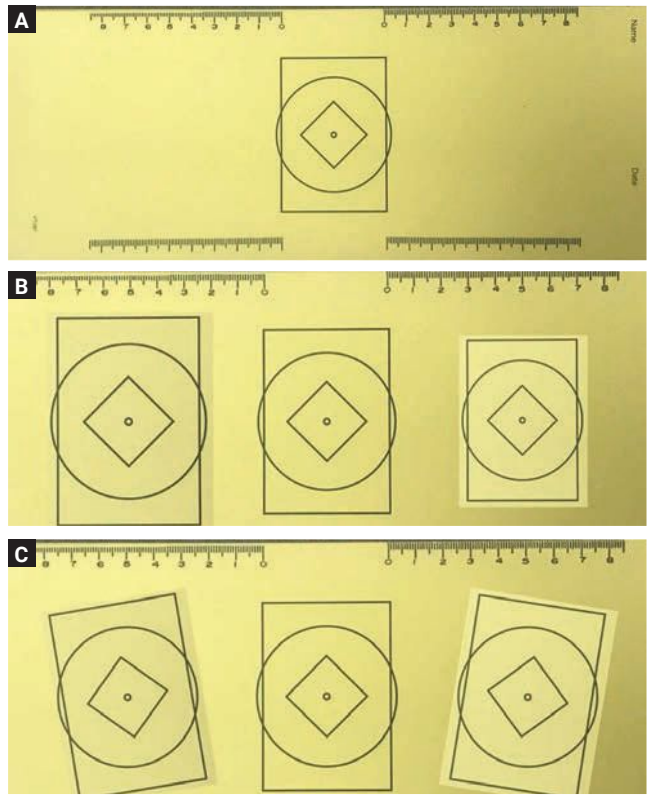

Fig. 3. On top (A) is the blank form used to perform the tracings. The middle image (B) shows a roughly 10% size difference. Note that each side must show a similar amount of size difference, but in the opposite direction. On the bottom (C) is a clinically significant cyclorotation.

Because the patient was not seeing double in primary gaze and showed normal global stereo acuity and no other signs of eye, vision or systemic health issues, we decided to take no further action and simply see him four to six weeks later. We expected him to continue recovering and explained our hypothesis that the episode of double vision was secondary to his sinus issues, which may have interfered with innervation to the extraocular motor system.

At the follow-up visit, the cyclo was fully resolved, though the patient still showed a mild exophoria at both distance and near. The slight vertical deviation had also resolved. He was able to move his eyes out to 50° in all directions at distance and near without diplopia interfering or any break in fusion occurring. A repeat of the cheiroscopic tracing showed equal-sized images and no evidence of the prior cyclorotation.

Imaging was not ordered because it seemed like he was already well along on the road to recovery by the time we saw him.

Takeaways

There are times when our standard chair and analytic tests don't give us the whole picture. Adding some insights from our optometric heritage, which in this case included cheiroscopic tracing and the double Maddox rod for confirmation, helped us understand how best to approach the care of our patient. Having these adjunct tests available when needed can save chair time as well as reduce patient anxiety, which might be triggered by additional testing. ■

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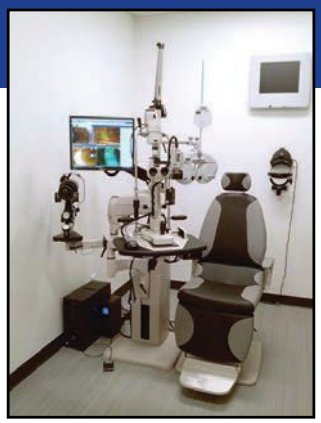

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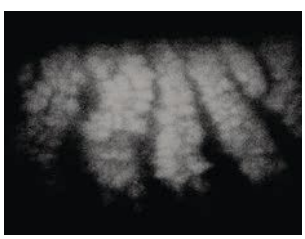

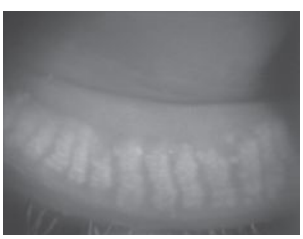

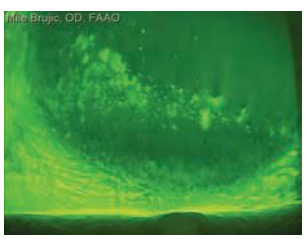

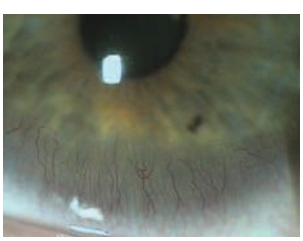

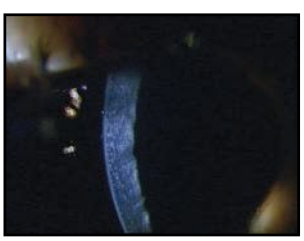

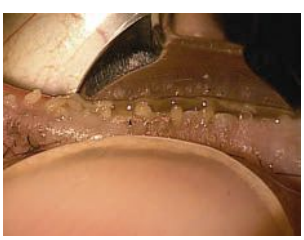

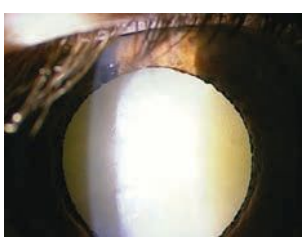

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BY JOHN RUMPAKIS, OD, MBA
CLINICAL CODING EDITOR

Bring the Retina into View

The often forgotten is now in focus.

The retina has traditionally been the domain of ophthalmology and not a typical area for optometrists. Over the past decade, that has changed with the advent of technologies that have since improved in clinical application. In January 2021, the redefinition of the CPT language associated with E&M services dictated the practitioner only perform "a medically appropriate history and examination." There is now no requirement to dilate to meet the definition of a 992XX code, and because of that, it may change how you decide to employ various technologies into your clinical regimen. Let's explore some of these technologies and their code requirements.

Fundus Photography (92250)

If medical necessity for bilateral use is not established in the record, it should be performed unilaterally and coded (92250-52-RT/LT). Photo documentation requires that the need for the image be determined on the day of service and after you examined the patient. It generally is not ordered in advance of the examination. Like the five following technologies, it requires an interpretation and report that demonstrates how it added to the care and management of the patient rather than just act as a confirmatory test.

Fundus autofluorescence is also coded as 92250 and follows the same rules for use and medical necessity. Watch out for performing 92250 on the same day of service as any of these other common ophthalmic codes: 92201, 92202, 99211, 92227, 92228, 92229, 92133, 92134, 92235, 92240.

OCT (92133 for Optic Nerve, -34 for Retina)

Described as a "unilateral or bilateral" procedure, OCT is coded and paid the same whether it is performed on one eye or both. 92133 and 92134 cannot under any circumstances be performed on the same date of service according to the CCI edit rules. OCT and OCT angiography are coded in the same manner and follow the same guidelines.

> **Medical necessity is required when performing the test based on clinical findings.**

In order to use OCT to follow a Plaquenil (hydroxychloroquine, Sanofi) patient, you must be using spectral domain–level equipment or better. Watch out for performing 92133 or 92134 on the same day of service as any of these other common ophthalmic codes: 76513 99211, 92227, 92228, 92229, 92250.

Dark Adaptation (92284)

Using this technology for early detection of macular degeneration has become more user-friendly. Like fundus photography, it is also a true bilateral test requiring that it be performed bilaterally. Like all special testing, medical necessity is required when performing the test based upon clinical findings.

In general, 92284 can be performed on the same day of service as most other ophthalmic tests but has a CCI edit rule that prevents it from being performed on the same day as 99211.

Extended Ophthalmoscopy (92201, -02)

Described as a "unilateral or bilateral" procedure, this is coded and paid the same whether it is performed on one eye or both. Redefined in January 2020, these codes now reflect examination of specific areas of the retina with specific examination techniques. Watch out for a Correct Coding Initiative (CCI) edit preventing you from performing either code on the same day as fundus photography.

Visual Fields (92081, -82, -83)

Described as a "unilateral or bilateral" procedure, visual fields are coded and paid the same whether performed on one eye or both. Make sure that the level of visual fields performed matches the level of medical necessity established in the medical record. In general, visual fields can be performed on the same day of service as most other ophthalmic tests but have a CCI edit rule preventing them from being performed on the same day as 99211.

Electroretinogram (92273, -74)

In January 2019, 92275 was supplanted by these two CPT codes specifying specific examination techniques. These tests are true bilateral tests. Electroretinography can be performed on the same day of service as most other ophthalmic tests but has a CCI edit rule that prevents it from being performed on the same day as 99211.

The retina is fertile territory for optometric specialization. Using technology to boost your clinical acumen and knowing the coding rules surrounding this technology will benefit your patients and boost your practice. ■

Send your coding questions to rocodingconnection@gmail.com.

About Dr. Rumpakis

Dr. Rumpakis is president and CEO of Practice Resource Management, a firm that provides consulting, appraisal and management services for healthcare professionals and industry partners. As a full-time consultant, he provides services to a wide array of ophthalmic clients. Dr. Rumpakis's full disclosure list can be found in the online version of this article at www.reviewofoptometry.com.

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BY BARRY SANTINI, ABOM,[1] VAN Y. RUE[2] AND BRENT McCARDLE, LDO[3]
[1]SEAFORD, NY, [2]RENTON, WA, [3]RALEIGH, NC

Eye care professionals (ECPs) probably spend more time trying to make their progressive wearers satisfied than any other category of eyeglass wearer. But despite advances in lens design and manufacture over the last 25 years, ECPs still yearn to discover the perfect progressive—the one design that balances acuity, comfort and utility just right. And while balancing these elements is essential, frame fit, cosmetics and perceived value also remain important ingredients in a successful experience.

To help both sides of the dispensing desk arrive at the best solution, let's start with a review of the fundamentals for dispensing progressive addition lenses (PALs). While challenging, these conditions can be comanaged successfully, and optometrists are in the perfect position to take a leadership role in the care of this sizable patient population.

The Juggling Act

One of human vision's biggest advantages—that it's inherently "squishy," or adaptable—does hinder choosing the best lens for any individual PAL wearer. The eye/brain's ability to adapt and function well in a matrix of less-than-optimal conditions, such as eyewear routinely made off-spec or with poor lens materials and centering, can hide what matters most for any individual patient. This squishiness also helps in defining the larger area of wearer satisfaction colloquially called "20/happy," which can guide in determining the most efficient use of professional time and materials.

But even 20/happy can be elusive, and therefore ECPs should always remain open to novel information as they juggle prescription, progressive design and placement to deliver a superlative progressive experience.

Prescription

Perhaps nothing impacts a prescriber's approach more than time spent performing eyeglass rechecks. By the time a wearer lands in the doctor's chair for a recheck, usually the Rx is the top suspect behind their eyewear dissatisfaction. Although creating the best progressive begins with an accurate refraction, it sometimes goes awry when the well-intentioned prescriber's discretion influences the final Rx in a way that works against progressive success. Below is a short list of suggestions that can improve progressive satisfaction:

1. Don't massage the astigmatism. Full correction of astigmatism delivers the best bang for the buck when chasing optimal acuity. But prescribing the full astigmatism can come with a price: an alteration of habitual perspective that negatively impacts comfort for some wearers. For this reason, prescribers often reduce or eliminate minor amounts of astigmatism up to 0.50D.

Additionally, orthogonalizing the axis—adjusting it closer to the prime meridians of 90º or 180º—is often used to further reduce perspective side effects. But all progressive lenses possess some residual astigmatism distributed across the lens surface.[1] This residual astigmatism can interact with any leftover refractive astigmatic error in a progressive lens and decrease acuity and utility.

About the authors **Mr. Santini** is a New York state licensed and ABOM-certified master optician and contact lens fitter at Long Island Opticians. **Mr. Rue** is an optical consultant and educator and progressive lens expert in the Seattle area. **Mr. McCardle** is a technical education specialist in North Carolina for Carl Zeiss Vision NA. Mr. Santini and Mr. Rue have no financial interests to disclose.

Recommendation: Prescribe the full cylinder found and avoid orthogonalizing the axis. Although satisfaction here is not guaranteed, the prospects for progressive success almost always improve when the eye's astigmatism is properly and optimally corrected. If the wearer experiences perceptual discomfort tracked to their astigmatism correction, try switching to a softer-design progressive.

2. Consider maximum plus and acuity. Approach this timeless recommendation with caution. With the standard exam distance set at 20 feet, which equals a vergence of +0.16D, the final subjective lens choices could easily flip into an undesirable over-plus situation for driving distance. Further, anything that compromises axial acuity will further degrade peripheral acuity by compounding the inherent peripheral blur of progressive lenses.

Recommendation: Trial frame the Rx outdoors to discover the optimal flip point for achieving best acuity while driving. There are exceptions—see *"Troubleshooting Mature Presbyopes"* on p. 39.

3. Be aware of pupil sampling. Pupil size controls more than luminous flux and near vision depth of field: the eye's pupil size determines the effective cross section of the eye's refractive optical train—cornea, crystalline lens and retina—that is contributing to the final refractive result. Sphere, cylinder power and axis can vary in value between a daytime pupil of 2.5mm diameter and a nighttime pupil of 5.5mm or greater diameter.

Recommendation: Again, trial frame the Rx outside the exam room, particularly at night if possible. The best correction for night use might be different enough to recommend two different prescriptions (*Figure 1*).

Design

Early progressives, such as the original Varilux (now Essilor) of 1959, came with substantial unwanted astigmatism in the transition zones near the progressive umbilic. As PALs evolved in the 1970s and 1980s, front-surface designs tried to reduce this in two ways: one approach—exemplified by the multi-add design of Varilux Infinity—favored adjusting corridor length to optimize the astigmatism gradient for increasing add powers, thereby optimizing corridor width and reducing swim-related effects.[2]

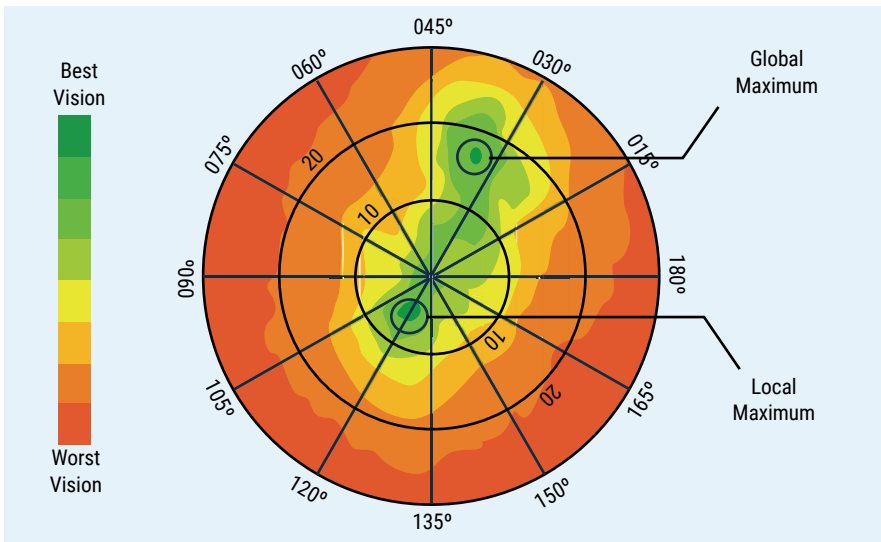
The other, exemplified by American Optical's Omni, dispensed with retaining areas of stable power almost altogether, and spread surface astigmatism throughout the entire lens through a bipolar design.[3] Nether design ultimately garnered sustained success for two reasons: Infinity required uncomfortable eye rotation in higher add powers, while unhappy Omni wearers found the bipolar design possessed insufficient areas of clear, stabilized prescription power.

But Omni did become the choice of wearers sensitive to swim effects—and provided insight on how residual astigmatism and magnification effects might be better managed. Eventually, progressive design evolved from just optimizing optics to full consideration of binocular needs. Zeiss's horizontal symmetry—which debuted in the Gradal HS lens—was among the first progressive lenses to address the importance of binocularity.[4] Gradal HS has since been discontinued.

Today, outcome-based visual satisfaction has become the target goal of PAL design. To this end, optimizing dynamic prism and dynamic magnification—the differing prismatic and magnifying effects encountered between the eyes as they gaze across the lens—becomes particularly important in any anisometropic and/or oblique cylinder axis prescription.

Optimization of these prescriptions can be achieved by using steeper or dissimilar base curves and differential lens thickness to help improve fusion and reduce swim. But the resulting cosmetics of these lenses are often less well-received by both practitioner and patient. However, wearers of lenses optimized in this way realize improved binocular function, with many reporting they are enjoying progressive comfort for the very first time. Shaw progressives are an example of this specialty approach, using an advanced, global iseikonic analysis to achieve binocular optimization.[5]

Today, the latest design process analyzes a wearer's head and neck posture over time, combined with comprehensive tracking data that reveals how we actually aim our eyes and tilt our heads in everyday tasks. This data is then used to optimize corridor length, width and specific distribution of residual astigmatism



Adapted from: Zeiss

Fig. 1. Pupil size determines the effective cross section of the eye's refractive optical train—cornea, crystalline lens and retina—that is contributing to the final refractive result.

MATCHING PROGRESSIVE DESIGN TO THE WEARER

Your assortment of 10 to 20 odd-sized and multi-colored contact lens trial sets says a lot more about your curated wisdom than you may realize. You carefully choose a contact lens because each fulfills a unique Rx or lifestyle need among your patient base. Every modality, base curve and diameter earns its place in your lens toolbox because it enables you to better serve patients or solve a problem. Progressive lenses are no different.

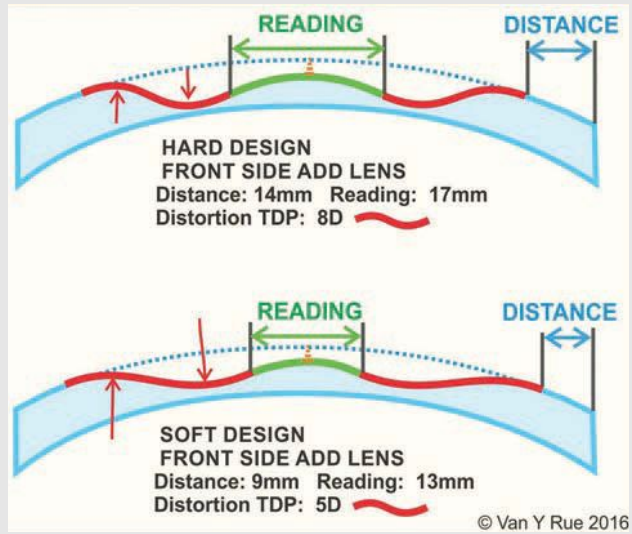
Older, cast-front progressives have an inescapable physics problem because about half of the surface area was rendered useless by an inability to optimally manage the greater surface astigmatism of these designs. The areas near the intermediate corridor and peripheral distance zones have been politely termed "soft-focus areas." To maintain a stable umbilic, traditional progressives defaulted to using a profile shaped like an hourglass. But this profile is problematic, as surface astigmatism of these older designs narrows the visual areas adjacent to the pupil while placing the widest and clearest areas at the very top and bottom of the lens.[1]

It's widely agreed that free-form progressive technology has delivered wider usable zones and reduced but never fully eliminated unwanted surface astigmatism. Not well discussed is that free-form technology gives designers enormous control over where that remaining distortion is placed, and as a consequence, what zones will benefit the most. Today, free-form lens designers can literally move distortion anywhere on the lens, thereby optimizing the zones they choose. As a result, free-form progressives—just like contact lenses—have become a vastly more specialized family of lenses and far more fit-specific in their application.[1]

During the early 2000s, progressive lens technology exploded. The "T-shape" design—only possible in free-form—appeared first in the Rodenstock Multigressiv (Free Form Technologies), followed by the Shamir Autograph. A T-shape could offer almost edge-to-edge distance clarity at the pupil height compared to the average 5mm to 12mm of usable distance width offered by traditional molded progressive designs of the time. Astute ECPs immediately found that patient complaints about driving at night utterly plummeted. As a trade-off, T-shape designs often suffer in intermediate width. And, in many cases, the reading zones of T-shape designs were found to be narrower than popular traditional lenses of the time, such as Varilux Comfort. Even free-form technology has limitations.

At the same time, Johnson & Johnson's Definity (now an Essilor lens) greatly improved on the original Owen Aves 1907 design, with its bi-surface design allowing advanced surface astigmatism management to push distortion away from areas nearest the pupil out to the upper and lower corners. The first advantage was increased intermediate width near the center—just below the pupil—not at the top or bottom lens edge. As a general purpose lens design, Definity became a computer user's dream. The second advantage was that the feeling of swim and sway, especially while walking, was greatly reduced, in part because distortion was pushed more outside the foveal cone. But a disadvantage was that usable distance width was sometimes perceived to be narrower than other traditional lenses.[1]

Matching a lens's inherent strengths to a patient's unique needs, lifestyle and Rx can tremendously improve patient satisfaction and reduce non-adapts. Most free-form lenses today are distance, intermediate or reading prioritized, and knowing which designs best match a wearer's needs is critical to improving satisfaction and creating viral patient referrals.



"Hard" and "soft" are the two main categories of progressive lens design. Both refer to the amount of blur located at the peripheral blending zones. Soft lenses spread the blending zones out into the distance and reading portions of the lens.

in creating the best balance between acuity, comfort and utility. A sampling of today's premium progressives that employ this comprehensive approach is found here:

- Hoya ID Mystyle 2 and ID Lifestyle 3
- Independent Owners Network (ION) Love Our Lens
- IOT Camber Steady and Camber Mobile
- Shamir Autograph Intelligence
- Varilux X and Comfort Max
- Zeiss Individual Smart Life and DriveSafe

There are further personalization frontiers to consider, such as the differences in axial length and center of rotation found between hyperopes and myopes. Finally, there's the dark arts of trying to match the best lens for any individual wearer (see "*Matching Progressive Design to the Wearer*").

Placement

PAL designers expect their lenses will be fit according to a stated protocol, where the fitting cross of the lens is placed over the center of the pupil with the facial plane vertical—meaning perpendicular to the floor (*Figure 2*). This is also the proper head alignment for measuring the pantoscopic tilt of the frame chosen.

Here are three interrelated factors to always keep in mind when placing a progressive:

1. Posture. A patient's habitual head posture often must be taken into account when specifying a fitting height. Habitual head posture reflects the routine manner in which an individual carries their head, *i.e.*, facial plane tilted forward or back—with tilt-back posing more significance

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

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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.

PM-US-CQA-0327 10/2019

SUN OPHTHALMICS

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

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

INDICATIONS AND USAGE

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

CONTRAINDICATIONS

None.

WARNINGS AND PRECAUTIONS

Potential for Eye Injury and Contamination

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

Use with Contact Lenses

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

ADVERSE REACTIONS

Clinical Trials Experience

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

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

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

USE IN SPECIFIC POPULATIONS

Pregnancy

Risk Summary

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

Data

Animal Data

Oral administration of cyclosporine oral solution (USP) to pregnant rats or rabbits was teratogenic at maternally toxic doses of 30 mg/kg/day in rats and 100 mg/kg/day in rabbits, as indicated by increased pre- and postnatal mortality, reduced fetal weight, and skeletal retardations. These doses (normalized to body weight) were approximately 3200 and 21,000 times higher than the maximum recommended human ophthalmic dose (MRHOD) of 1.5 mcg/kg/day, respectively. No adverse embryofetal effects were observed in rats or rabbits receiving cyclosporine during organogenesis at oral doses up to 17 mg/kg/day or 30 mg/kg/day, respectively (approximately 1800 and 6400 times higher than the MRHOD, respectively). An oral dose of 45 mg/kg/day cyclosporine (approximately 4800 times higher than MRHOD) administered to rats from Day 15 of pregnancy until Day 21 postpartum produced maternal toxicity and an increase in postnatal mortality in offspring. No adverse effects in dams or offspring were observed at oral doses up to 15 mg/kg/day (approximately 1600 times greater than the MRHOD).

Lactation

Risk Summary

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

Pediatric Use

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

Geriatric Use

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

PATIENT COUNSELING INFORMATION

Handling the Vial

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

Use with Contact Lenses

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

Administration

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

Rx Only

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Cranbury, NJ 08512

PLR-00020 2018

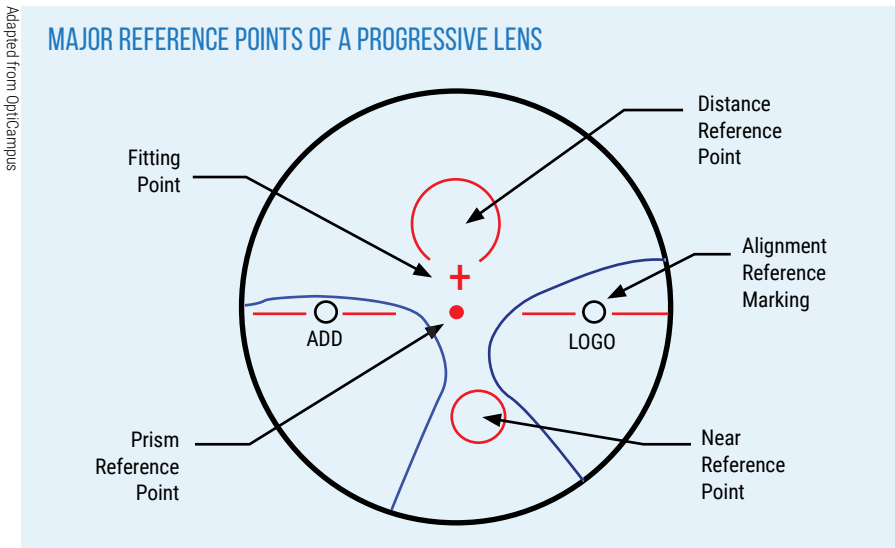
SUN OPHTHALMICS

in progressive fitting. The normal approach has been to adjust the fitting height to compensate for these postural deviations.

Alternately, the corridor length may be adjusted as well—longer corridors for the head-back tilt or shorter for head-forward postures. An example of a combined approach might lower placement of the fitting cross by 1mm to 1.5mm and compensate the reading area by specifying a 1mm to 2mm shorter corridor.

2. Corridor length. Today, issues arising from cell phone usage have required revising normal eye depression angles from 26° to 28° to a less demanding 22° to 25°.[1] But as the effective corridor value is often defined differently between manufacturers, comparing corridors between different PALs remains a challenge. For example, manufacturers may use either a varying metric of 85%, 95% or 100% of add power to define their corridor length.[6]

3. Frame fit. To further complicate achieving the best placement for a progressive, factors such as patient preference for a loose frame fit, eyelash clearance and sinus or skin sensitivity may require modifying the fitting cross placement from its position in front of the pupil.



Adapted from OptiCampus

Fig. 2. The fitting cross is an important marker that designates the point of the lens that should be placed along the center of the patient's pupil.

Fig. 3. Expand your knowledge and skill with progressive lenses to create a personalized experience that keeps patients coming back.

Troubleshooting Dissatisfaction

Despite the best laid plans of prescriber, fitter and lens designer, wearers will inevitably return dissatisfied with their progressive lenses. When rechecking the Rx, here are two exceptional situations to keep in mind:

1. Mature presbyopes. Defined as near additions of +2.00D and above, lenticular changes often precipitate changes seen in total astigmatism and sphere power. With all progressive lenses possessing some residual astigmatism, not fully correcting the eye's astigmatism can result in patients having to hold their head "just so" to see clearly in the distance—which is colloquially called "hot-spotting."

As stated previously, this effect arises from uncorrected refractive cylinder alternately interacting with the varying surface astigmatism of the progressive surface. Do not shy from prescribing the full change found in the cylinder and axis.

Further, reducing habituated plus power in the distance Rx can backfire in mature presbyopes—with new distance or driving improvements offset by unexpected loss of face-level computer utility.

2. Eye dominance and the binocular pupillary distance (PD). Essilor introduced the importance of using monocular PDs when fitting progressive lenses to help better align the narrow intermediate and near areas of early progressives. But maintaining an accurate binocular PD is of greater importance. Patients will naturally align their dominant eye to that lens's progressive umbilic—thereby placing the total error in binocular PD, if any, into the companion eye.[7]

This means that the current ANSI fabrication tolerance of 1mm per eye could actually result in a 2mm error in corridor placement for the companion

NEAR VISION NEEDS CHANGE THE GAME

In today's world, people are performing more varied and different types of near activities, many of which are centered on multitasking while using a phone or tablet. Whether it's walking down the street, cooking a meal, or having a conversation while waiting for an important text, the arrival of the smartphone, smartwatch and a cornucopia of associated apps has changed the visual experience dramatically. And people are using their phone for more than just calling and texting. They use them to secure home loans, pay bills, watch movies and even to monitor their workouts. All of this added near activity comes with a cost to our eyes and our visual comfort.

Zeiss has assembled a portfolio of progressive lens products call SmartLife for this very reason, with a goal of creating general-use progressive lenses that will—in one lens—attempt to better address all these new visual challenges.[1] But remember that even today's most robust, premium designs cannot fully solve all the unique challenges of every situation in one lens design.

Throughout our day we do many tasks that require differing visual needs, which in turn requires a lens specifically made for that task. For example, 67% of middle-aged drivers complain of traveling at night, which requires a design that is very wide in the distance and intermediate to allow drivers the freedom to move their eyes. Zeiss's DriveSafe attempts to address these needs.[2]

Drivers prefer to move their eyes rather than move their head—Zeiss DriveSafe starts the progressive corridor 2mm to 3mm lower than general purpose designs. The result is more freedom of eye movement in the distance and intermediate areas.

Zeiss DriveSafe is optimized for a mesoscopic pupil, which results in wider areas of lateral vision from smoother surface contour and reduced astigmatism power.

Newer LED headlights are a source of debilitating glare and emit an elevated amount of blue spectrum compared with halogen headlights. DriveSafe has an advanced blue light filter tailored to reduce glare specific to LED headlights.

While DriveSafe is an excellent lens choice for everyone who drives at night, its lowered progressive corridor may be less ideal for office tasks like face-level computing. There are many types of task-specific lenses that you can recommend alongside an optimized, general purpose progressive, such as a computer lens. So, the best approach to choosing one, best general-use progressive might just be to prescribe two or more pairs.

1. Zeiss. Smartlife fact sheet. www.zeiss.com/content/dam/vision-care/download/pdf/factsheets/fact-sheet-zeiss-smartlife.pdf. 2019. Accessed May 14, 2021.

2. Zeiss. Zeiss Drivesafe white paper. glarminy.files.wordpress.com/2019/07/d1b1a-9aff8-zeiss-drivesafe-white-paper.pdf. 2019. Accessed May 14, 2021.

Image: Zeiss

Personal technology, intended for frequent use throughout the day, has radically changed the habitual visual needs of patients—and, with that, their expectations.

eye—something most wearers will find disturbing to their reading comfort and utility.

A proven technique to effectively evaluate optimal binocular progressive corridor overlap involves a chart and light source, wherein the patient views the chart and wears the light source during a progressive lens verification process.[8]

Squishy Satisfaction

The one aspect of vision care that online vendors cannot address easily is the ability to follow-up on the complaints of an unsatisfied progressive wearer. So, never shy from welcoming anyone who requires after-sale attention—whether they've purchased the glasses from you or not.

From the ECP's end, the time spent learning to troubleshoot any spectacle wearer's problems informs and readies oneself better for the next patient who may have similar problems (*Figure 3*).

My mantra has always been: "What do you learn from a patient who never returns?" So, while getting wearers to 20/happy is a convenient goal, true progressive experts never refrain from exploring where the limits of progressive satisfaction lie for an individual wearer. Patient satisfaction will always be a moving target and new lens designs will always be introduced, so remain a lifelong learner in order to stay at the top of your game.

Keep in mind that trying to choose an overall "one best lens" solution for many patients may not really be a solution at all, and this is where prescribing two or more pairs is really the best recipe for optimal visual comfort and utility. ■

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~ Rob Szeliga, OD

"I found the lenses very easy to adapt to. I am typically a "progressive lens whiner" that adapts VERY slowly to new lenses (and I complain a lot!!). However, that was not the case with these new lenses."

~ Mario Gutierrez, O.D., F.A.A.O.

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PEER REVIEWED

RETINA CARE: THE NEXT WAVE

Fifteen years into the anti-VEGF era, protocols are evolving to improve outcomes, reduce treatment burden or both. Here's what's new and what's coming soon.

BY ANNA BEDWELL, OD, AND LARISSA KRENK, OD
INDIANAPOLIS, IN

As always, an extensive number of promising clinical trials are underway in the retina world. Much of the current focus is on therapeutic development and drug delivery systems, with the ultimate goal of reducing the treatment burden for those receiving anti-VEGF. Many of the therapeutics in the pipeline—mainly injectables—will have minimal direct influence on an optometrist's role, as those patients still need a referral, but they could substantially change the patient's experience at the retina specialty clinic that we prepare them for at the outset of care. So, it's incumbent on us to be as up to date as possible even on interventions we do not personally administer.

More directly affecting our own protocols, there are also a number of current and completed clinical trials of particular interest to ODs that would allow us to keep patients in the chair longer, have an active role in treatment or change our referral patterns.

Most of the focus is on diabetic eye disease and macular degeneration, but there are also exciting prospects for inherited retinal dystrophies. Here are several trials, active or recently completed, with the potential to influence our clinical practice.

Nonproliferative Diabetic Retinopathy

There may well be no topic in retina more deserving of attention from researchers than diabetic eye disease, especially in its earliest stages, given the pervasiveness of diabetes and its unfortunately inevitable increase in prevalence in the coming years.

• DRCR Protocol W
Status: in progress
Anticipated completion: May 2022
• PANORAMA
Status: complete

The goal with diabetic retinopathy (DR) certainly is to preserve vision. But when is intervention necessary? Historically, only eyes with proliferative diabetic retinopathy (PDR) or diabetic macular edema (DME) have been treated, and optometrists have referred those patients to retina specialists. Protocol W is looking at those patients with severe nonproliferative diabetic retinopathy (NPDR) to see if earlier intervention leads to a better outcome; specifically, whether it prevents progression to PDR and/or center-involved DME (CI-DME).[1,2]

The study boasts 328 participants with half randomized to sham injection and the other half to aflibercept injection.[1,2] In the trial, aflibercept is dosed at months one, two and four, and then every four months thereafter. At the two-year mark, dosing will be decided as needed by the examining investigator.[1,2]

Protocol W is not the first prospective study to look into treating NPDR. The PANORAMA study

The DRCR Retina Network, formed in 2002, is a collaborative group dedicated to clinical research applicable to improving patient care. The network originally focused on diabetic retinopathy research, but since 2018 has expanded their research to all retinal disorders. DRCR protocols, past and present, can be accessed at public.jaeb.org/drcrnet

About the authors

Dr. Bedwell is a clinical associate professor at Indiana University School of Optometry. She serves as editor of the Optometric Retina Society's e-newsletter. **Dr. Krenk** completed a primary care residency at the Indianapolis Eye Care Center. She is now a visiting lecturer for Indiana University School of Optometry and continues to teach optometry students and see patients at the Indianapolis Eye Care Center. They have no financial interests to disclose.

demonstrated an impressive two-step reduction in Diabetic Retinopathy Disease Severity Scale (DRSS) for 58% of aflibercept-treated participants at six months.[3,4] Unlike Protocol W, PANORAMA included eyes with both moderately severe NPDR (DRSS level 47) and severe NPDR (DRSS level 53). PANORAMA included two different treatment groups: Q8-week aflibercept dosing and Q16-week.[3,4]

With the two-year results of PANORAMA, announced in spring 2020, additional questions arose regarding treatment frequency. For study year two, the Q16-week treatment group maintained a two or more step improvement.[5] However, the Q8-week group, when switched at week 52 to PRN dosing, showed a decline from 80% to 50% with two or more step improvement.[5]

The PANORAMA results unquestionably support earlier treatment in NPDR, but there are still lingering questions, such as how frequently to treat and when anti-VEGF injections can be stopped or tapered.

Clinical take home: *Change in management of moderately severe and severe NPDR is on the horizon. Consider referral for patients that fall into either category.*

• DRCR Protocol AF

Status: enrolling

Anticipated completion: January 2027

The DRCR recently announced the start of a Phase III clinical trial called Protocol AF that will study the effect of fenofibrate on NPDR.[6,7] Fenofibrate is a peroxisome proliferator-activated receptor alpha agonist used to treat hypercholesterolemia by lowering triglycerides and low density lipoprotein and increasing high density lipoprotein.[8]

Fenofibrate's positive effect on DR was first recognized by the FIELD study, which showed the number of patients on fenofibrate that went on to need laser treatment for DR or DME was significantly lower compared to controls.[9] Additionally, the ACCORD study found a lower rate of NPDR progression in those taking fenofibrate plus simvastatin compared to the placebo plus simvastatin group.[10]

Neither the FIELD or ACCORD studies were primarily focused on the outcome of DR progression, which makes Protocol AF unique. Even though this study is still in the enrollment phase, it is worthy of being on optometrist's radar as this is a potential means for our involvement—whether in comanagement or direct treatment roles—in an intervention for NPDR. Protocol AF seeks to enroll 910 participants with NPDR (without CI-DME) to follow over four years.[6,7] Participants will be randomized to once daily fenofibrate 160mg or placebo.[6,7]

Clinical take home: *Fenofibrate is an oral treatment under investigation for mild to moderately severe NPDR.*

TABLE 1. DIABETIC RETINOPATHY SEVERITY SCALE

Level	Severity	Characteristics
10	No DR	–
20	Very mild NPDR	MAs only
35	Mild NPDR	MAs + HEs, CWS, +/- mild RHs
43	Moderate NPDR	43A: moderate RHs in four quadrants or severe in one
		43B: mild IRMA in one to three quadrants
47	Moderately severe NPDR	47A: 43A + 43B
		47B: mild IRMA in four quadrants
		47C: severe RH in two to three quadrants
		47D: venous beading in one quadrant
53	Severe NPDR	53A: ≥ 2 level 47 characteristics
		53B: severe RH in four quadrants
		53C: moderate-severe IRMA in 1+ quadrant
	Very severe NPDR	53D: ≥ two level 53A-D characteristics
61	Mild PDR	NVE < 0.5 DA in 1+ quadrants
65	Moderate PDR	65A: NVE ≥ 0.5 DA in 1+ quadrants
		65B: NVD < ¼ to ⅓ DAs
71, 75	High-risk PDR	Larger NVD, or NVE ≥ 0.5 DA with VH or PRH, or VH or PRH obscuring ≥ 1 DA
81, 85	Advanced PDR	View partially obscured by VH or PRH from NV, or macula involving retinal detachment

Level 43 and higher all require MAs.

Abbreviations—CWS: cotton wool spot, DA: disc area, HE: hard exudate, IRMA: intraretinal microvascular abnormality, MA: microaneurysm, NPDR: non-proliferative diabetic retinopathy, NVD: neovascularization at the disc, NVE: neovascularization elsewhere, PDR: proliferative diabetic retinopathy, PRH: preretinal hemorrhage, RH: retinal hemorrhage, VH: vitreous hemorrhage

Source: Adapted from the American Society of Retina Specialists Clinical Practice Guidelines

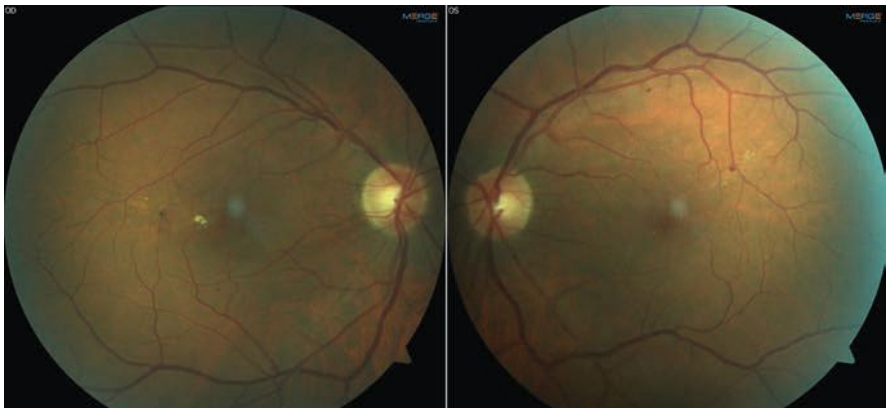
Diabetic Macular Edema

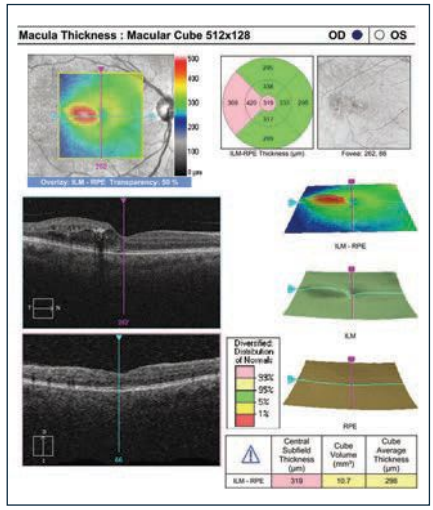
Recalcitrance to treatment makes DME particularly nettlesome, but new treatment protocols offer hope for substantial improvement.

• DRCR Protocol V

Status: complete

In the current era, with OCT at our fingertips, DME is assessed as either center involved or non-CI, depending on the presence of thickening within the central subfield zone at the fovea.





Fundus images and OCT of a patient with moderate NPDR OU and CI-DME OD. The patient's best corrected acuities were 20/25 OD and 20/20 OS. The comanaging retina specialist elected to monitor the CI-DME OD based on the findings of DRCR Protocol V.

This is a change from the historical means of looking for characteristics of clinically significant macular edema (CSME). But what about patients who have mild CI-DME and a preserved visual acuity? Protocol V proved instrumental in providing guidance, as it assessed CI-DME eyes with 20/25 vision or better.[11]

A total of 702 participants were randomized to observation, treatment with aflibercept or treatment with laser photocoagulation.[11,12] After two years, the study found no significant difference in visual acuity between each treatment groups and observation.[11,12] The patients in the observation group that went on to decline in visual acuity were ultimately treated with aflibercept.[12]

Even though this trial has been completed and published, it is still not widely known among ODs, who have to pull the trigger on referral. It is further challenging for patients alike who have good visual acuities with minimal visual complaint and are not ready to be locked into a sometimes-endless cycle of injections.

Clinical take home: *Patients with CI-DME and a visual acuity of 20/25 or better can be safely observed. This warrants a discussion with your local retina specialists to see if these patients necessitate a referral or can continue observation by you.*

• YOSEMITE, RHINE

Status: in progress
Anticipated completion: summer/fall 2021

A new therapeutic that allows for fewer injections would be quite exciting news for our patients, particularly those in rural areas who may not be able to easily get to a retina specialist. Anti-VEGF therapy has done wonders for DME and wet AMD, but it comes at a price: frequent office visits impose a time burden on patients and their caregivers, and its cost weighs heavily on the healthcare system.

Faricimab (Roche) could be a game-changer. It's not just another anti-VEGF treatment but rather a bispecific antibody that targets two pathways: VEGF-A and angiopoietin-2.[13] The Phase III clinical trial data, released in February, was positive, showing half of faricimab-treated individuals were able to be dosed every four months.[13] The YOSEMITE and RHINE trials are investigating faricimab in the treatment of CI-DME, with each trial including over 900 participants.[14,15] There are sister trials underway for wet AMD (TENAYA and LUCERNE) that anticipate completion in 2022.

Clinical take home: *Faricimab is an investigational drug targeting two pathways with the potential to be dosed less frequently than traditional anti-VEGF.*

Dry Macular Degeneration

One of the toughest nuts to crack in retina, dry AMD may be on the verge of breakthroughs that could finally offer more direct interventions.

• SAGA

Status: in progress
Anticipated completion: March 2022

Even though many therapeutics have been explored, none to date have been successful in slowing the progression of geographic atrophy (GA), much to the frustration of patients and optometrists alike.

Currently, there a few oral treatments in trial that, if successful, could bring optometrists into the treatment protocol. SAGA is a Phase II/III multicenter placebo-controlled clinical trial investigating the role of ALK-001 (Alkeus) in patients with GA.[16]

This drug is a modified form of vitamin A taken daily as an oral capsule. Over time, aggregates of vitamin A create dimers that accumulate in the RPE and underlying Bruch's membrane.[16] The dimers are thought to be toxic to the retina, which has been proposed as a mechanism for the AMD development.[17] Synthetic ALK-001 contains vitamin A with deuterium, which slows formation of vitamin A dimers without compromising the normal function of the visual cycle.[17]

The primary outcome of the study is to assess the growth rate of GA

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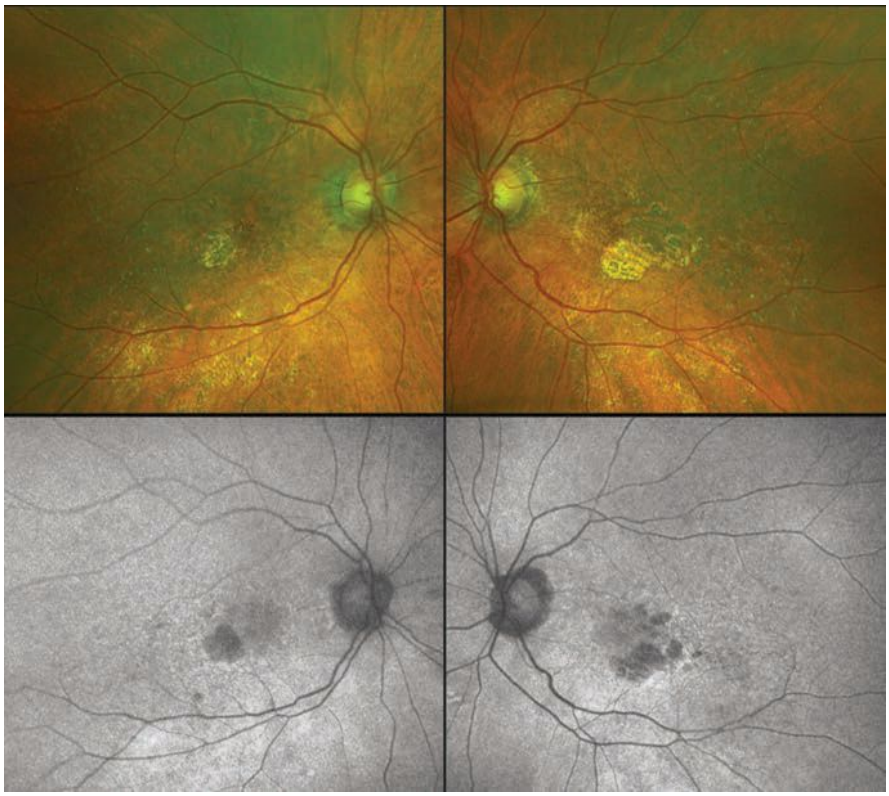

Color fundus images (top) and fundus autofluorescence (bottom) of a patient with geographic atrophy (GA) from macular degeneration OU. The SAGA study is exploring modified vitamin A treatment for GA patients such as seen here.

lesions over a two-year period in those taking ALK-001 compared to placebo.[16] If ALK-001 proves to be effective in slowing the progression of atrophy, it could provide a new treatment option to help reduce vision loss in those with geographic atrophy from AMD.

Additionally, Stargardt's disease is caused by a mutation in the *ABC4* gene, which affects the processing of vitamin A.[18] This also leads to the accumulation of toxic vitamin A dimers that may contribute to vision loss.[18] A similar clinical trial is being conducted for patients with Stargardt's and is also expected to be completed in early 2022.[18]

Clinical take home: *Oral supplementation with ALK-001 is in trial for slowing the progression of geographic atrophy in patients with AMD and Stargardt's.*

• LIGHTSITE III

Status: enrolling
Anticipated completion: June 2022

Photobiomodulation (PBM)—a growing trend in medicine—is low-level light therapy that uses specific wavelengths in the visible light to near-infrared ranges, to target certain tissues and stimulate cellular function.[19] The idea behind PBM focuses on changes at the mitochondrial level, leading to the upregulation of ATP production, which is a major form of energy necessary for normal cellular functions.[19] Other changes produced at the cellular level may also work to reduce oxidative stress.[19]

LIGHTSITE III is a two-year study consisting of 96 subjects with dry AMD that will receive repeated sham or PBM treatments at several time-points using the Valeda Light Delivery System (LumiThera).[20] Findings from LIGHTSITE I demonstrated clinically significant improvements in best-corrected visual acuity and contrast sensitivity after a series of nine treatments over three weeks.[19] Approximately 50% of PBM-treated subjects showed improvement of five or more letters vs. 13.6% in sham-treated subjects directly after treatments at one month.[19] This was followed by a decline over the next six months and repeated treatments over time will be needed to maintain efficacy.[19]

Additionally, improvements in drusen volume and thickness were observed, although the authors admitted that more long-term evidence is needed to correlate these anatomical changes with disease regression or progression.[19]

If this therapy is approved in the United States, optometrists could be at the forefront of providing regular PBM treatments in addition to the close monitoring already provided to patients with dry AMD.

Clinical take home: *Photobiomodulation is a low-level light therapy that may improve visual function in patients with dry AMD. Treatments will likely be needed at regular intervals to maintain efficacy.*

Wet Macular Degeneration

The most successful area of retina care—anti-VEGF therapy for wet AMD—is swiftly evolving beyond traditional monthly or bimonthly injections to more patient-friendly approaches.

• ARCHWAY

Status: in progress
Anticipated completion: late June 2021

The introduction of intravitreal anti-VEGF agents significantly changed the treatment of wet AMD, allowing patients to retain and even improve visual function. However, repeated injections, often every four to six weeks, place a considerable burden on patients and make it difficult for optometrists to continue follow-up care after referral to ophthalmology.

ARCHWAY is an exciting development in that it studies a new way to deliver anti-VEGF therapy through a port delivery system (PDS, Genentech).[21] The PDS is a permanent, refillable intraocular implant that provides continuous

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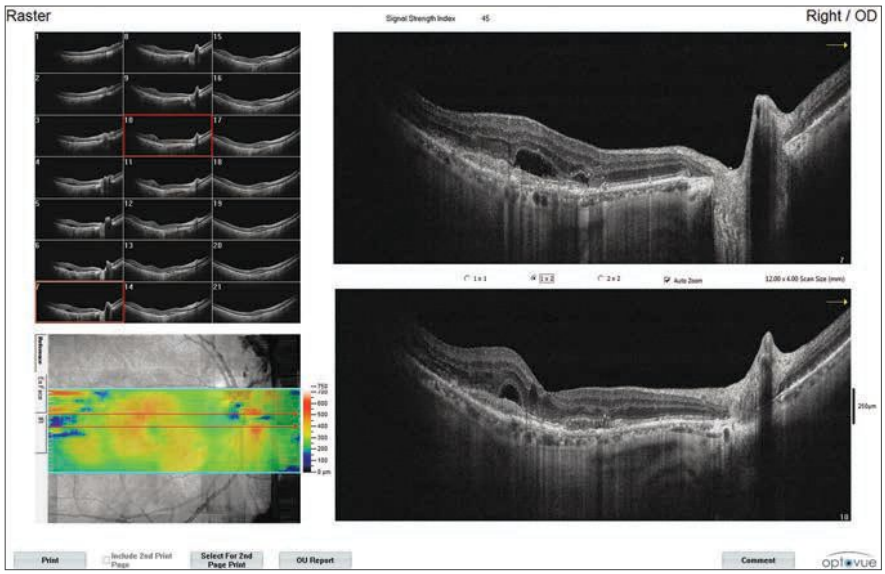

ARCHWAY marks a change in anti-VEGF treatment as it focuses on how the therapeutic is delivered, as opposed to the drug itself.

delivery of a customized formulation of ranibizumab.[22] The device is surgically implanted and then can be refilled in a normal clinical setting.[22] ARCHWAY is a Phase III trial that compares PDS refilled every six months to monthly ranibizumab intravitreal injections.[21]

PDS was shown to be non-inferior and equivalent to monthly ranibizumab injections, both in terms of BCVA and controlling retinal thickness.[22] Additionally, 98.4% of patients were able to maintain the six-month refill schedule, which could significantly reduce the number of anti-VEGF treatments to as few as two per year.[22] An extension of the ARCHWAY study, called PORTAL, is underway to examine long-term effects of PDS.

At this time, it is difficult to say how this effort might affect optometrists, as refills of the PDS are still necessary to maintain therapeutic levels of anti-VEGF. However, this study marks an exciting change in anti-VEGF treatment, as it focuses on how the therapeutic is delivered, as opposed to the agent itself.

Additionally, there are two trials underway to study the safety and efficacy of PDS in subjects with DME and those with DR without CI-DME, the PAGODA and PAVILION trials, respectively. They are still actively enrolling and are estimated to be completed in September 2024.[23,24]

Clinical take home: *Continuous anti-VEGF therapy through use of an intraocular implant may reduce the frequency of anti-VEGF intravitreal injections in patients with wet AMD and DME.*

• OPTIC

Status: in progress
Anticipated completion: June 2022

In one of the most promising trials to date, OPTIC has the potential to greatly reduce the frequency of intravitreal anti-VEGF injections, using gene therapy as a treatment approach.[25] The candidate, ADVM-022 (Adverum), is a gene therapy vector that can be delivered in-office by intravitreal injection.[25] It contains an adeno-associated virus capsid carrying a coding sequence for aflibercept, which the eye can use to endogenously produce continuous levels of anti-VEGF molecules.[26]

The safety and efficacy of prolonged intraocular expression of aflibercept was evaluated in preclinical trials and therapeutic levels were confirmed out to at least 30 months with no adverse effects on normal retinal structure or function in non-human primates.[26] OPTIC is a Phase I clinical trial studying the safety and tolerability of high and low doses of ADVM-022 in subjects with active choroidal neovascularization secondary to AMD.[25]

New interim data as of November 2020 showed that ADVM-022 is well tolerated with a favorable safety profile at both high and low doses.[27] Secondary outcomes found that mean BCVA is maintained and mean central subfield thickness is maintained or improved at both doses.[27] In addition, most patients remain free of supplemental anti-VEGF injections with one patient in the higher dose cohort showing sustained efficacy out to 92 weeks from initial injection.[27] In the lower-dose cohorts, two-thirds of patients did not need supplemental anti-VEGF injections with follow-up ranging from 34 to 68 weeks after injection.[27]

The results of the OPTIC trial thus far are extremely promising and could potentially represent a one-time treatment for wet AMD. This could allow optometrists to continue to be a more active part of a patient's management and follow-up care, as frequent anti-VEGF injections would be obviated. Two Phase III trials are planned to begin at the end of 2021.[28] Additionally, the INFINITY trial, a Phase II study looking at the effect of ADVM-022 on subjects with DME, is set to finish in January 2022.[29]

Clinical take home: *ADVM-022 is a gene therapy vector given as a one-time intravitreal injection and carries a genetic coding sequence for endogenous intraocular production of aflibercept.*

Inherited Retinal Disease

Gene therapy continues to be investigated for inherited retinal diseases (IRDs), even though new applications into wet AMD and DME—described above—have been initiated. At this time, Luxturna (Spark therapeutics) continues to be the only FDA-approved gene therapy for an IRD. It is specific to a biallelic *RPE65* mutation

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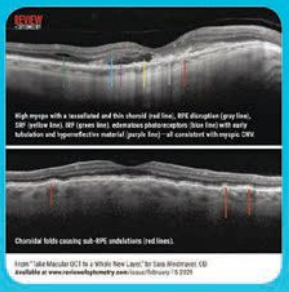

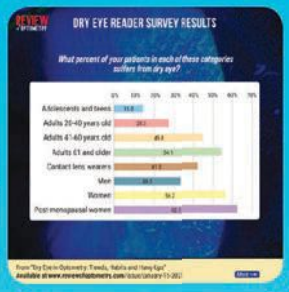

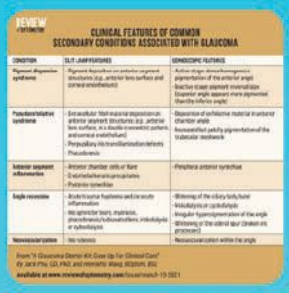

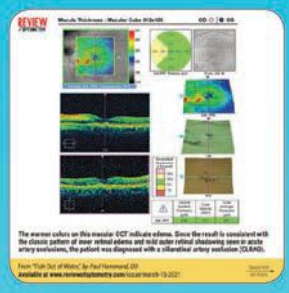

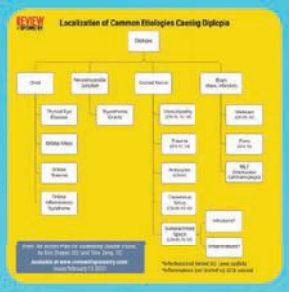

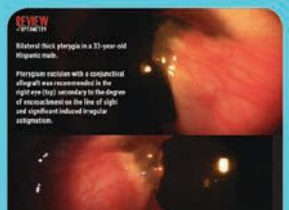

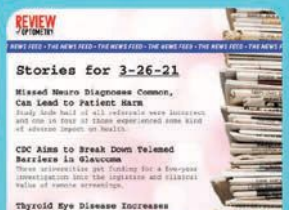

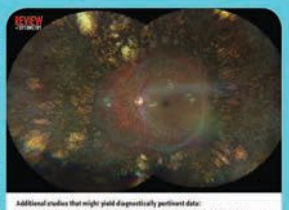

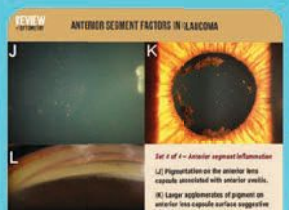

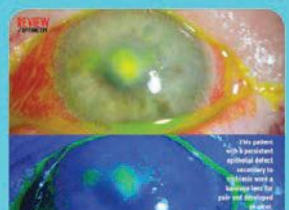

TABLE 2. CLINICAL TRIALS UNDERWAY FOR INHERITED RETINAL DISEASES

Drug	Manufacturer	Disease Studied	Stage
AAV-RPGR	MeiraGTx	X-linked RP	Phase I/II; 46 participants
AAV-CNGB3 or AAV- CNGA3	MeiraGTx	Achromatopsia	Phase I/II; 72 participants
AAV2-REP1 (BIIB111)	Biogen	Choroideremia	Phase III; 160 participants
rAAV2tYF-GRK1-RPGR (AGTC-501)	Applied Genetic Technologies	X-linked RP	Moving into Phase II/III (SKYLINE, VISTA)
AGTC-402 or AGTC-402	Applied Genetic Technologies Corporation	Achromatopsia	Phase I/II; 24 participants CNGA3 mutation, and 28 with CNGB3
HORA-PDE6B	Horama	Autosomal recessive RP (PDE6B mutation)	Phase I/II; 15 participants

Source: Manufacturers' press releases and clinicaltrials.gov.

that is present in a small subset of those with either Leber's congenital amaurosis or retinitis pigmentosa.[30] Other clinical trials show promise, particularly in choroidermia. *Table 2* details the gene therapy trials that are underway for IRDs. The majority of gene therapy trials use an adenovirus vector and subretinal delivery.

Nevertheless, trials with intravitreal delivery and optogenetic gene therapy have also made their way to human clinical trials. However, such a small portion of the population is afflicted with an IRD, which adds to the challenge of designing clinical trials with sufficient participants.

No cost, in-office genetic testing programs for inherited retinal diseases can diagnose these conditions promptly and isolate the causative gene mutation, which helps to identify patients eligible for trials.

Takeaways

At times, the clinical trial landscape in the retina world may seem to be dominated by injectable therapeutics—it certainly has been since the early 2000s—but there are still many retina clinical trials applicable and relevant to optometry. These research projects guide our referral patterns, facilitate our patient education and identify prospective treatment, including some potentially prescribed by ODs. Several of these trials are influencing patient care at the moment, while others should be kept on our radar in the years to come. ■

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Menicon Celebrates Milestone 70th Anniversary

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"We are so proud to be celebrating this milestone 70th anniversary. Menicon manufactures specially controlled medical devices, so our top priority has always been the safety of our customers' eyes,"
— Dr. Hidenari Tanaka, President & CEO of Menicon.

"We are so proud to be celebrating this milestone 70th anniversary. Menicon manufactures specially controlled medical devices, so our top priority has always been the safety of our customers' eyes," said Dr. Hidenari Tanaka, President & CEO of Menicon. "Challenge, safety, end-user first philosophy and high-quality products remain the cornerstone of Menicon's continued growth and success. These principles are why we are able to celebrate such a significant anniversary."

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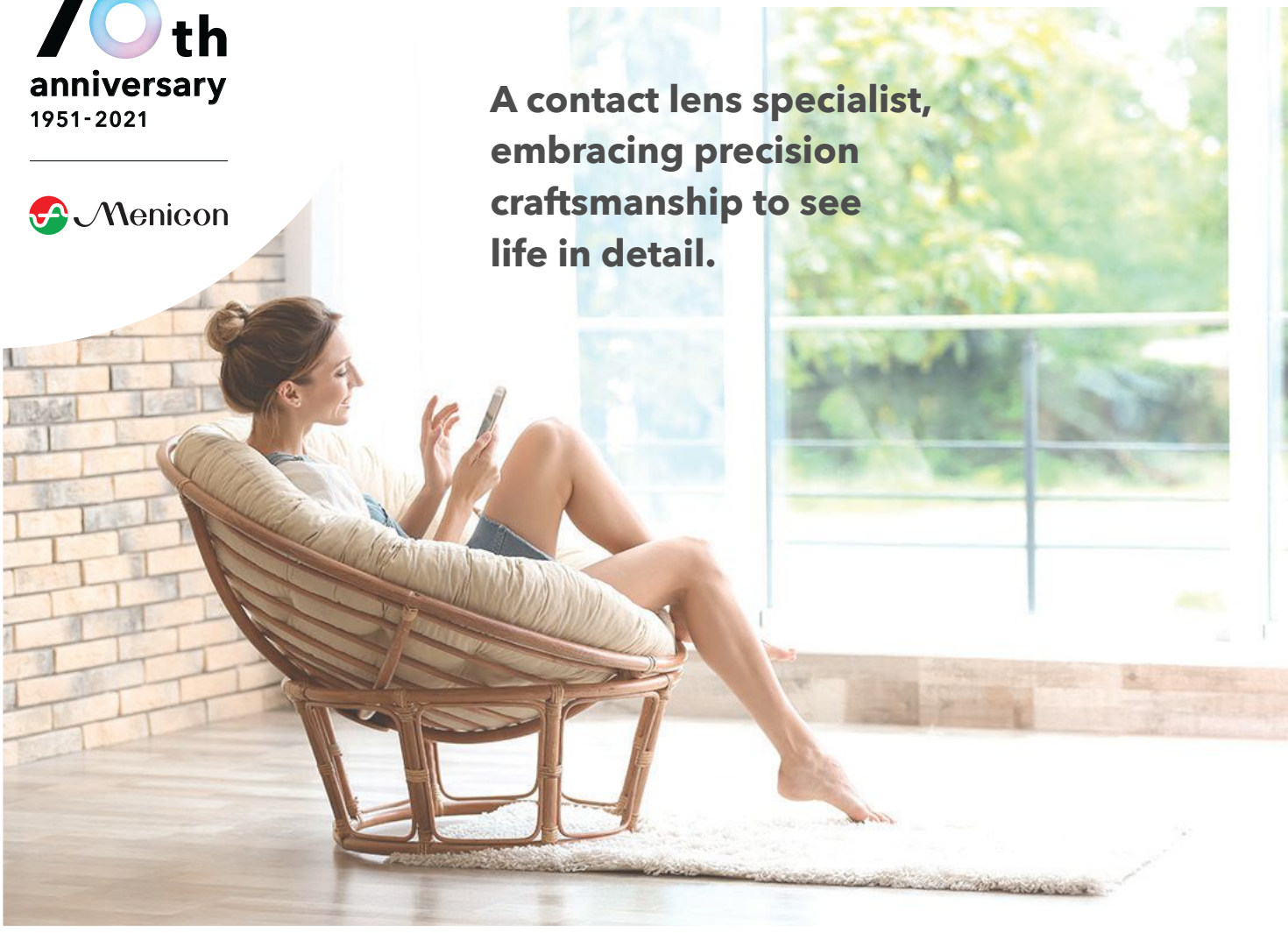

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GET SERIOUS ABOUT CENTRAL SEROUS CHORIORETINOPATHY

Mineralocorticoid receptor antagonists may present an effective option for early intervention.

BY MOHAMMAD RAFIEETARY, OD[1]
JESSICA HAYNES, OD[1]
ROYA ATTAR, OD[2]
[1]MEMPHIS, TN, [2]JACKSON, MS

Central serous chorioretinopathy (CSCR) is a common retinal disorder that results in vision loss and alteration of visual function.[1] It is considered a pachychoroid disease, a category of diseases that includes polypoidal choroidal vasculopathy, pachychoroid neovasculopathy and pachychoroid pigment epitheliopathy.[2] Despite CSCR's prevalence as the fourth most common non-surgical retinopathy behind macular degeneration, diabetic retinopathy and branch retinal vein occlusion, there is still no standard treatment regimen for the condition.[1]

A variety of therapies and potential lifestyle modifications have been explored with different outcomes, including focal photocoagulation, photodynamic therapy (PDT) with verteporfin, intravitreal anti-vascular endothelial growth factor (anti-VEGF) agents and several classes of oral and topical medications. More recently, investigation into mineralocorticoid receptor antagonist (MRA) drugs has produced hopeful results. With these medications in mind as an early intervention option, this article proposes a potential treatment scheme for patients diagnosed with CSCR.

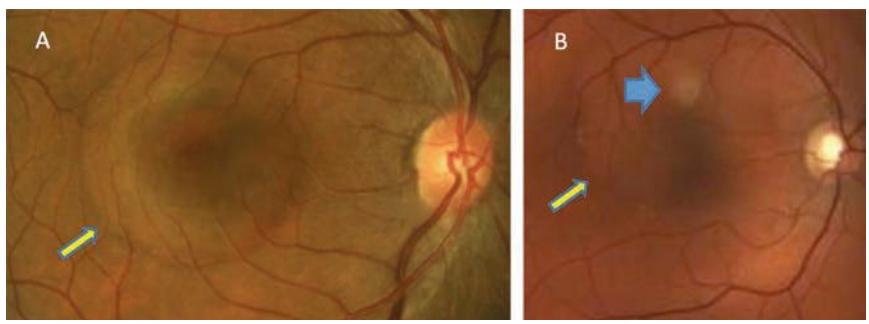

Fig. 1. In patients with acute CSCR, serous retinal detachment can be seen (yellow arrows). PED is not clinically detected in patient A but can be seen in patient B (blue).

Background

CSCR is typically found in men and women of all ethnic backgrounds after the third decade of life. The common acute presentation occurs most often in middle-aged Caucasian males.[3,4] The incidence rate is approximately 10 men and two women per 100,000 people.[3,4]

The most common risk factors for the condition include "type A" personality, pregnancy and systemic use of corticosteroids and adrenergic receptor inhibitors. Other identified potential risk factors include systemic hypertension, *Helicobacter pylori* gastrointestinal disease, testosterone supplementation and sleep apnea.[5-7]

Clinical findings depend on the duration of CSCR, with findings associated with the condition detectable during retinal examination. In acute cases, patients will present with varying degrees of visual disturbance. Usually, a round serous retinal detachment (SRD) that varies in size is noted within the posterior pole. A pigment epithelial detachment (PED) may or may not be detectable clinically (*Figure 1*).

About the authors

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Ancillary tests, such as spectral-domain OCT, fluorescein angiography (FA), indocyanine green (ICGA) angiography, OCT angiography and fundus autofluorescence (FAF), are helpful to detect the various associated features of CSCR.[8-10] These imaging modalities are not only useful in proper diagnosis of the condition, but also in determination of staging (acute or chronic) (*Figures 2 and 3*).

On OCT, a common feature of CSCR is a greater-than-average choroidal thickness (*Figure 4*).[11] One study reported that patients with CSCR had a mean subfoveal choroidal thickness of 475±138µm compared with 372±120µm in healthy patients.[11]

Although most acute cases spontaneously resolve with minimal to no long-term vision loss, persistent or chronic CSCR can result in permanent alteration of the retinal pigment epithelium (RPE) and retinal photoreceptors (RPs). This can consequently cause permanent vision loss. Therefore, in chronic cases lasting longer than four months, therapy should be targeted to lessen the risk of permanent functional visual loss.[12-14]

The exact pathophysiology of CSCR is not well known, thus a gold standard of care has not yet been established.[6] Investigational treatments have been based on the potential pathophysiological and etiologic factors.

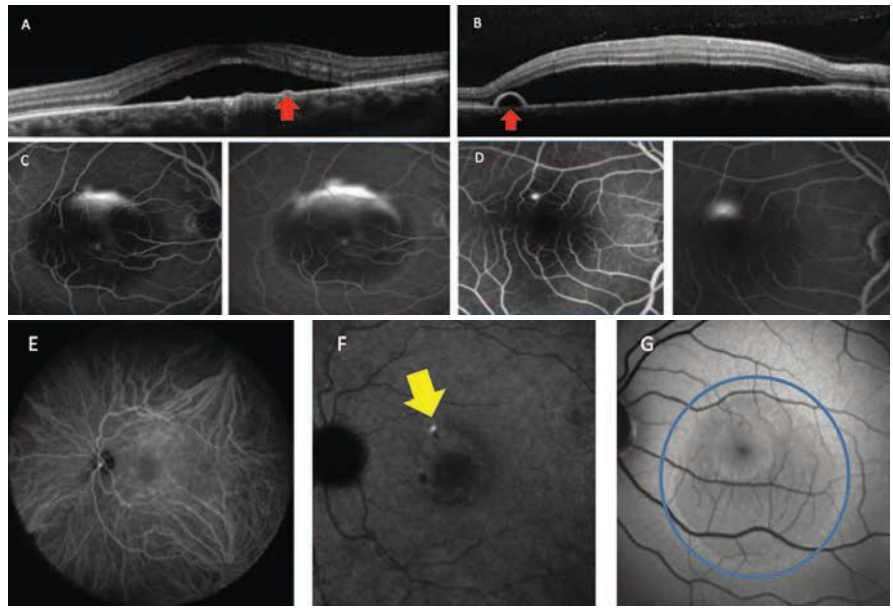

Fig. 2. This patient has acute CSCR. OCT imaging demonstrates neurosensory serous retinal detachments, with (A) showing a flat PED while (B) shows a bulging PED (red arrows). Fluorescein angiography (C) shows the classic "smokestack" pattern of leakage and (D) the more common "inkblot" pattern. Indocyanine green angiography shows the mid (E) and late (F) phases. Active leakage can be seen in the late phase (yellow arrow). Fundus autofluorescence (G) shows hypo-autofluorescence in acute or early cases (blue circle).

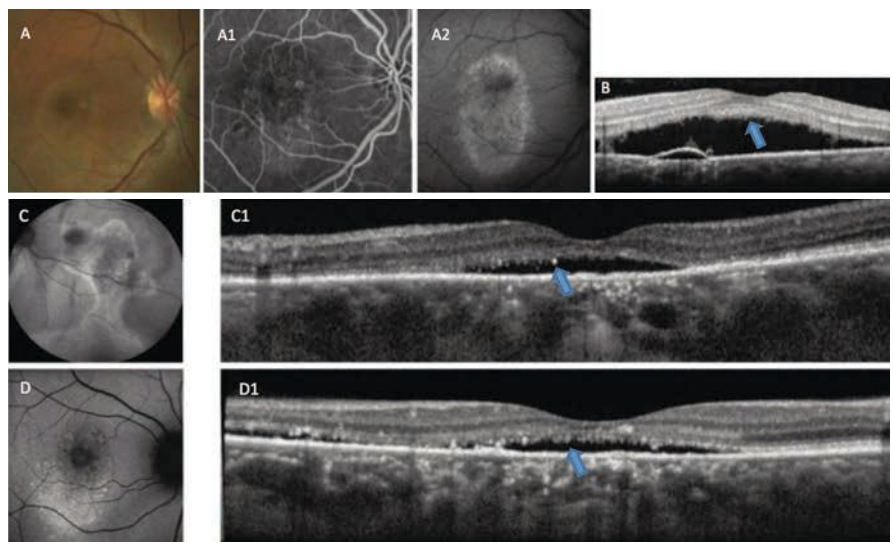

Fig. 3. This patient is suffering from chronic CSCR. In (A), RPE changes can be seen in the macula. Fluorescein angiography (A1) shows staining of the altered RPE, and fundus autofluorescense (A2) shows hyper-autofluorescence indicative of RPE damage. OCT (B) shows accumulation of RP outer segment shedding ("shaggy" photoreceptors), a phenomenon associated with choroidal melanomas (blue arrow). Autofluorescence (C and D) in other cases shows a variable mix of autofluorescence associated with chronicity and recurrence. OCT (C1 and D1) shows adverse alteration of the outer retina (blue arrows).

Treatment Strategies

Optometry, in collaboration with retina surgeons, has taken several different routes in treating CSCR and establishing a consistent treatment protocol.

Thermal photocoagulation with argon laser. This modality—the oldest form of treatment—is based on mechanically treating the PED or RPE defect, which is the assumed defective, "leaky" area causing SRD. This area can be detected by FA and ICGA.[15,16] Damage to this area can terminate the active leakage and result in faster resolution of the SRD; however, due to collateral damage caused by the thermal laser, this therapy cannot be applied to diffuse areas or regions that are too close to the fovea. Risks include formation of scotoma, formation of laser scars and potentially development of choroidal neovascular membranes.[15]

PDT with verteporfin. This treatment addresses the notion that CSCR is a condition of leaky, abnormal choroidal vessels, specifically within the choriocapillaris. Application of PDT

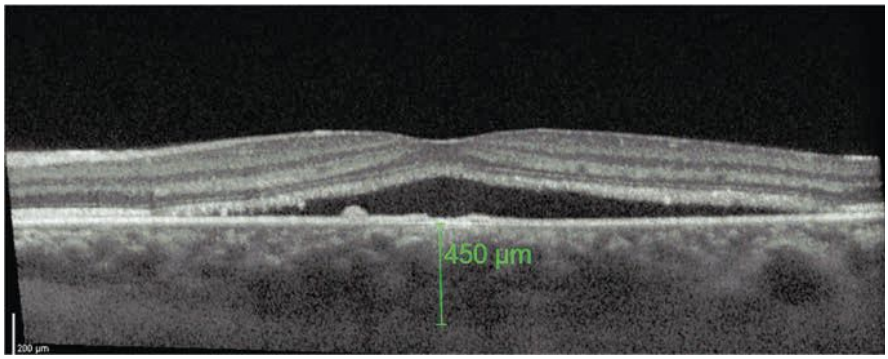

Fig. 4. A thicker-than-average choroid is characteristic of CSCR.

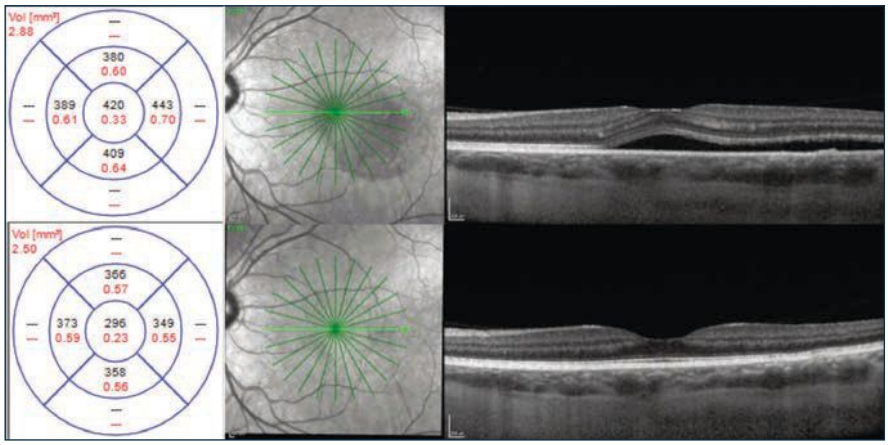

Fig. 5. Acute case of CSCR (top). Resolution at one year without treatment (bottom).

laser is thought to lead to a cascade of events causing reduced choriocapillaris congestion and vascular remolding, resulting in decreased choroidal permeability.[17] PDT laser can be applied to broader and more central areas than focal argon laser, and is often guided by FA or ICGA to target leaky areas. The initial short-term case series followed standard PDT protocol.[18] Since then, investigation into modified strategies, such as half-dose PDT and half-fluence PDT, have shown good results with a better risk profile. Unfortunately, a consensus still hasn't been reached on PDT protocol in CSCR patients.[19] Clinically, PDT laser availability and cost coverage are practical barriers to the treatment.

Intravitreal injection of anti-VEGF. This route has also been explored as a possible therapeutic option for CSCR, as a chemical method of altering choriocapillaris hyper-permeability. Multiple small case series show potential benefit with anti-VEGF treatment.[20-24] However, skepticism of the treatment exists, as CSCR does not seem to be VEGF-driven and increased levels of VEGF are not found in those with the condition. A recent meta-analysis could not verify a positive effect of intravitreal bevacizumab in patients with CSCR.[25] It is of importance to note that patients with CSCR are at increased risk of developing choroidal neovascularization, of which anti-VEGF is the treatment of choice.[1]

Topical meds. Various topical agents have been investigated, including non-steroidal anti-inflammatory drugs (NSAIDs) and carbonic anhydrase inhibitors (CAIs). Multiple small-cohort retrospective trials and case studies report increased rates of subretinal fluid (SRF) reabsorption in acute CSCR with use of topical NSAIDs.[26-30]

Critics of this treatment modality suggest a possible placebo effect, especially in those with a type A personality, and dispute a proven inflammatory etiology of CSCR.[31] A prospective trial investigated topical dorzolamide in patients with chronic CSCR and found a greater improvement in central macular thickness at three months compared with controls.[32]

Systemic meds. Another known driving factor behind this condition is increased levels of corticosteroids, as a high incidence of CSCR is associated with systemic steroid use, pregnancy and potentially episodes of high stress in susceptible individuals. A number of off-label systemic medications have been explored with this mechanism in mind, showing a range of therapeutic efficacy. These therapeutic agents include MRAs, rifampin and CAIs such as acetazolamide.[33-37] At present, the most promising drug category for the treatment of CSCR is MRAs.

MRAs. A number of reports have shown the benefit of two steroidal compound mineralocorticoid receptor antagonists in the treatment of acute and chronic CSCR: spironolactone and eplerenone.[38-47]

Spironolactone is a non-selective aldosterone (a mineralocorticoid hormone) antagonist and potassium-sparing diuretic indicated for the management of primary hyperaldosteronism and edematous-related conditions, congestive heart failure (CHF), cirrhosis of the liver and nephrotic syndrome. It is also indicated for treatment of essential hypertension, hypokalemia and severe heart failure.

This agent is contraindicated in patients with anuria, acute renal insufficiency, significant impairment of renal excretory function or hyperkalemia. It interacts with angiotensin-converting enzyme inhibitors, alcohol, barbiturates, narcotics, pressor amines and skeletal muscle relaxants.

While spironolactone has a good affinity for the mineralocorticoid receptor, being non-specific it also binds to progesterone receptors, causing dose-dependent hormonal side effects.[4]

Adverse reactions may include gastric bleeding, nausea/vomiting, diarrhea, gynecomastia, erectile dysfunction, irregular menses, agranulocytosis, hypersensitivity, hyperkalemia, mental

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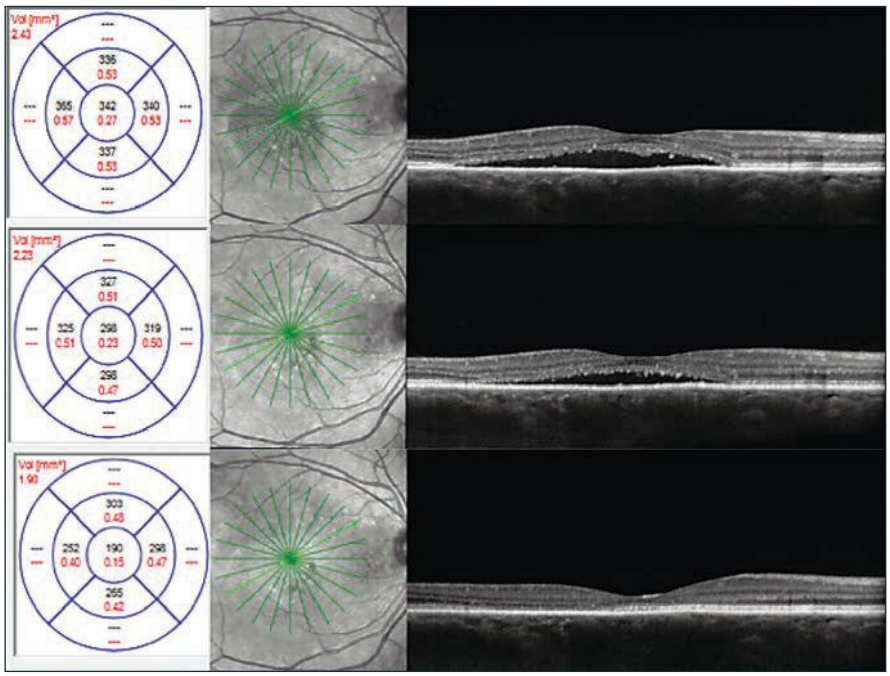

Fig. 6. CFT was 342µm at initial presentation (top), 298µm after one month of treatment with eplerenone (middle) and 190µm after two months (bottom).

dysfunction, headache, drowsiness and renal dysfunction.

Eplerenone, created to lessen the hormonal side effects of spironolactone, is a selective aldosterone antagonist indicated for improving survival of stable patients with left ventricular systolic dysfunction, CHF after myocardial infarctions or hypertension as a mono or combination therapy. While the side effects of eplerenone are favorable compared to those of spironolactone, it is a much less potent MRA. Spironolactone has a 40-fold higher binding affinity to the mineralocorticoid receptor in comparison with eplerenone.[4]

For a complete list of contraindications, precautions and adverse reactions associated with both MRAs, refer to the prescribing information that accompanies each. In addition to obtaining a complete history of illnesses, medications and allergies, prescribers should consult the patient's primary care physician and obtain baseline serum potassium levels before administering these medications.

The usual off-label administered dose for treatment of CSCR with spironolactone is 50mg daily and 25mg to 50mg daily for eplerenone until resolution of SRF.[38-47] At this dose, spironolactone can reduce both SRF and subfoveal choroidal thickness compared with placebo in patients with persistent SRF due to CSCR.[38-40] Eplerenone has also resulted in significant structural and functional improvement in patients suffering from chronic CSCR.[41-47]

Proposed strategy. Due to the variability of treatment response, there is currently no standard of care in managing CSCR patients. As evidenced by combined treatment strategies and ongoing investigation, no single therapy works sufficiently in all patients. As such, treating patients with CSCR is a highly individualized process with many different considerations and options to take into account. Weighing the risk vs. the benefit of therapy must always be at the forefront of the physician's treatment approach. A tentative treatment protocol, incorporating MRA as an early treatment option, involves the following:

- Obtain case history.
- Alter any modifiable risk factors.
- Determine if condition is chronic or acute.
- If acute, monitor. If the case doesn't resolve in four months, proceed to chronic staging.
- If chronic, initiate treatment with MRA if not contraindicated and obtain baseline serum potassium levels. Monitor for one month.
- If improvement is observed, continue therapy until SRD resolves before discontinuing and monitoring.
- If improvement is not observed, consider switching to an alternate MRA and obtain FA or ICGA to guide laser treatment.
- In the case of localized, non-central leakage, apply a focal laser.

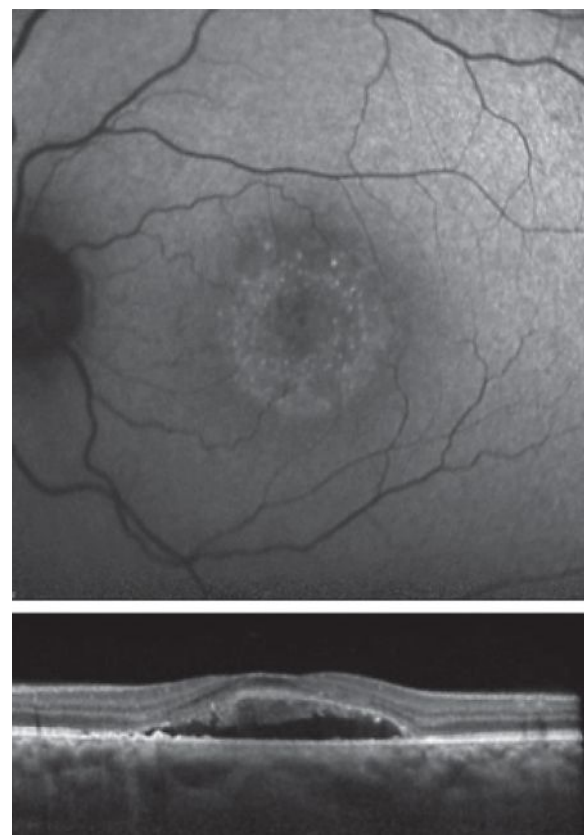

Fig. 7. Fundus autofluorescence (top) and OCT (bottom) are both suggestive of chronic disease.

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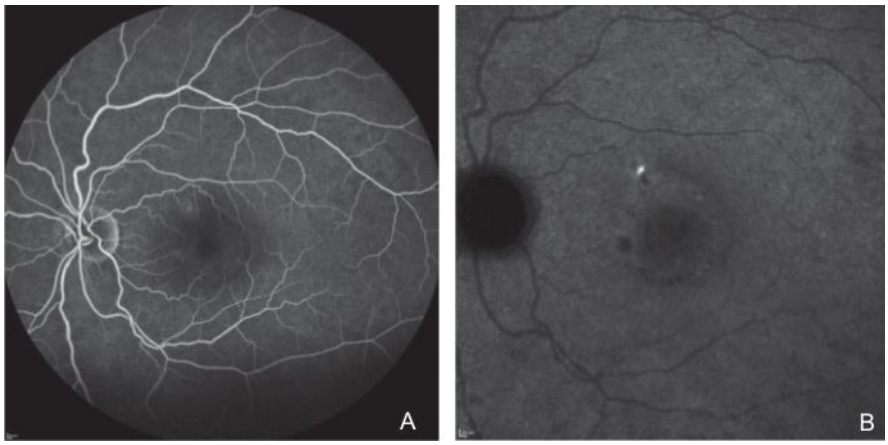

Fig. 8. Localized area of leakage on fluorescein angiography in (A) and ICGA in (B).

- In the case of diffuse, central leakage, apply PDT.
- If improvement is observed, monitor and coordinate additional therapy as needed.
- If improvement is not observed, consider treatment with anti-VEGF.

Case Examples

The following cases illustrate the proposed treatment strategy for various clinical scenarios:

Acute. A 46-year-old white male presented with an acute-onset dark spot in his vision OS for 10 days. The patient reported no recent increase in stress and no use of any steroid medications. OCT imaging was consistent with an acute case of CSCR, with an entering central foveal thickness (CFT) of 420µm. His baseline visual acuity was 20/25.

Due to the acute nature of the condition, the patient was monitored without treatment. The one-month follow-up showed significant improvement of CFT. The patient was lost to follow-up at this point but presented one year later with complete resolution of SRF and visual acuity of 20/20 (*Figure 5*).

Chronic. A 53-year-old white female presented with a history of chronic CSCR for two years. Her baseline visual acuity was 20/50. Her previous treatment included intravitreal bevacizumab injections and oral acetazolamide without improvement. She was using Flonase (fluticasone, GlaxoSmithKline) daily. At presentation, her CFT was 342µm.

With a case history positive for steroid use with Flonase, it was recommended that the patient discontinue the medication immediately. The condition was deemed to be chronic based on the patient's history and OCT findings. Due to the chronicity of the CSCR, treatment with eplerenone 25mg daily was initiated following a review of risks, benefits and alternatives.

The patient's CFT at one month was 298µm, and at two months follow-up, it was 190µm with complete resolution of SRF (*Figure 6*). Upon resolution of SRF, treatment with eplerenone was discontinued. Her final visual acuity was 20/30.

Unknown. A 46-year-old African American female presented with a history of CSCR. She reported symptoms that had been present for four months. Her medical history was positive for high blood pressure, asthma, anemia and kidney disease with a prior kidney transplant. Her medications included amlodipine 5mg BID, calcium carbonate 500mg BID, clonidine 0.1mg TID, aspirin 81mg daily, docusate 100mg BID, dapsone 100mg daily, fluconazole 100mg daily, hydralazine 25mg TID, furosemide 40mg daily, two tablets of magnesium oxide 400mg QID, metoprolol tartrate 25mg BID, mycophenolate sodium 180mg TID, pantoprazole

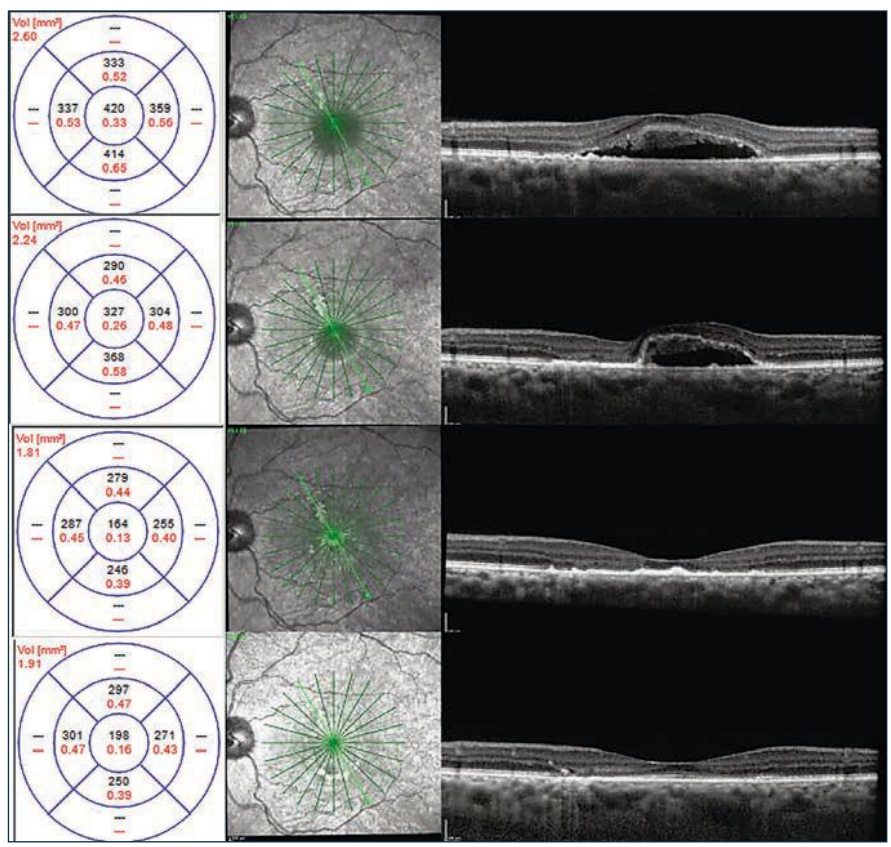

Fig. 9. Chronic CSCR with shaggy photoreceptors at time of laser therapy (top), one month (top middle), two months (bottom middle) and six months (bottom) after focal laser.

40mg daily, prednisone 5mg daily, montelukast 10mg daily and tacrolimus 1mg six times daily.

OCT and FAF imaging were both suggestive of chronic CSCR (*Figure 7*). While the patient's medical history was positive for steroid use, her options for discontinuation were limited by her prior kidney transplant. Since her condition was chronic, therapy was initiated with oral spironolactone 50mg daily. Due to her extensive medical history, the patient's nephrologist was consulted prior to initiation of treatment. There was no improvement with spironolactone therapy; therefore, treatment was changed to eplerenone 25mg daily, also without success.

FA and ICGA were performed. Both revealed a focal, non-central area of late leakage (*Figure 8*). Focal laser was applied to this area. There was significant improvement of serous fluid one month after and complete resolution two months after, with continued improvement in outer retinal anatomy six months post-treatment (*Figure 9*).

Takeaways

While the management of patients with CSCR remains up to each clinician's discretion, the proposed treatment protocol takes into account the risk vs. benefit of treatment as well as the currently understood effectiveness of proposed therapies.

While no therapy is without risk, the possibility of improved outcomes in CSCR patients with readily available oral medications makes treatment with MRA a good first-line option in cases of chronic disease persisting longer than four months. Those who do not respond well to MRAs should be considered for additional therapies such as angiographically guided PDT or focal laser, and possibly intravitreal anti-VEGF. Observation of acute cases initially is recommended. ■

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STROKE OF THE EYE: ARE YOU PREPARED?

When retinal arterial occlusion strikes, you and the patient have only a few hours to act if you want the best odds of preserving vision.

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There are quite a few eye conditions that cause discomfort and pain, but some require immediate attention and prompt action not just from eyecare providers, but medical professionals at emergency rooms.

Retinal arterial occlusion (RAO) is one of those conditions and should be considered a true ocular emergency. This is because the retinal ischemia that is produced creates immediate and permanent cell damage—even when timely interventions are properly dispensed. The condition compromises tissues so quickly there is little likelihood of even modest functional improvements. Today, treatment extends beyond heroic ocular measures, recognizing that emergent medical interventions are required to curtail associated systemic comorbidities.

Acute retinal arterial ischemia can be caused by any process that interrupts the blood flow within the retinal arterial blood supply.[1-3] Embolic events are just one mechanism; vascular compression (mass effect and acute IOP rise), arteritic and vasospastic pathologies also have the ability to interrupt arterial circulation, resulting in ischemia and permanent tissue damage.

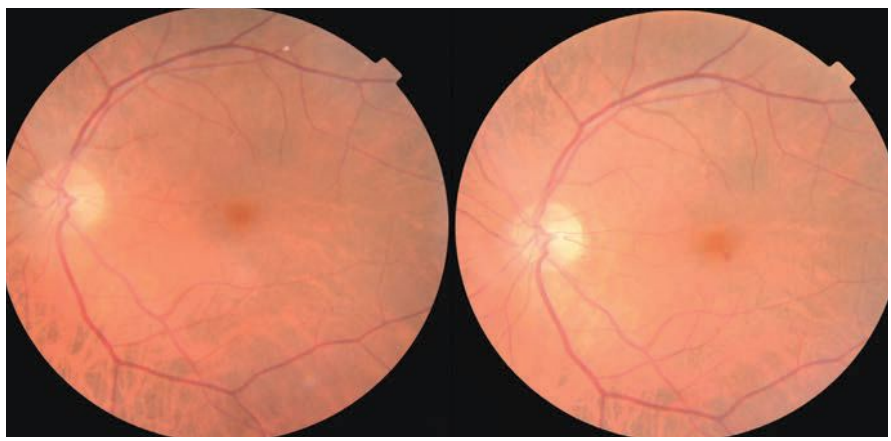

Fig. 1. The photo on the left shows a Hollenhorst cholesterol plaque within the superior-temporal vascular arcade. At follow-up three months later, the plaque is not present.

These events can be transient or permanent and may involve the ophthalmic artery, central retinal artery or a branch retinal artery.[1-3]

Transient Monocular Vision Loss

Episodes of visual compromise that are reversible and last less than 24 hours are termed transient vision loss (TVL); these can be binocular or monocular.[4,5] Transient monocular vision loss (TMVL) may be permanent or reversible and has an incidence of approximately 14 per 100,000 people per year.[2] Patients generally report that episodes of TMVL last 20 to 30 minutes and describe it as a curtain of

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darkness occluding the affected eye.[4,5] Specifically, TMVL is caused by occlusive arterial pathology anterior to the chiasm, at the level of the optic nerve or retina.[4,5]

The underlying cause of an episode is uncovered thorough history—which serves to verify timing, pattern, provoking factors and associated symptoms—laboratory testing and imaging.[1-3] The most common sources of emboli consist of cholesterol, platelet-fibrin material and calcium emanating from the heart, aorta or internal carotid artery.[1-3]

In instances of binocular TVL, lesions are localized to the optic chiasm or retro-chiasmal visual pathway and may be the result of disease processes that involve the vasculature of both optic nerves.[4,5]

Sources of RAO

Non-embolic events producing TMVL are accompanied by unique patterns of signs and symptoms, including retinal migraines, which commonly last up to 20 minutes and may recur several times a day. These attacks include reversible visual phenomena such as scintillating scotoma and are frequently accompanied by headaches.[4,5] Retinal vasospasm can produce a constellation of symptoms similar to those of ocular migraines without the associated headache. A relative afferent pupillary defect (RAPD) may be present during the episode, but can be resolved as perfusion is restored so long as retinal tissues are not permanently damaged.[4,5]

Giant cell arteritis (GCA) has clinical features that include headache, jaw claudication, scalp tenderness, fever, polymyalgia rheumatica and TMVL.[4,5] Episodes of vision loss are of short duration—under five minutes—may be exacerbated by postural changes, can recur over a short period of time and have associated photopsia.[4,5]

Ocular ischemic syndrome (OIS), which results from carotid artery disease, presents with ophthalmic signs that include vascular congestion of the conjunctiva, rubeosis irides, mid-peripheral retinal hemorrhages and non-tortuous retinal veins.[4,5] TMVL events associated with OIS typically have a gradual onset that lasts from seconds to minutes.[4,5]

Systemic hypotension and reduced cardiac output may result in hypoperfusion of the eye. In such cases, the TVL event is binocular and accompanied by lightheadedness and confusion.[4,5] Other, less common diagnoses include the hypercoagulable states and orbitopathies.[4,5]

In contrast to TMVL, long-term or permanent vision loss associated with acute retinal arterial ischemia is the result of longer lasting partial or complete occlusion of the retinal arterial system.[3,6-10] Ophthalmic artery occlusion, central retinal artery occlusion (CRAO) and branch retinal artery occlusion (BRAO) often lead to permanent visual dysfunction depending on the region of the retina involved, and may produce reduced visual acuity and noticeable visual field deficits.

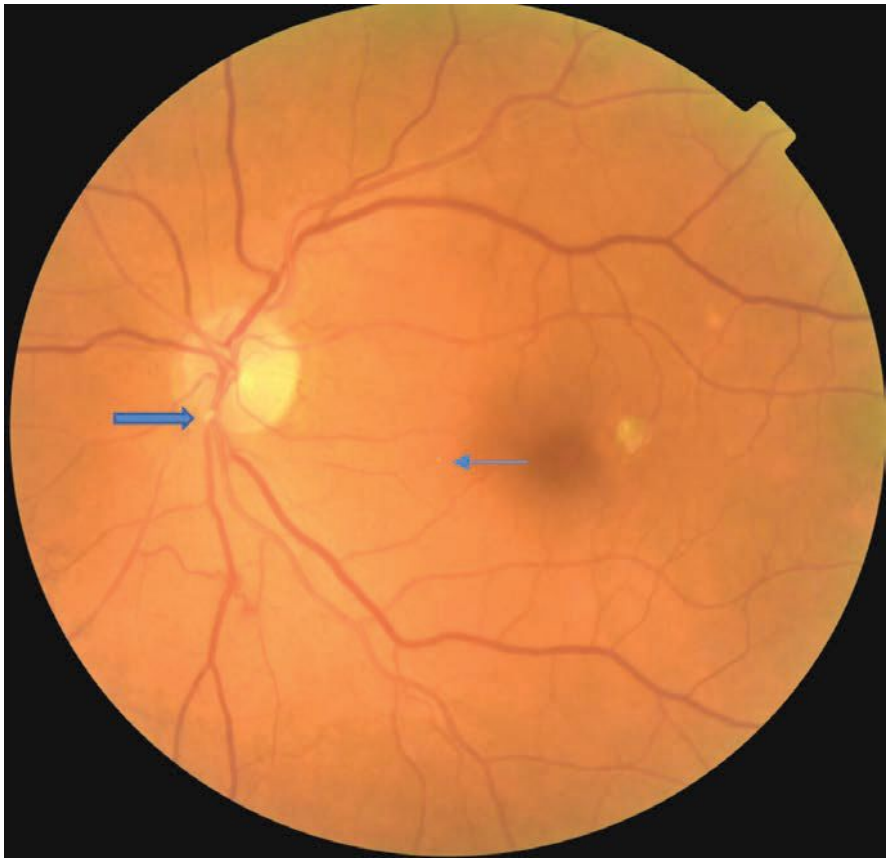
The most common cause of acute retinal arterial ischemia is embolism (non-arteritic) originating from atheromatous stenosis of the ipsilateral internal carotid artery (ICA).[3,6-10] Extracranial ICA stenosis of greater than 70% has been seen in up to 40% of patients diagnosed with CRAO.[3,6-10] Emboli also travel from distant sources such as the heart and the aortic arch, and may consist of cholesterol, calcium or platelet fibrin aggregates.[3,6-10]

There are less common sources of emboli that may result in the occlusion of the retinal arterial system (*Table 1*). For instance, a fat embolus can occur when a fractured long bone releases droplets of fat into the bloodstream. These commonly travel to the lungs or the brain, resulting in pulmonary emboli or stroke.[6-10] Septic emboli consist of bacteria or bacteria-containing tissues entering the bloodstream from a site of infection, such as an infected heart valve (infectious endocarditis).[6-10] Air emboli are formed when small amounts of air enter the blood circulation during a medical procedure such as surgery or catheterization, or during injectable substance abuse.[6-10]

Amniotic fluid embolus—while rare—is the result of amniotic fluid entering a mother's circulation via the placenta during childbirth.[6-10] Also rare is the formation of a foreign body embolus. These emboli form as the result of a medical procedure (iatrogenic) or recreational drug abuse, such as cocaine use or additives like talc. Iatrogenic causes may also be the result of an injection during a dental procedure or from a facial cosmetic

TABLE 1. EMBOLI AND THEIR COMMON ORIGINS

Type of Embolus	Origin of Embolus
Cholesterol	Extracranial carotid artery
Calcium	Valve of heart
Platelet fibrin aggregates	Aortic arch, artery system
Fat	Fat droplet from a fractured long bone
Septic (bacteria containing tissue)	Infected tissue (*e.g.*, endocarditis)
Air	Air enters circulation via needle
Amniotic fluid	Enters mother's circulation via placenta during birth
Foreign body	Injection of material into blood circulation, ingestion of foreign substances (*e.g.*, cocaine, talc)
Paradoxical (originates from a vein)	Patent foramen ovale



A fibrinogen platelet embolus is seen at the location of the large blue arrow. A small Hollenhorst plaque is seen at the small blue arrow.

procedure where a drug or filler material is injected into a vessel.[11] An embolus that originates in a vein and eventually causes the occlusion of an artery is a "paradoxical" embolus. These emboli require a pathway from the right side of the heart to the left, as in the case of a patent foramen ovale.[3,6-10]

Giant cell arteritis is the most common arteritic cause of retinal arterial occlusion and should be included in every vascular workup, even when suspicion is minimal.[3,6-10] The doctor must be aware of the clinical features of GCA, as mentioned above, when investigating the etiology of sudden vision loss and its association with an arterial occlusive event.[3,6-10] Other non-embolic causes of retinal arterial occlusion may include hematologic abnormalities resulting from sickle cell hemoglobinopathies, leukemia and systemic non-Hodgkin's lymphoma.[3, 6-10]

Rapid changes in intraocular pressure have the potential to result in occlusion of the retinal arterial system by way of compression. Acutely elevated intraocular pressure in cases of glaucoma, ocular compression as a result of pressure on the eye due to positioning for an extended period of time in supine position (as in the case of spinal surgery), orbital lymphoma, retrobulbar injection and peribulbar anesthesia have all been associated with retinal arterial occlusion.[1-10]

Epidemiology

In the United States, CRAO has an incidence of 1.9 per 100,000 people.[1-12] This incidence increases to 10.1 per 100,000 for those age 80 years and older.[12] Retinal and ophthalmic artery occlusive events are marked by acute, painless monocular visual acuity and/or visual field loss, and it is only when these symptoms persist that patients make their way to either the emergency department or eye doctor.

Men reportedly have a higher incidence than women, and the average age of presentation is approximately 65.[3,10-13] The patient's medical history will frequently include cardiovascular disease, diabetes, hypercholesterolemia and/or a history of cigarette smoking.[14,15] These systemic diseases are also confounding factors in the presence of an acute ischemic stroke.[9,16,17]

Signs and Symptoms

A clinical examination will confirm a precipitous reduction in visual acuity (20/400 or worse), visual field loss and an afferent pupillary defect. Color vision impairment directly correlates with the decline of visual acuity and involvement of the macular region. General medical procedures that may be performed in-office include measurements of blood pressure, pulse, pulse oximetry and assessment for carotid bruit.

Posterior segment findings are highly variable, depending on the duration of the event and the time from onset of symptoms to examination. The alteration of retinal blood vessels may include "cattle trucking" (segmentation of the blood column within the vessel) and retinal arterial attenuation.[1-3,6] Other findings that may be observed include pallid edema of the optic disc, the presence of an embolus, pale and swollen retinal tissue and a cherry red-spot involving the macula.[1-3,6]

Pathophysiology

The posterior segment examination may reveal the presence of an embolus within an artery of the retina. In many cases, the embolus is not able to be visualized. The presentation of an embolus is helpful because it provides information regarding its consistency and origin.

A plaque that is highly reflective and white in color most likely consists of cholesterol originating from the ipsilateral carotid artery (Hollenhorst

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TABLE 2. LABORATORY STUDIES AND MEDICAL INDICATIONS

Laboratory Test	Indication for Test
Complete blood count (CBC)	Overall health, anemia, infection, leukemia
Basic metabolic panel	Measures glucose, electrolyte, kidney function
Prothrombin time/Partial thromboplastin time	Bleeding disorder, excessive clotting disorder
International normalized ration (INR)	Time to prevent formation of blood clot
Lipid panel	Measure of total cholesterol levels
Hemoglobin A1c (HA1c)	Average level of blood sugars over 60-90 days
Erythrocyte sedimentation rate (ESR)	Degree of inflammation present in the body
C-reactive protein	Measure a liver protein that responds to inflammation

plaque). Plaques often originate from the bifurcation or within the internal carotid artery.[3,6-9] Emboli consisting of calcific debris or platelet fibrin aggregates present as elongated dull grey opacities. Calcium emboli commonly originate from a heart valve. Platelet fibrin aggregates are frequently a byproduct of artery damage caused by atherosclerosis and can involve the aortic arch or the carotid or internal carotid artery.[3,6-9]

Emboli have the greatest likelihood of occluding the central retinal artery where its lumen is narrowest, at the point where it pierces the dura of the optic nerve, and is less likely just posterior to the lamina cribrosa.[3,10] As both of these anatomical sites are behind the globe, the vast majority of emboli that cause CRAO are not able to be visualized during dilated funduscopy.[3,10]

The phenomenon known as "migration of retinal emboli" can occur in both CRAO and BRAO, and may account for why emboli are not found at the time of the acute examination. In this case, by the time the patient presents to the clinic, the embolus has migrated from its original position within a retinal artery and may no longer be visible.[3,10]

Interestingly, calcific emboli are rough in texture and tend to get impacted within the vessel walls. Cholesterol and platelet-fibrin emboli have a less rough texture and tend to migrate easily.[3,10] Therefore, in a case of monocular acute painless vision loss, a conclusion that "this is not a retinal artery occlusion" may not be made just because no retinal embolus was observed.[3,10]

Retinal Survival Time

Occlusion of any retinal artery ultimately results in the infarction of the retinal ganglion cells found within their vascular range.[18] Research suggests that the ganglion cell layer of the retina can survive interrupted blood flow if circulation is restored in up to 97 minutes of the occlusion.[18] Beyond the 97-minute mark and up to 240 minutes, more extensive and irreversible damage occurs. Occlusion beyond the 240 minutes results in catastrophic, irreversible retinal damage.[18]

Hayrah's classic research of CRAO included experimental occlusion of the central retinal artery of rhesus monkeys. These experiments provided us with the 97 to 240 minute boundaries that are recited today.[18] Recently, retinal survival times have been challenged based upon a review of the original research designs.[10] It has been proposed that clamping the central retinal artery, as was done in the original work, would not have resulted in complete obstruction of blood to the retina. Further, it would not have eliminated the collateral circulation to the retina. In this instance, the created arterial occlusion would be incomplete. While collateral circulation would not permit complete perfusion of the retina, it certainly might have lengthened the time necessary to achieve inner retinal ischemia.[10,18] This has led to thinking that retinal survival time is actually much less.

Tissue of the brain and the retina are thought to be the most energy consuming structures of the body. Comparative studies of brain and retina reveal that retinal oxygen consumption per gram is greater than that of the brain, making the retina at least as vulnerable to oxygen deprivation.[19-23] Hence, opponents of the original models postulate that during complete central retinal artery occlusion, irreversible retinal ganglion cell death begins after just 12 to 15 minutes.[19-23]

Not all CRAOs or BRAOs are permanent and/or complete, as the involved vessels may only be partially occluded or the obstruction transient. These factors, along with the presence of a cilioretinal artery (a retinal vessel deriving its blood supply from the uninvolved choroidal circulation, present in 15% to 30% of eyes) allow for a prognosis that predicts spared sectors and potential for recovery.[3,6,24] The variability in both visual acuity and spared field will correlate with the extent of the distribution of the anatomical variation of the cilioretinal artery.[3,6]

Ocular Management

The critical period in any case of retinal artery occlusion is from the onset of symptoms to when the patient presents for ophthalmic care. The goal of acute ocular treatment

is to reverse retinal ischemia by restoring perfusion before permanent cell death occurs.[1-3] Unfortunately, acute treatment remains controversial, as there is no proven standard of care.

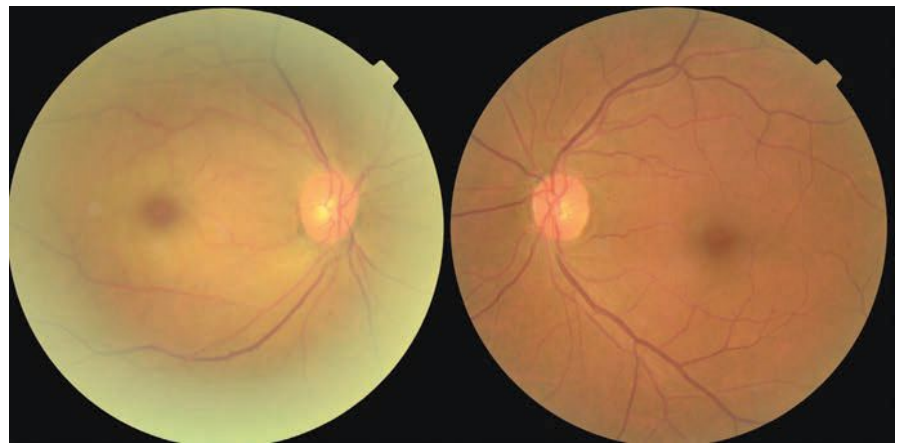
The intervention of IOP lowering (topical timolol, iopidine, oral acetazolamide and IV mannitol, anterior chamber paracentesis) is directed toward decreasing the resistance to perfusion into the eye. By making it easier for blood to enter the eye, perhaps an increased flow might destabilize and move the embolus downstream or provide adequate nutrition to avert cell death.

Inhaling carbon dioxide or taking other oral or IV vasodilating agents are efforts directed toward dilation of the blood vessels of the retina. As retinal arterioles become pharmacologically enlarged, the involved arteriole might loosen its grip on an embolus, permitting it to be jettisoned downstream, restoring perfusion.

Direct pressure to the globe ("mashing" the eye or digital ocular massage) in combination with the first two interventions is intended to create a back pressure for the blood that is trying to enter the eye, such that when the direct pressure is released, an increase of blood flow might impact the embolus with such force that it dislodges the embolus, restoring retinal perfusion.[3-6,8]

Another treatment being explored is hyperbaric oxygen. Here, by supersaturating the red blood cells and "collateral retinal systems" (the choroidal circulation), unused oxygen might diffuse forward into the starving retina to emergently nourish it and postpone cell death.[25]

Unfortunately, there is little if any evidence-based data that supports any of the above therapies as a way to reverse the often catastrophic outcome.[3,6,8,9]



This shows the early presentation of a CRAO of the left eye with pale edema of the retina, a cherry red spot at the time of presentation of a central retinal artery occlusion. Artery attenuation is also noted.

Systemic Management

A March 2021 statement from the American Heart Association formally recognized an ischemic stroke as an "episode of neurological dysfunction caused by focal cerebral, spinal or retinal infraction."[9] The common pathophysiology shared by stroke patients and retinal artery occlusion patients has prompted experts to categorize RAO as a "stroke of the eye." This philosophy mandates concomitant and rapid triage of CRAO patients with an aggressive systemic workup, with the goal of reducing cardiac and associated central nervous system (CNS) comorbidities.[9,25,26] Specifically, intravenous tissue plasminogen activator (tPA) and intra-arterial thrombolysis (IAT) are commonly used in the management of occlusive ischemic cerebral infarcts and are being more closely investigated as a more standardized treatment of CRAO.[9,26,27]

Intravenous fibrinolysis most commonly uses the infusion of alteplase (which acts as a tPA) and is proven to be most efficacious when administered within 4.5 hours of the onset of symptoms.[26,27] A meta-analysis of observational studies found that patients with acute CRAO treated with this modality had a 50% rate of clinical recovery (visual acuity of 20/100 when initial acuity was 20/200) when treated within 4.5 hours of onset of symptoms.[28] Unfortunately, treatment must be used selectively, as symptomatic intracranial hemorrhage can occur. To reduce this risk, confounding factors such as active bleeding, recent stroke or hemorrhage or use of anticoagulation therapy should be identified.[9,28]

IAT involves the introduction of the thrombolytic agent (tPA) directly into the ophthalmic circulation via microcatheterization.[29] The advantage is that therapy is delivered to the thrombus, limiting systemic circulation and reducing the risk of intracranial and systemic hemorrhage.[29] Inherent risks include possible arterial dissection, catheter-induced spasm and dislodgement with distal embolization of an atheromatous plaque withing the ophthalmic circulation.[29]

Prompt triage and referral to an emergency department, with the goal of admission to a stroke floor for a comprehensive medical assessment by experts who understand this syndrome, is now the standard of care.[1,2,9,27] Laboratory studies should include a complete blood count, basic metabolic panel, prothrombin time/partial thromboplastin time, international normalized ratio, lipid panel, hemoglobin A1c, erythrocyte sedimentation rate and C-reactive protein (*Table 2*). These studies assess the overall wellness of the patients and target the most common vascular risk factors (HTN, DM, hyperlipidemia) that may lead to other vascular occlusive events or myocardial infarction.[1-3]

Diagnostic imaging should be ordered in all cases of retinal vascular occlusion to search for the potential origin of an embolus. If vascular narrowing or blockage exists, it must be identified to eliminate the potential for subsequent occlusive events in the brain or heart.[7-9,14] Imaging studies include computed tomography (CT), magnetic resonance imaging (MRI), CT angiogram, MR angiogram, carotid doppler, transthoracic echocardiogram and transesophageal echocardiogram. Ambulatory cardiac rhythm monitoring is frequently performed to diagnose atrial fibrillation.[7-9,14]

Treatment goals on the stroke floor include tight management of vascular risk factors that have a common impact on CRAO, stroke and carotid artery disease.[7,14] Control of hypertension (the leading risk factor for retinal ischemia), hyperlipidemia (the second most common risk factor associated), diabetes and obstructive sleep apnea are of most importance. Lifestyle changes and education include smoking cessation, exercise and dietary restrictions to reduce obesity and BMI.[7-9,14]

Prognosis

Prompt recognition of the signs and symptoms associated with acute retinal ischemia secondary to retinal artery occlusion play a critical role in the preservation of visual function and prevention of stroke and cardiovascular events. It is has been reported that 25% of patients presenting with CRAO have had a "silent" stroke that is visible upon MRI, with no patient awareness or symptoms.[1,2] The risk of an ischemic neurologic event occurring within months following a CRAO is 2.7 times higher when compared to control subjects.[9]

Studies have documented a range of 7.4% to 24.2% of patients having a stroke in the first four years following a CRAO.[30-33] The incidence of acuter coronary syndrome in patients with CRAO 70 years of age and higher was 2.48 times higher versus aged matched controls.[34] The lifetime of CRAO patients who do not make lifestyle changes has been estimated to be reduced by 10 years vs. healthy controls.[6-9]

Summary

Historically, even the most heroic ophthalmic treatments rarely result in the restoration of functional vision. Acknowledging the association between cardiac and central nervous system conditions makes retinal ischemic events a true medical emergency and the need to refer cases of retinal ischemia to the emergency department must not be understated. Eyecare providers should have a plan that incorporates the primary care provider, the emergency department and a stroke center into the global care of the patient with CRAO. ■

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BE A RETINA REFERRAL ROCK STAR

ODs can—and should—take the lead on screening and monitoring routine cases, only sending to specialists the patients that truly need advanced care.

BY CATLIN NALLEY
CONTRIBUTING WRITER

Fostering viable comanagement relationships requires time and effort from all parties. There must be a desire to collaborate as well as mutual trust and respect. As the primary providers of eye care, ODs are perfectly positioned to take a leadership role when working with ophthalmologic subspecialists and other medical specialists.

Effective comanagement is beneficial for a number of reasons. It gives patients easier access to care than waiting for an opening with an ophthalmologist and lessens travel and cost burdens, to name a few. From the standpoint of the surgical provider, sharing post-op responsibilities allows them to allocate more time to surgical care. For ODs, it is a way to enhance your practice and professional development.

ODs often have strong relationships with their patients developed over years or even decades of care. They not only understand their visual demands and needs, but also their personalities and what approach will work best. The specialist, on the other hand, does not have this baseline knowledge.

"They don't know whether the patient who is noncompliant with treatment needs a little bit of tough love, or perhaps they are scared and need more of a nurturing environment," notes Jessica Haynes, OD, of the Charles Retina Institute in Memphis. "Then on the other side, the patient walking into a specialist's office has never seen this doctor before. They may not know what to expect, and when certain treatments are recommended, they may be distrustful of that advice," she notes.

The procedures of a retina practice—anti-VEGF injections, panretinal photocoagulation and the array of complex surgeries—all require strong buy-in from the patient for things to go smoothly. Having an existing connection with a trusted doctor can help immensely.

Optometrists must use the relationships they build with patients to walk with them through any journey of referral and treatment, Dr. Haynes emphasizes. "By building good comanagement strategies, patients will have better outcomes. And what more can a physician strive for than better care and outcomes for their patients?"

Building Connections

Successful comanagement requires strong relationships and open lines of communication. This begins by building connections with ophthalmologists and other specialists in your area.

"First, we have to realize that these relationships and trust have to work both ways. So, at times the ophthalmologists are the ones reaching out to develop referral relationships," says Mohammad Rafieetary, OD, also of the Charles Retina Institute in Memphis. "Often, ODs learn who to refer to by word of mouth from other area colleagues, either through personal connections or local society meetings."

If ophthalmologists host seminars or social events, Dr. Rafieetary encourages ODs to attend. This is an opportunity to get to know doctors and staff on both a personal and professional level.

Being proactive is key. Dr. Rafieetary also recommends optometrists take the initiative and invite specialists to events. "This is not only beneficial to establish personal relationships but create referrals for services an ophthalmologist does not offer in their practice," he notes. "It always works better if both sides of the

referral relationship know each other on a personal level."

Spending time shadowing local providers is another way to build connections as well as learn more about their practice and how they interact with patients. "This type of approach is very likely to open up the lines of communication," notes Dr. Haynes. "You may even get the doctor's personal cell phone number, so the next time you have a patient who needs to be seen emergently, you don't have to rely on their receptionist getting your patient in. You can go directly to the source."

Inviting specialists into the optometric community can lay the foundation for comanagement. For instance, many state and local optometry chapters ask ophthalmologists to speak at their meetings. "A good turnout and discussion shows ophthalmologists our bond as a community and builds respect," says Dr. Haynes. "So, if you aren't active in your state and local chapters, that's another good place to start."

It's also important to note that true comanagement depends on a level of respect that goes beyond education and mutual referrals. "If a retina specialist is truly respectful of the optometric level of care, they will support optometry not just in patient care but also in the political arena," explains James Fanelli, OD, of Wilmington, NC. "A retinologist who opposes scope expansion by optometry but is happy to receive your referrals is not really a respectful referral partner."

Optimizing Retina Care

Unlike cataract and refractive surgery, there is no standard or protocol for retina comanagement; however, it is still an important aspect of successful patient care.

Effective comanagement begins with the basics, notes Dr. Haynes. This includes dilating and properly examining patients as well as a comprehensive understanding of diagnostic imaging in retina care, such as OCT, and staying current on the latest treatment options.

The conversations you have with your patients before they even see a retina specialist are a crucial component of care and comanagement. ODs must feel confident in their ability to educate patients and help them through this process. And, depending on the patient, it may take multiple discussions to help them truly understand their condition and the need for a referral as well as the role of their optometrist.

As with other forms of comanagement, the optometrist should take the lead to ensure they maintain their position as the patient's primary eye care provider. "If you believe in the importance of continuity of care, stay in control of your patient's care," emphasizes Dr. Rafieetary. "Schedule a follow-up appointment for your patient regardless of why you are referring them."

Even if the condition does not require a set follow-up, scheduling another appointment perhaps three months out helps you maintain your connection with that patient. The appointment can always be adjusted if the specialist asks you to see them sooner, suggests Dr. Rafieetary.

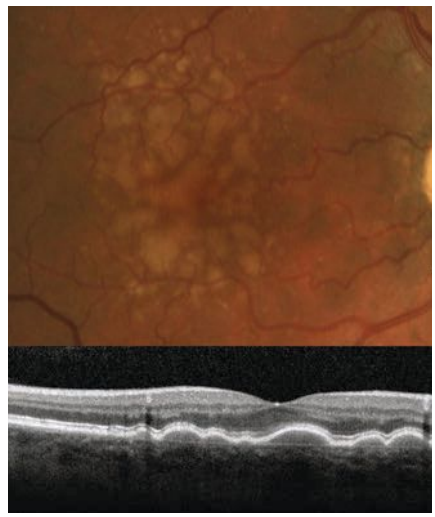

Photo: Jessica Haynes, OD

Monitoring dry AMD patients for risk factors for conversion to the wet form (such as the large, soft drusen shown here) is an ideal role for optometric skills in the continuum of retina care.

Taking the lead also means clearly defining your role with the retina specialist. For instance, a common reason for retina comanagement is neovascular AMD. Today's standard of care is intravitreal anti-VEGF injections.

"This is a recipe for the patient being lost to an ophthalmology retina practice," notes Dr. Fanelli. "However, ODs can take this as an opportunity to maintain control and even determine the injection schedule."

While you don't know if the patient will need six monthly injections for six months or monthly injections for the rest of their life, you can schedule the patients for a series of injections, explains Dr. Fanelli. "The OD then is seeing the patient between injections, evaluating the effect of the injections using their OCT, and forwarding that info to the retina specialist prior to the next visit. By doing this, the OD stays actively involved with the management and acre of the AMD patient."

Optometrists must stay current on the latest advancements in anti-VEGF therapies as well as how to manage and follow patients long-term. This

REFER, BUT DON'T RELINQUISH, YOUR PATIENTS

- Don't tell patients that you are referring them because you don't have the right equipment, know-how or legal privileges. It undermines your position as a provider.
- Never tell the patient you will see them "when the specialist is done with you." Some of these patients will require ongoing care from a specialist. Such phrasing also takes you out of the chain of decision-making. Instead, communicate that you will continue to see them whenever appropriate, even if it's just for routine eye exams and vision correction plus a "check in" on their progress with the specialist's regimen during such a visit.
- Set a follow-up appointment for patients to return to your office regardless of referral. This follow-up can always be changed if needed; however, you have a better safety net to make sure the patient is receiving appropriate treatment and more importantly is not lost to follow-up.
- Make sure to follow through on the referral and all recommended follow-up with the retina specialist.

includes recognizing potential adverse events and any other safety concerns. ODs need to be aware of what the injected eye should look like 24 hours after the injection, as well as what signs and symptoms can develop shortly thereafter that indicate, for example, early endophthalmitis.

Externally, injection sites should look relatively benign, save for the occasional subconjunctival hemorrhage, notes Dr. Fanelli. Immediately post injection, visual acuity will be reduced, but that should return to pre-injection levels within 24 to 48 hours. Decreased vision and increased discomfort are red flags for complications.

Creating a dynamic where the optometrist takes the lead goes back to finding the right ophthalmologist and fostering a relationship of mutual trust and respect. "Not only must the OD have confidence in their own skills, but the retina specialist must also believe in your abilities," Dr. Fanelli says. "If they do, they will be comfortable following your lead and this also helps them eliminate unnecessary visits to their practice.

"There are retina specialists out there who will work with you at a level where you are the one making the determinations," he emphasizes. "They will see your patient and say, 'Dr. OD will let you know if you need to see me again.' And that, I think, is the perfect, two-way referral relationship."

Challenges and Gaps

Recognizing when and when not to refer to a retina specialist is an important, yet challenging, aspect of optometric practice.

One example is diabetic retinopathy. In these cases, the OD should first determine the severity. And then, no matter the stage, the optometrist must identify if diabetic macular edema (DME) is present, notes Paul Chous, OD, an expert in diabetes and diabetic eye disease from Tacoma, WA. "Retina specialists don't need to see patients with mild, nonproliferative diabetic retinopathy without any diabetic macular edema," he explains, noting that in those cases ODs should monitor patients for progression and counsel on the importance of individually optimized metabolic control, which is far more important in early rather than later-stage diabetic retinal disease.

As the disease progresses and moves beyond moderate severity in nonproliferative cases, the likelihood of patients developing a vision-threatening complication like proliferative diabetic retinopathy or center-involved DME increases dramatically, according to Dr. Chous. Therefore, it is critical that ODs are well-versed in the staging criteria and have the knowledge to monitor and refer these patients appropriately.

For DME, it is recommended that ODs send the patient to a retina specialist; however, Dr. Chous notes, there is a role for the optometrist, especially among patients who require observation and not immediate treatment, such as non–center involved DME or center involved DME with normal visual acuity. To make this determination and diagnose accurately, Dr. Chous says an OD must have access to an OCT.

"I would encourage every optometrist seeing patients with diabetes to have an OCT, but if they don't, working with a nearby optometric colleague who does can be very helpful and spare patients an unnecessary visit to a retina specialist's office," he explains, noting this is also an opportunity to get another opinion on whether or not that patient should be referred to a specialist.

One condition that is often referred unnecessarily to ophthalmology is retinal artery occlusions. These patients, in particular those with branch retinal artery occlusions, do not need retinal surgery, Dr. Fanelli says, noting that they will need an internal medicine or vascular work-up.

In these cases, the OD can and should begin the initial workup by assessing the stroke risk of the patient (medical history, medications, carotid auscultation) and make the determination of whether the patient needs immediate referral to a stroke center or the emergency department for carotid and cerebral imaging, explains Dr. Fanelli. If the artery occlusion appears more to be related to fibrin deposits, then cardiology involvement is necessary.

This is also true for retinal vein occlusion without macular edema. Patients with retinal vein occlusions need evaluation of those etiologies that precipitate occlusion, such as poorly controlled hypertension, atherosclerosis and diabetes, or in patients without retinal vascular evi-

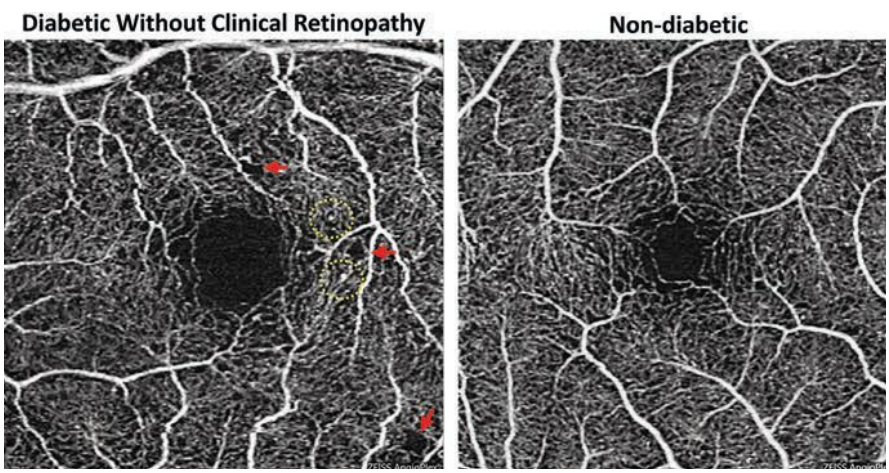

Photo: Carolyn Majcher, OD, Susan Ly Johnson, OD

New technology is elevating the importance of early diagnosis and, with it, the profile of optometrists equipped to do so. These OCT-A images show foveal enlargement and perifoveal capillary remodeling in a diabetic eye without funduscopically visible diabetic retinopathy. Red arrows point to subtle areas of capillary nonperfusion while yellow circles highlight microaneurysms.

REFERRAL LETTER DOS AND DON'TS

A key component of any comanagement relationship is the referral letter. This an important line of communication with the specialist and helps lay the foundation for clinical management.

"Start with the basics. Why am I referring this patient? What am I worried about right now?" suggests Dr. Chous. "Try to list the specific pathology and provide as much detail as possible. ODs should also share the fundamental findings, including best-corrected vision and IOP as well as any other factors that might be responsible for a decline in vision, like media opacity or history of amblyopia."

While you want to be as specific as possible, don't provide a diagnosis if you are unsure. "Call it as you see it," notes Dr. Rafieetary. "Do not make up a diagnosis. 'Retinal detachment' is a frequently mispresented diagnosis, to justify or expedite a referral. Better to describe your findings generally than give a wrong, specific diagnosis."

The referral letter is an opportunity to clearly outline your expectations. Do you simply want the retina specialist to evaluate your patient and send them back if treatment is not indicated? Or is this something you want the specialist to monitor long-term? Let the specialist know if and when you have scheduled a follow-up with your patient.

"Be specific in what you are referring a patient for and provide any necessary background information that may be needed in the form of recent patient records or a short summary," Dr. Haynes advises.

Use this letter as a tool to reiterate the comanagement relationship, suggests Dr. Chous. "Emphasize to the specialist that you want to comanage this patient and look forward to working together."

It's also a good idea to note if specific patients are more likely to be lost-to-follow-up (LTFU) based on known risk factors like poverty, presence of other vascular comorbidities, and educational and cognitive status, so that both doctors can work to mitigate this, Dr. Chous notes. "Studies suggest that more than 25% of patients referred to retina specialists are LTFU, resulting in worse outcomes, so it's a big deal."

dence of the above, perhaps evaluation of coagulability, notes Dr. Fanelli.

"If we're going to market ourselves as frontline eye care providers, we can't just cherry-pick the easy cases and manage those," Dr. Fanelli urges. "We must create a culture of, 'It's OK to treat corneal ulcers on the visual axis, it's OK to manage vein occlusion and so on, because I know what I'm doing and I'm comfortable with that.'"

Optometrists must also step up and be more forthcoming about discussing prevention. "We are the primary eye care providers and it behooves all of us in optometry to talk to our patients about preventing retinal disease," says Dr. Chous. "We have the chance to educate our patients on the benefits of healthy lifestyle choices and encourage them to make a change."

"From a comanaging perspective, I inform the retina specialist that I have advised the patient on lifestyle management of the retinal disease, including better diet, exercise, smoking cessation, compliance with sleep therapy and other prudent lifestyle changes," he explains.

Here again the strength of your relationship with longstanding patients may make you uniquely well-positioned to discuss such things. An ophthalmologist with no prior relationship and level of trust with a new patient may have a harder time of it.

Learning and Development

Effective comanagement depends not only on the relationship between providers, but also an optometrist's understanding of retinal disease, varying clinical presentations, diagnostic imaging findings and available treatment options.

"Just because optometrists are not performing retinal surgeries, laser procedures or intravitreal injections for these conditions doesn't give us an out to not understand the diseases and treatment options available to our patients," Dr. Haynes notes. "A greater understanding of retinal disease is going to allow us to make better referrals." Dr. Fanelli concurs. "Not only will it facilitate appropriate referrals, it can eliminate the need for unnecessary referrals," he says.

As diagnostic technology becomes increasingly more able to detect early retinal disease or at least risk factors for development, the momentum in retina care is inevitably shifting toward optometry, where routine screening and monitoring visits are commonplace for many ocular conditions. Dark adaptation testing, for instance, is capable of picking up the earliest signs of AMD, which manifest at a point in the disease course when the optometrist can discuss lifestyle modifications, recommend AREDS vitamins and initiate a monitoring regimen to give patients a better shot at minimizing the disease's effects.

With ongoing education—whether through continuing education, conferences or mentorship—ODs can hone their skills and be more equipped to recognize what requires a referral and what can be handled in their own practice. Additionally, optometrists will be able to better educate patients on their condition and what to expect from a referral visit, suggests Dr. Haynes.

"We can make a difference in the visual outcomes of these patients," she adds. "A patient who comes into a retina clinic with a better baseline understanding of their condition from the get-go is much more likely to be compliant and have a good final outcome."

Optometrists can and should take the lead in comanagement, using their role as primary eye care providers to drive disease management while also building strong relationships with specialists that allow them to practice to the full extent of their scope and abilities. ■

KEY TAKEAWAYS

- Build strong connections with specialists.
- Be confident in the skills of the specialists you are referring to.
- Take the lead when determining a management plan.
- Constantly hone your skills and knowledge.
- Communicate with patients throughout the referral process.

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WHAT TO DO WHEN IT'S NOT AMD

Learn how to identify and diagnose the multitude of other macular dystrophies and degenerations.

BY JESSICA HAYNES, OD
MEMPHIS

Optometrists have become well versed in the presentations of age-related macular degeneration (AMD), given its prevalence. Once that's been firmly entrenched in your clinical skillset, to take it to the next level you'll want to make sure that you're inclusive of the non-AMD macular problems that present in your practice. By doing so, you'll be able to isolate and differentially diagnose these conditions to better counsel and manage your patients.

The list of AMD masqueraders is lengthy and variable, including conditions that are degenerative, infectious, inflammatory, toxic, vascular, traumatic, neoplastic and paraneoplastic. Any condition that affects the retinal pigment epithelium (RPE) and outer retina may lead to drusenoid or lipofuscin deposition and/or pigmentary alteration that can mimic AMD.[1] In this article, we will focus only on the non-AMD dystrophies and degenerations that affect the macula—a list that is already quite diverse and extensive.

What Stresses the Macula?

As it turns out, the lifelong responsibility of converting light energy into electrical potential to initiate the process of sight is a very stressful job. The photoreceptors, RPE and choroid must constantly work in sync to maintain the visual cycle, regenerate photoreceptor outer segments and remove and phagocytize metabolic waste products. Environmental factors such as UV light exposure and tobacco smoke put strain on this delicate balance, as do systemic conditions such as vascular disease. In addition, numerous faulty pathways can disrupt this system through various mechanisms.[2,3]

Phenotypical outcomes of various stressors can present similarly. Different pathways of damage may lead to clinically similar presentations that are difficult to distinguish from each other. Diagnostic imaging such as optical coherence tomography (OCT), fundus autofluorescence (FAF), fluorescein angiography (FA) and OCT angiography (OCT-A) alongside evaluation of retinal function with tools such as electrodiagnostics may help to narrow down a diagnosis. Additional factors such as age of onset, presenting symptoms and family history are also important, as these vary among different conditions.

What is a Dystrophy?

This umbrella term loosely describes various progressive degenerative disorders. The term dystrophy also implies a monogenic or Mendelian inheritance, meaning the condition results from a specific variant of a single gene. Numerous degenerative conditions, such as AMD, are not considered dystrophies as they do not exhibit Mendelian inheritance.

Stargardt's Disease

The most commonly encountered inherited macular dystrophy, Stargardt's, affects one in 8,000 to 10,000 individuals.[4] Stargardt's disease is most commonly inherited in an autosomal recessive fashion primarily by disease-causing variants of the *ABCA4* gene.

This condition typically presents between the ages of 10 and 20, with a resultant visual acuity around 20/200.[5,6] Presentation later in life usually results in better visual acuity outcomes. Patients often present with classic pisiform-shaped, yellow lesions or fleck-like lesions as well as macular atrophy with a "beaten bronze" appearance (*Figure 1*).[6] Presentation of

About the author

Dr. Haynes is a consultative optometrist at the Charles Retina Institute in Germantown, TN, and a consulting faculty member at the Southern College of Optometry in Memphis, TN. She has no financial interests to disclose.

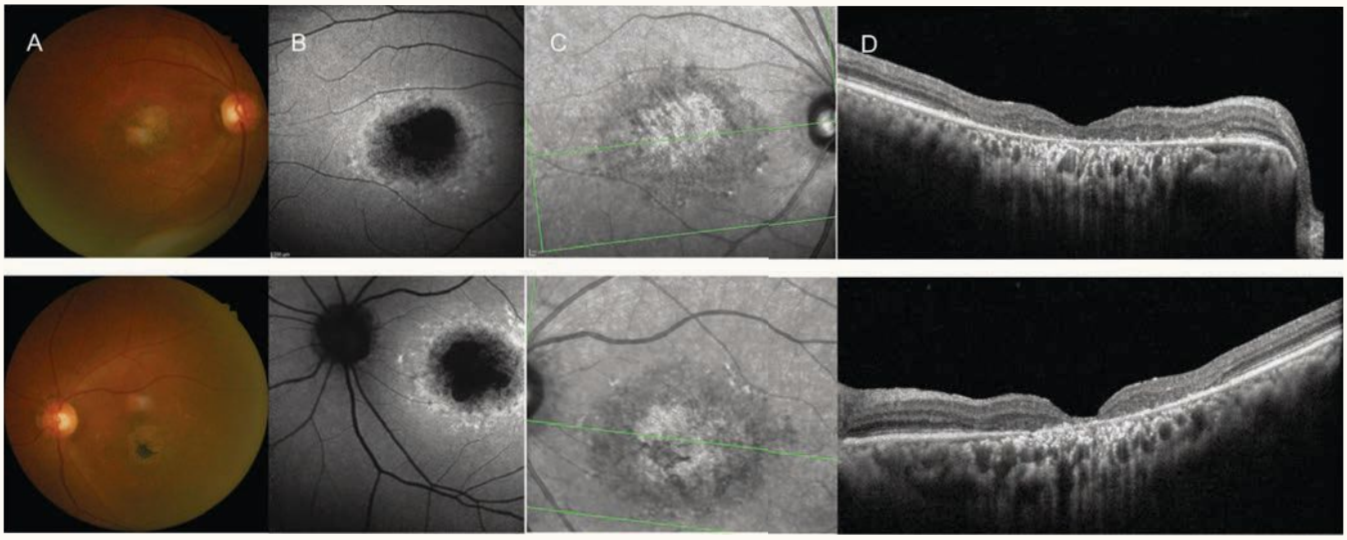

Fig. 1. A 60-year-old white female with longstanding vision loss since her late teens and 20/150 best-corrected vision OD and OS. She presented with: (A) macular atrophy and surrounding yellow fleck lesions, (B) a bull's eye-type pattern, (C) infrared reflectance OU on FAF and (D) outer retinal atrophy on OCT OU. One of her three siblings (a brother) had the same ocular condition. The others had normal vision, and there were no other affected family members, including her parents. The patient's history was consistent with an autosomal recessively inherited condition, and she was clinically diagnosed with Stargardt's disease. Genetic testing was offered, but she declined.

pisiform lesions without evidence of macular atrophy was initially termed fundus flavimaculatus, but is now recognized as a phenotypic variant of Stargardt's disease.[7]

Diagnostic imaging is very useful in identifying and differentiating patients with Stargardt's. Presentation is often subtle at first with visual symptoms being more severe than clinical signs.[8] Care must be taken to identify these patients as to not misdiagnose them or perform unnecessary testing or procedures. OCT may show early thickening of the external limiting membrane. FAF may uncover early alterations and lipofuscin accumulation.[8]

In later stages, OCT imaging may demonstrate drusen-like subretinal, hyper-reflective deposits and varying amounts of photoreceptor atrophy and RPE disruption. FAF often reveals a reticular pattern of hyper-autofluorescent lipofuscin deposition. Areas of RPE atrophy will present as hypo-autofluorescent regions. In addition, a bull's eye pattern of altered autofluorescence may be seen on FAF.[9,10]

The classic sign of Stargardt's is a silent or dark choroid on FA with some citing its presence in up to 80% of patients.[11] This sign is not present in all cases, however, and its absence cannot rule out Stargardt's. While the condition is classified as an autosomal recessive condition, reports of altered visual function and retinal appearance have been described in carriers as well (*Figure 2*).[12,13] Genetic testing should be considered to aid in diagnosis.

Electrodiagnostic testing is variable in patients with this condition. Pattern

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Estimated Time to Complete Activity: 2 hours

Jointly provided by Postgraduate Institute for Medicine (PIM) and Review Education Group

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

- Understand the pathophysiology of macular disorders and dystrophies.
- Differentiate between common macular disorders and dystrophies.
- Use the clinical exam techniques and tools to diagnose their patients.
- Understand how different macular conditions progress as well as their role in monitoring and managing care.

Target Audience: This activity is intended for optometrists engaged in eye care of macular disorders and dystrophies.

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 (ACCME), the Accreditation Council for Pharmacy Education (ACPE) and the American Nurses Credentialing Center (ANCC) 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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ERG and focal or multifocal ERG are typically significantly diminished or abolished, suggestive of macular disease. Some patients have a normal full-field ERG, while others have more widespread disease.[14]

There is currently no cure or treatment for Stargardt's. Patients should be monitored for the rare occurrence of choroidal neovascularization (CNV). In addition, low vision training should be offered to those with reduced visual acuity. Confirming a genetic variant may be useful in light of clinical trials and future treatment options.

Cone and Cone Rod Dystrophies

This heterogenous group of disorders involves progressive, widespread atrophy of cone photoreceptors leading to symptoms of visual acuity loss, decreased color vision and photophobia. In the same spectrum of disease are cone rod dystrophies that also involve rod photoreceptors.

Patients with clinically diagnosed cone dystrophies may eventually develop rod involvement with age leading to symptoms of nyctalopia and visual field loss. These conditions are genetically diverse and share affected genes with other retinal and macular dystrophies such as retinitis pigmentosa, considered a rod cone dystrophy, and Stargardt's. Inheritance may be either autosomal dominant, recessive or x-linked.[14-16]

Clinical appearance, age of onset and visual outcome are variable. Visual acuity reduction typically presents in the first decade of life.[14] Patients may present with pigmentary abnormalities, a bull's eye macular appearance, macular atrophy or normal signs and symptoms.[14-17] Those with cone rod dystrophy may later develop peripheral bone spicules.[14] Varying levels of disc pallor are also reported.[15]

OCT is very useful in identifying photoreceptor atrophy, which may present as loss of the photoreceptor integrity line to more advanced loss of outer retinal tissue including the outer nuclear layer and RPE.[14] FAF is useful in identifying alterations to the RPE that may not be readily visible clinically (*Figure 3*).[14] The earliest finding on ERG is delayed 30Hz flicker implicit time followed by reduced 30Hz amplitude and reduced a-wave and b-wave amplitude with full-field photopic ERG. Those with cone rod dystrophies will later develop scotopic dysfunction.[14]

Phenotype vs. Genotype

The attempt to discuss this group of conditions with strict, discrete categorization is very difficult due to their convoluted and often not entirely understood inheritance patterns alongside the phenotype/genotype conundrum.

Genotype describes what genes are responsible for a particular condition, while *phenotype* outlines how a condition presents clinically. What does it actually look like? Even among family members who share the same genotype, the phenotypical presentation of the same condition may be inconsistent due to variable gene expression.

Conditions were originally grouped primarily based on phenotypical appearance. With more information about these conditions and the genes that cause them, the classification and nomenclature used has evolved. However, this has left us with a bit of a mess to sift through when trying to give a name to a particular presentation.

As genetic testing becomes more readily available and more is known about the genetic variants that cause certain conditions, we are able to arrive at more definitive clinical diagnoses. Access to genetic testing has significantly increased in recent years, becoming more standard of care in the management of inherited conditions. We must use the tools at our disposal along with patient demographics and our current knowledge and access to genetic testing to differentiate these conditions as best we can.

Genetic testing should be considered if available to aid in the diagnosis.

There is currently no cure or treatment for cone or cone rod dystrophies. Patients should be monitored for the rare occurrence of CNV. In addition, low vision training should be considered for those with impaired ability to perform activities associated with daily living. Identifying underlying genetic variants may be beneficial when considering clinical trials and future treatment options.

Pattern Dystrophies

This is an umbrella term that includes adult-onset vitelliform dystrophy (AOVD), butterfly-shaped pattern dystrophy (BSD), reticular dystrophy, multifocal pattern dystrophy simulating Stargardt's and fundus pulverulentus. These conditions were initially categorized and classified based on clinical appearance. Pattern dystrophies in general were once thought to be inherited autosomal dominantly through disease-causing variants of the *PRPH2* gene; however, a wide variety of affected genes and inheritance patterns are now being recognized.

In general, while pattern dystrophies are progressive conditions,

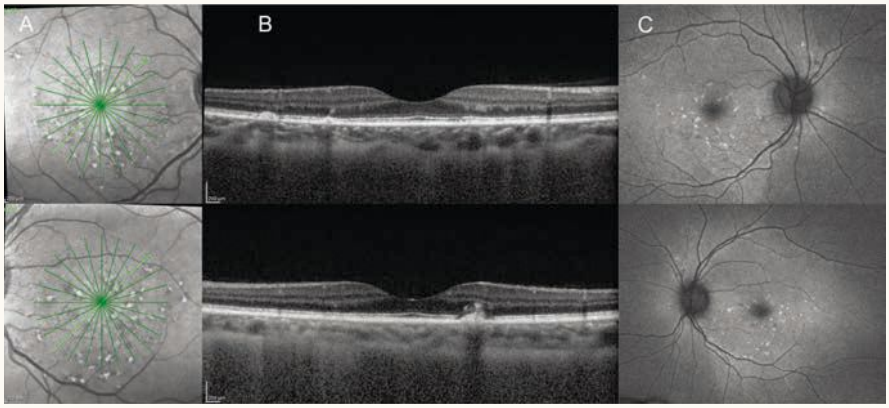

Fig. 2. A 39-year-old Black female with 20/20 BCVA OD and OS presented with yellow, pisciform-shaped subretinal lesions. Images: (A) infrared photography reveals a reticular pattern of hyper-reflectance, (B) OCT shows areas of subretinal drusen-like deposits and RPE disruption and (C) FAF shows hyper-autofluorescence of the pisciform lesions. Genetic testing revealed she is an *ABCA4* pathogenic variant carrier for Stargardt's.

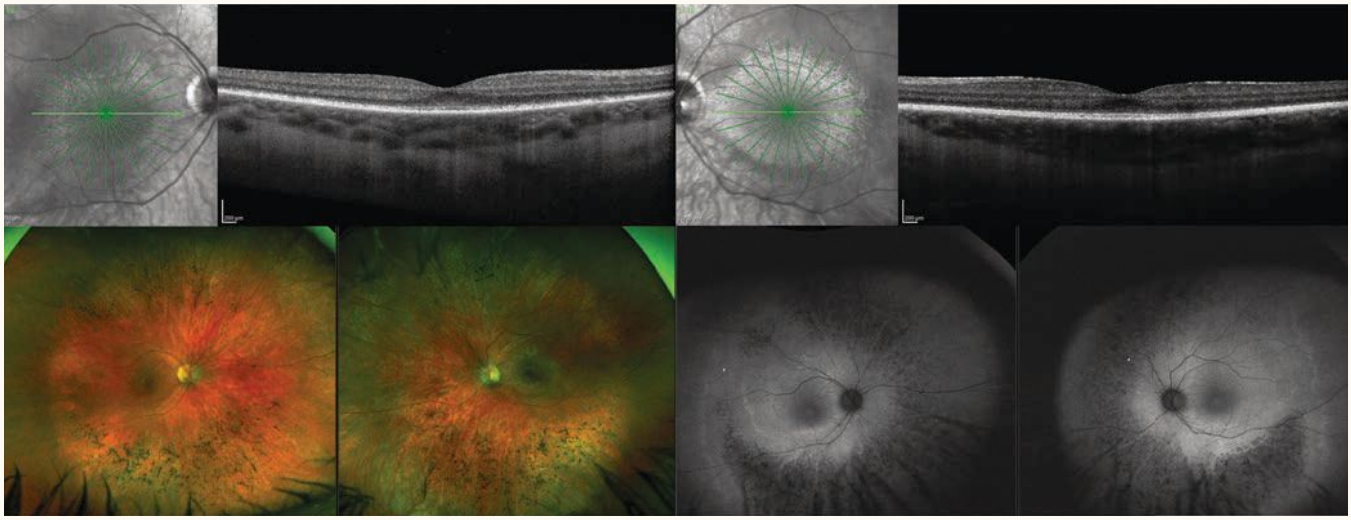

Fig. 3. A 22-year-old white male presented with a homozygous pathogenic variant identified in *TTLL5*, consistent with cone rod dystrophy. OCT images show diffuse disruption of the photoreceptor integrity line. The macula has mild pigmentary alterations while the peripheral retina has a bone spicule-type appearance. FAF shows hypo-autofluorescence in the peripheral retina with hyper-autofluorescence centrally indicating both central (cone) and peripheral (rod) dysfunction.

patients tend to maintain good visual acuity. However, vision loss can occur from formation of macular atrophy, for which there is no treatment, or development of CNV, for which treatment with anti-VEGF is beneficial.[18] Due to the development of visual symptoms later in life, these patients are more easily misdiagnosed with AMD.

AOVD. This commonly encountered pattern dystrophy was first described as an autosomal dominant condition with bilateral, symmetric, circular subretinal lipofuscin deposits called vitelliform lesions (*Figure 4*).[19] Since then, numerous publications have described the clinical, OCT, FAF and electrodiagnostic findings of the disease as well as its inheritance patterns. Confusion does exist in the literature due to the wide variety of names given to this condition as well as a lack of exact criteria needed to make the diagnosis.[20] Another challenge is that vitelliform lesions can occur in a wide variety of outer retinal disease including AMD.

While the *PRPH2* gene is causative in some patients with AOVD, a variety of other genes including *BEST1* have been implicated in the condition as well. Most patients present without a family history, and in many, a responsible gene variant cannot be identified.[20] This leads to the consideration that in some individuals AOVD may be more of a degenerative condition than a true dystrophy. Diagnosis is typically made in the sixth to eighth decade of life and is based on the clinical finding of bilateral, central vitelliform lesions.[20] Variable amounts of additional RPE disruption and drusen deposition including reticular pseudodrusen have also been described.[21]

OCT is a useful diagnostic tool in this case. Vitelliform lesions present as hyper-reflective deposits between the RPE and the photoreceptors. Hypo-reflective regions of the lesion may also be present, causing confusion with the presence of "fluid."[22-24] On FAF, these lesions are typically hyper-autofluorescent since they are accumulations of lipofuscin (*Figure 4*).[22,23] Electrodiagnostic testing is typically normal in these patients.[20]

BSD. The diagnosis of BSD is primarily clinical with a bilateral butterfly-shaped pattern of lipofuscin deposition and RPE disruption. OCT imaging reveals variable amounts of subretinal deposition and RPE disruption. FAF often highlights the butterfly-shaped pattern of disease. Patients are typically diagnosed in the second to third decade of life and usually have normal electrodiagnostic

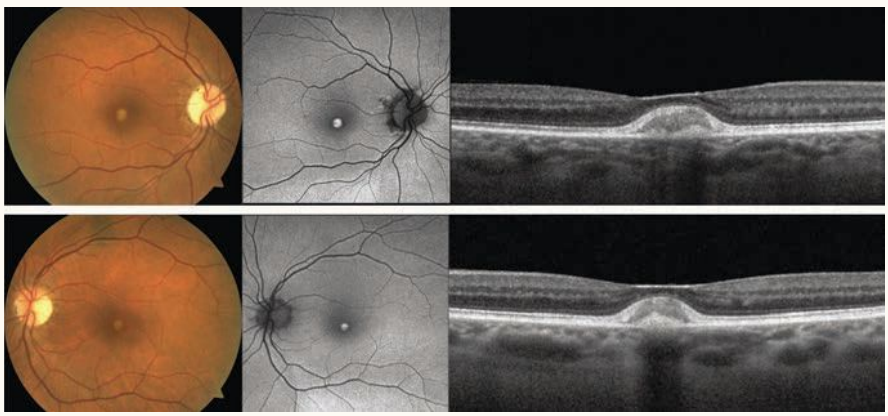

Fig. 4. A 69-year-old white female with AOVD has bilateral, circular, yellow, central vitelliform lesions (left). These lesions are hyper-autofluorescent on FAF (middle) and present on OCT as reflective subretinal deposits between the RPE and the photoreceptors (right). The patient presented with good visual acuity of 20/30 OD and OS.

Table 1. Clinical Staging Criteria of Best Disease

I	Normal fundus, abnormal EOG
II	Egg yolk lesion
III	Pseudohypopyon (yellow vitelliform material settles inferiorly)
IV	Vitelleruptive (scrambled egg appearane)
V	Central RPE atrophy
VI	CNV

studies. The condition is still thought to be autosomal dominantly inherited due to variants in the *PRPH2* gene.[25,26]

Reticular dystrophy. This condition is typically diagnosed clinically by the presence of bilateral, subretinal yellow deposition and pigmentary alterations in a reticular pattern. OCT and FAF are also helpful in visualizing and distinguishing alterations to the outer retina and RPE in this disease as other pattern dystrophies.[27]

Multifocal pattern dystrophy simulating Stargardt's. Considered to be inherited autosomal dominantly by pathogenic *PRPH2* variants, this condition appears clinically and diagnostically similar to Stargardt's disease (*Figure 5*). It may be differentiated through family history consistent with autosomal dominant inheritance; however, incomplete penetrance and variable expression may mask the inheritance pattern. Patients will typically present with findings later in life (fifth decade) and have on average a more stable disease course and better visual acuity later in life than those with Stargardt's. In addition, they do not present with findings of a dark choroid on FA which is reported in the majority of Stargardt's patients.[28,29]

Fundus pulverulentus. The least commonly encountered pattern dystrophy, fundus pulverulentus, is characterized by bilateral course pigment deposition in the macula. FA is helpful to show a typical pattern of hypo-fluorescent spots corresponding to the areas of pigment deposition. OCT and FAF findings have been rarely described.[30-32]

Pseudoxanthoma Elasticum

PXE is caused by autosomal recessive inheritance of mutations on *ABCC6*, leading to calcification of elastic fibers in the eye, skin and vasculature. In the retina, this can lead to calcification of Bruch's membrane causing pigmentary abnormalities such as peau d'orange and angioid streaks. Peau d'orange presents as a pebbly orange appearance of the retina typically in the temporal macula and mid-peripheral retina. Angioid streaks present as linear, radial pigmentary alterations extending from the optic nerve.[33] In addition, PXE has been associated with pattern dystrophy-type appearances.[32,34] PXE is a progressive condition in which vision loss can occur through tissue atrophy or development of CNV.[35] Patients should be monitored carefully for development of CNV as it is common.

OCT can be used to image RPE and Bruch's membrane alterations, and is also useful in identifying presence of CNV.[33] Angioid streaks may be more apparent with FAF imaging than with fundus evaluation, and more diffuse alteration and atrophy of the RPE may be visible with FAF than on clinical examination.[33,36] OCT-A and FA are useful for further evaluation and identification of CNV.[37]

Angioid streaks present during the lifetime of almost all patients with PXE, but they are not exclusive to PXE.[38] They are also seen in patients with Ehlers-Danlos syndrome, Paget's disease and sickle cell disease, and they may be idiopathic.

Bestrophinopathies

This is a term used to describe a phenotypically heterogenous group of disorders caused by variants of the *BEST1* gene. Most gene mutations lead to the phenotype consistent with Best disease, which we will focus on in this article.

Best disease. An autosomal dominant dystrophy, this condition can present as early as the first decade of life. A generally accepted staging criteria for Best disease is shown in Table 1. Lesions in Best disease are typically bilateral and fairly symmetric. While

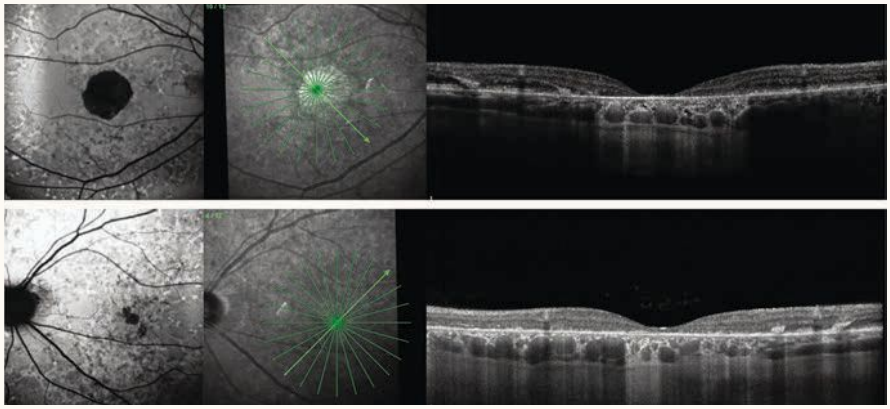

Fig. 5. A 54-year-old white male presented with BCVA 20/70 OD and 20/50 OS. He complained of gradually worsening vision over the last two to three years. He had no family history of blindness. Fundus exam revealed bilateral yellow, fleck-type lesions scatted in the posterior pole and macular atrophy. FAF showed diffuse alteration of the autofluorescent signal. OCT showed hyper-reflective outer retinal deposits OU, photoreceptor atrophy OS and more extensive macular atrophy OD. The appearance is similar to Stargardt's disease, but genetic testing identified a heterozygous pathogenic variant in *PRPH2* more consistent with multifocal pattern dystrophy mimicking Stargardt's.

Table 2. Grading Criteria for North Carolina Macular Dystrophy

Grade	Fundus Findings
1	Central yellow drusen deposits
2	Confluent drusen, possible pigmentary changes
3	Well-defined chorioretinal atrophy

lesions tend to be solitary, reports of multiple lesions per eye do exist. This has been termed "multifocal Best disease."[39] Patients often present with good visual acuity despite the striking fundus appearance. As the condition progresses, patients can develop loss of central acuity, metamorphopsias and central scotomas. Symptoms typically begin in the vitteleruptive stage with more severe loss occurring with progressive RPE atrophy or development of CNV. Sudden vision loss can occur with development of CNV.[15]

OCT is useful to image vitelliform lesions, which appear similarly to those in AOVD. On FAF, the lesions are typically hyper-autofluorescent due to the presence of lipofuscin. RPE atrophy may appear as hypo-autofluorescent. OCT-A and FA are useful in identifying the development of CNV.[40] ERG is normal, but EOG is abnormal.[15]

Macular Drusen

Doyne honeycomb retinal dystrophy (DHRD) and Mallatia Leventinese (ML) are names both used to describe a phenotype of radially oriented macular drusen seen in relatively young individuals who may be in their 20s. These individuals often have peripapillary drusen as well.

The phenotype was first described by Walter Doyne in 1899 in Oxford, England, who coined the name DHRD.[41] In 1932, a similar condition was described in several individuals in the Leventine Valley of Switzerland, then called ML.[42] It is now generally accepted that these two names represent the same condition, caused by autosomal inheritance of a defect in the *EFEMP1* gene that codes for a protein called fibulin 3, an extracellular matrix protein.[43] Other names used to describe the phenotype are dominant drusen and familial drusen. However, evidence suggests that many patients with a phenotype of dominant drusen do not have the *EFEMP1* gene mutation consistent with DHRD and ML.[44,45]

More recently, evaluation of drusen subtypes with multimodal imaging (OCT, FAF, intravenous FA, etc.) revealed that many in this young patient demographic have a phenotype of drusen called cuticular drusen. The term *cuticular drusen* was first used by Donald Gass in 1977 to describe drusen that appeared as numerous small hyper-fluorescent lesions on VA, appearing like a "starry sky." This phenotype is currently being considered as a specific clinical subtype of AMD.[46] Cuticular drusen seem to have a strong genetic component. Multiple genes are currently associated with the condition.[18]

OCT imaging can be used to visualize the drusen deposition and is also useful for the detection of CNV. Cuticular drusen often have a saw tooth-type appearance on OCT (*Figure 6*). FAF findings are variable, but FAF is useful to get a sense of the distribution and amount of RPE disruption and to identify regions of RPE atrophy.[46] OCT-A and FA may be useful for identifying CNV. Electrophysiology testing in those confirmed with *EFEMP1* macular disease has been reported as normal.[47]

Despite the significant amount of drusen seen on clinical examination, patients tend to present with good visual acuity. These conditions are progressive, however, with the possibility of visual decline from macular atrophy or development of CNV.[46]

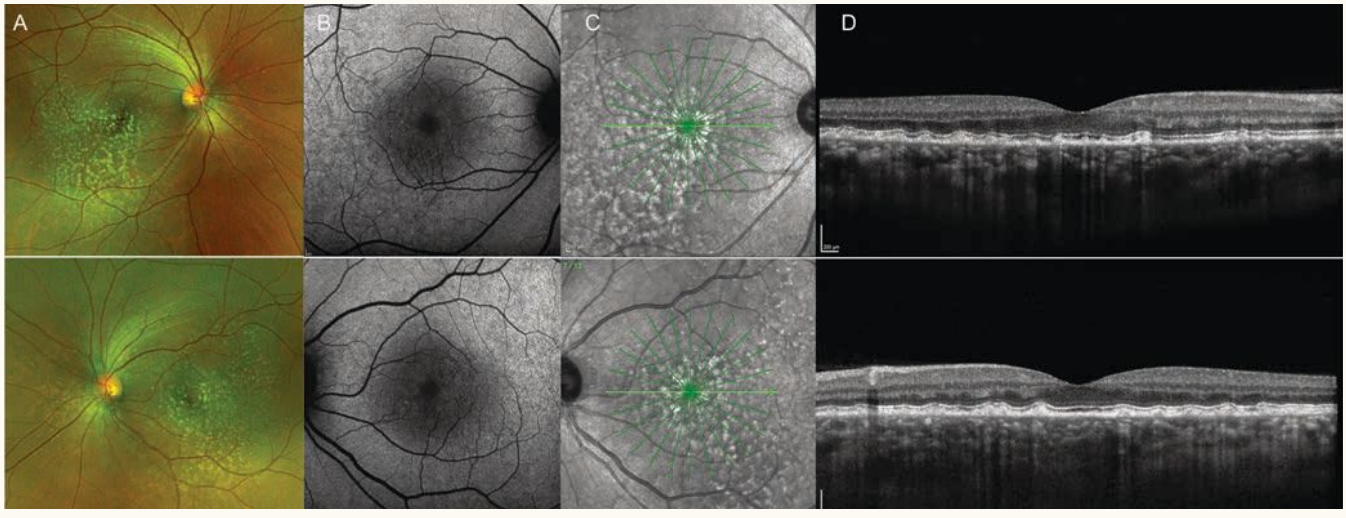

Fig. 6. A 42-year-old Black male presented with a phenotypic appearance of autosomal dominant drusen. He had no family history and presented asymptomatically with 20/20 vision OD and OS. Images: (A) widefield photo, (B) FAF, (C) infrared reflectance and (D) OCT.

Table 3. Clinical Staging of Central Areolar Choroidal Dystrophy

Grade	Fundus Findings
I	Slight parafoveal RPE changes
II	Pigment mottling encircling the fovea
III	Choriocapillaris atrophy without central involvement
IV	Central choriocapillaris and RPE atrophy

North Carolina Macular Dystrophy

This autosomal dominant condition has highly variable expressivity. A grading system has been created for the condition, but rather than acting as a staging criteria of disease progression, this is more of a staging of the phenotypic presentation as the condition is usually non-progressive (*Table 2*). The condition tends to be bilateral and symmetric. Vision loss is grade-dependent, but patients often have surprisingly good visual acuity on fundus presentation. The median acuity is reported to be 20/50. Patients of all grades must be monitored for development of CNV.[15,48]

Grade 3 lesions may appear excavated, and the terms staphyloma and coloboma have been used to describe them. Researchers describe this excavation as a deep chorioretinal excavation and have suggested the name "macular caldera" to describe these lesions.[48] Dispute over the appropriate nomenclature for this excavation still exists.[49-51]

OCT and FAF can demonstrate the level of RPE and photoreceptor disruption. In addition, the deep chorioretinal excavation can be well visualized with OCT imaging. OCT-A and FA may be useful for identifying CNV. EOG and ERG are typically normal.[15,48]

Central Areolar Choroidal Dystrophy

CACD is a rare autosomal dominant condition. A variant in *PRPH2* is one known cause of CACD, but this is not the only gene identified in the condition. Patients present in the second decade of life with subtle bilateral, symmetrical pigment mottling centrally. This progresses to atrophy of the choriocapillaris, RPE and photoreceptors with vision loss beginning in the fourth and fifth decades of life.[52] The condition presents as well defined, circular areas of macular atrophy. CACD progression has been staged in *Table 3*.[15]

Color vision is often abnormal, and later stages lead to visual acuity loss and central scotomas. Full-field ERG tends to be normal, aside from reduction in some advanced cases.[15] Multifocal ERG has recently been reported to show dysfunction in broader regions than appear clinically diseased.[53] Pattern VEP and pattern ERG are reported to be the earliest electrophysiological indicators of disease in patients with normal fundus appearance.[53]

OCT and FAF are very useful in detecting RPE disruptions from early to more advanced stages of CACD. Alterations may be subtle at first, but more advanced stages will show significant RPE and photoreceptor atrophy on OCT and FAF.[54,55]

Myopic Macular Degeneration

MMD describes the atrophic changes that occur in highly myopic eyes, attributed to axial elongation. There is increased risk of MMD with higher refractive error, longer axial length, presence of posterior staphyloma involving the macula and older age (*Figure 7*). Findings in MMD include lacquer cracks (LCs), pigmentary alterations, macular atrophy and development of CNV.[56]

LCs are breaks in the RPE, Bruch's membrane and choriocapillaris complex. These are typically seen in younger patients because as patients age, LCs often coalesce to form larger areas of macular atrophy.[57] Patients with LCs can develop spontaneous, not CNV-related, subretinal hemorrhage due to choriocapillaris ruptures. This is a sign of likely LC expansion.[58] In addition, MMD patients are at high risk for CNV development, and any presence of subretinal hemorrhage must be thoroughly investigated to rule out the presence of CNV on

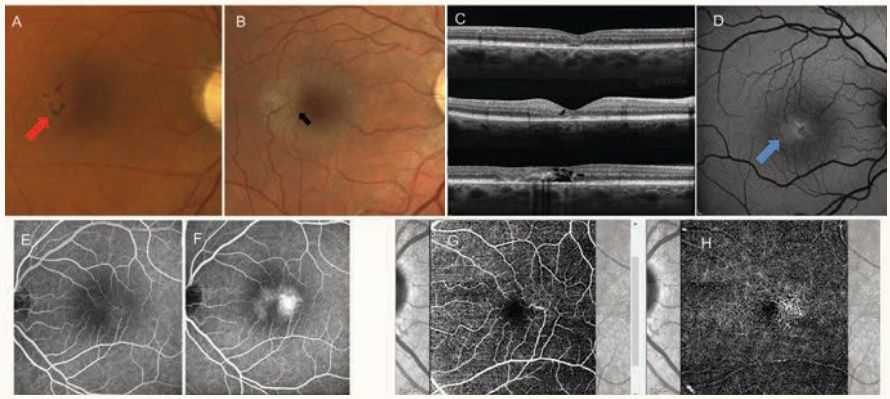

Fig. 7. Patients with MT2 may present with the following findings: (A) pigmentary plaques (red arrow), (B) juxtafoveal whitening concentrated temporally, crystalline deposits and right angle vessels (black arrow), (C) variable retinal and photoreceptor atrophy and classic appearance of internal limiting membrane drape on OCT, (D) altered autofluorescent patterns typically showing first as hyper-autofluorescence temporally (blue arrow), (E) juxtafoveal telangiectatic vessels concentrated temporally on intravenous FA that leak in late stages and (F) telangiectatic vessels concentrated temporally in both the superficial (G) and deep (H) vascular plexus on OCT-A. These images are all examples of different patients.

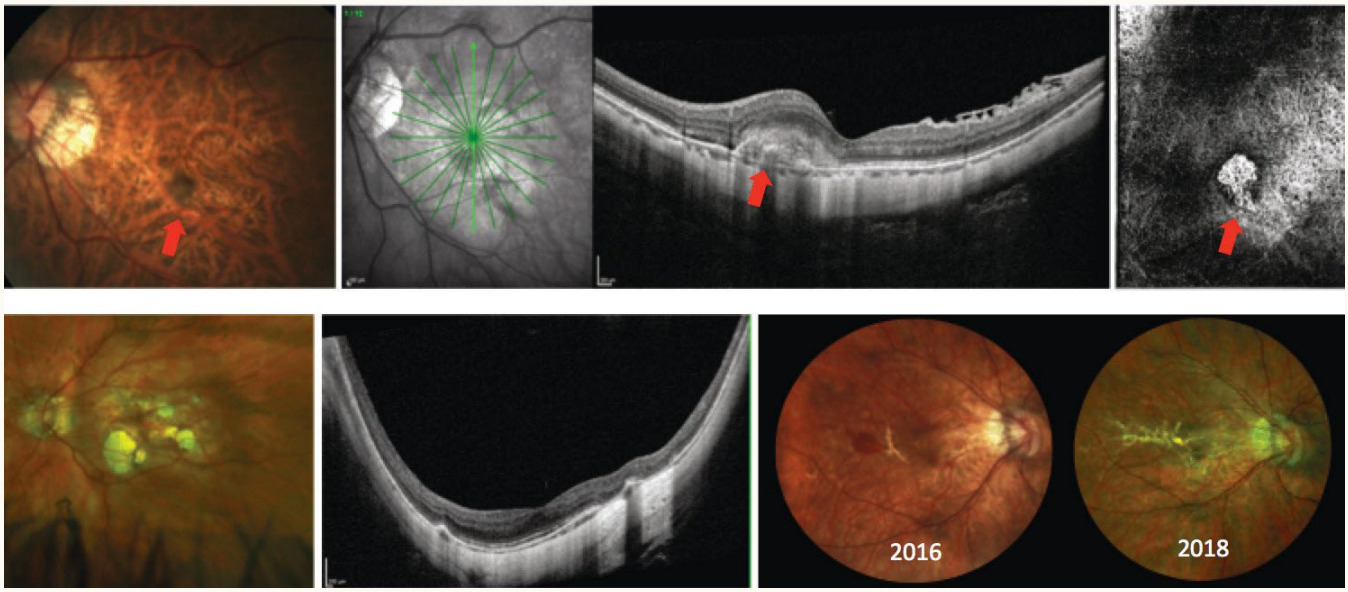

Fig. 8. Various presentations of myopic degeneration. The top images show myopic CNV (red arrows) on fundus photo, OCT and in the avascular complex of the OCT-A. Bottom left shows a patient with posterior staphyloma and myopic macular atrophy. The bottom right shows progressive lacquer crack formation. In 2016, the patient had subretinal hemorrhage not associated with CNV.

OCT, OCT-A and FA as needed.[59]

Macular atrophy can also develop without the presence of LCs. Regions of atrophy expand at variable rates with an increased risk of vision loss with age.[56] While there is no treatment for macular atrophy, early detection of CNV and treatment with anti-VEGF can lead to better visual outcomes.[60]

Macular Telangiectasia Type 2

Patients with MT2 have abnormal parafoveal retinal capillaries most concentrated temporally. While these capillary abnormalities have given rise to the condition's name, at its core MT2 is best described as a neurovascular macular degenerative condition. The pathophysiology is unclear, but it has been said that the cause may be from dysfunction in Muller cells that are vital for the maintenance of retinal health.[61] While a genetic component is suspected, MT2 is not considered a macular dystrophy.[61]

Patients with MT2 tend to present with good visual acuity. The MacTel study group showed that 42% of all patients had best corrected vision of 20/25 or better.[62] Symptoms tend to occur in the sixth or seventh decade of life with impaired reading being the most frequently reported initial symptom.[63] Patients with MT2 may present with subtle retinal findings, making misdiagnosis easy (*Figure 8*). Initially, there is a mild, juxtafoveal retinal whitening most concentrated temporally. Later findings such as reflective deposits, pigmentary plaques and right-angle vessels may also be visible.[61] The condition is bilateral, affecting the temporal juxtafoveal region to the greatest extent, but findings can be asymmetric.

The cause of vision loss in these patients stems from retinal and photoreceptor atrophy that typically affects the temporal juxtafoveal region. This can create scotomas in the presence of good central visual acuity, hence the difficulty with reading. Progression of the disease can lead to macular atrophy and decreased visual acuity. In addition, patients may develop CNV.[61]

OCT shows variable levels of retinal and photoreceptor atrophy. The presence of internal limiting membrane drape on OCT is classic for MT2. FAF shows hyper-autofluorescence temporally early in the disease with increased disruptions in the autofluorescent signal as the disease progresses. Pigmentary plaques appear as hypo-autofluorescent. Early-phase FA shows irregular juxtafoveal telangiectatic vessels most concentrated temporally. These vessels leak in the later stages, but the condition is not considered to be an exudative disease in the absence of CNV. This irregular vasculature can also be detected on OCT-A, showing evidence of telangiectatic vascular alterations most concentrated temporally in both the superficial and deep capillary plexus. OCT, FA and OCT-A are also helpful in identifying the presence of CNV.[61]

There is no treatment to slow the progression of MT2. Treatment of CNV with anti-VEGF has shown to be favorable.[64]

Conclusions

When the macula encounters stress, either from extraneous sources or underlying defects in the system, the phenotypic results may not be unique to a particular condition. Findings such as drusen, lipofuscin deposition, pigmentary alterations, macular atrophy and CNV are seen in a wide variety of conditions.

Careful clinical examination along with imaging strategies such as OCT, FAF, OCT-A and FA help to guide us to a particular condition. Additional

information such as electrodiagnostic studies, age of onset and family history also help to narrow the possibilities.

While we are extremely limited in our treatment options for macular dystrophies and degenerations, arriving at an accurate diagnosis allows us to educate patients and their family members about their visual prognoses. It can also help guide decisions on the value of genetic testing.

All patients with macular degenerations or dystrophies are at increased risk of developing CNV and should be monitored for this occurrence. In general, treatment with intravitreal anti-VEGF is favorable in those who develop CNV. Those left with visual impairments that affect their daily routine should be referred to a low vision specialist. ■

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1. What is reported as the most common inherited macular dystrophy?
a. Best disease.
b. Stargardt's disease.
c. Reticular dystrophy.
d. MT2.

2. What is the typical age of onset in Stargardt's disease?
a. Before 10 years old.
b. 10 to 20 years old.
c. 20 to 40 years old.
d. 40 to 50 years old.

3. What is the earliest reported ERG abnormality in cone dystrophies?
a. Reduced full-field scotopic ERG.
b. Reduced full-field photopic ERG.
c. Delayed 30Hz flicker implicit time.
d. Reduced 30Hz amplitude.

4. Which of the following is not considered a pattern dystrophy?
a. Best disease.
b. AOVD.
c. BSD.
d. Fundus pulverulentus.

5. What is the typical age of diagnosis of AOVD?
a. 20 to 40 years old.
b. 40 to 50 years old.
c. 50 to 70 years old.
d. 70 to 80 years old.

6. How do vitelliform lesions present on FAF?
a. Hyper-autofluorescent.
b. Hypo-autofluorescent.
c. Hyper-reflective.
d. Hypo-reflective.

7. Which of the following is not typical of multifocal pattern dystrophy simulating Stargardt's disease?
a. Early vision loss around 20 years old.
b. Autosomal dominant inheritance.
c. A more stable disease course with better visual acuity than Stargardt's disease.
d. Absence of silent choroid on FA.

8. What is the inheritance pattern of PXE?
a. Autosomal dominant.
b. Autosomal recessive.
c. X-linked.
d. The inheritance pattern is unknown.

9. Which of the following is not typically seen in patients with PXE?
a. Angioid streaks.
b. Peau d'orange.
c. Pattern dystrophy-type appearance.
d. Macular caldera.

10. What gene is most often responsible for conditions considered to be bestrophinopathies?
a. *ABCA4*.
b. *ABCC6*.
c. *BEST1*.
d. *EFEMP1*.

11. When does Best disease typically present?
a. Childhood.
b. Early adulthood.
c. Mid-life.
d. In the elderly.

12. DHRD and ML are caused by autosomal dominant inheritance of which gene?
a. *ABCA4*.
b. *ABCC6*.
c. *BEST1*.
d. *EFEMP1*.

13. Patients with autosomal dominant drusen have increased risk of vision loss from which of the following?
a. Optic nerve atrophy.
b. Macular atrophy or CNV.
c. Cataracts.
d. Retinal detachment.

14. How would you describe the grading system of North Carolina macular dystrophy?
a. Staging system for the progression of the disease.
b. Staging system of the phenotypic presentation as it tends to be non-progressive.
c. Staging system based on ERG findings.
d. Staging system based on the age of onset of the disease.

15. Which of the following accurately describes electrodiagnostic findings in CACD?
a. Full-field ERG is always reduced.
b. Multifocal ERG shows reduction only in areas of visible disease.
c. Pattern VEP and pattern ERG are reported to be the earliest electrophysiological indicators of disease.
d. The EOG is abnormal.

16. Which is not a risk factor for MMD?
a. Higher myopic refractive error.
b. Longer axial length.
c. Presence of posterior staphyloma in the macula.
d. Younger patient age.

17. What is the most frequently reported visual symptom of MT2?
a. Photophobia.
b. Nyctalopia.
c. Impaired vision while reading.
d. Loss of peripheral vision.

18. What is a classic OCT finding of MT?
a. Internal limiting membrane drape.
b. Subretinal drusenoid deposits.
c. Thickening of the external limiting membrane.
d. Vitelliform lesions.

19. Which of the following conditions would be most suitable to recommend genetic testing for?
a. MT2.
b. Stargardt's disease.
c. AOVD.
d. MMD.

20. What is generally true regarding the management of non-AMD macular dystrophies and degenerations?
a. Patients should be supplemented with AREDS 2 vitamins.
b. Treatment options are limited, but anti-VEGF should be recommended for CNV development, and those with vision impairements should be referred to a low vision specialist.
c. Multiple FDA-approved gene therapies are now on the market for macular dystrophies and degenerations.
d. There is no way to improve the outcomes for patients with macular dystrophies and degenerations, so monitoring and referring to specialist providers is not necessary.

Examination Answer Sheet

What To Do When It's Not AMD
Valid for credit through June 15, 2024

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.

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

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Answers to CE exam:

1.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
2.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
3.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
4.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
5.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
6.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
7.	Ⓐ	◯	Ⓒ	Ⓓ
8.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
9.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
10.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
11.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
12.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
13.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
14.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
15.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
16.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
17.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
18.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
19.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
20.	Ⓐ	Ⓑ	Ⓒ	Ⓓ

Post-activity evaluation questions:

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

21. Understand the pathophysiology of macular disorders and dystrophies. ① ② ③ ④ ⑤
22. Differentiate between common macular disorders and dystrophies. ① ② ③ ④ ⑤
23. Use the clinical exam techniques and tools to diagnose their patients. ① ② ③ ④ ⑤
24. Understand how different macular conditions progress as well as their role in monitoring and managing care. ① ② ③ ④ ⑤
25. Based upon your participation in this activity, do you intend to change your practice behavior? (Choose only one of the following options.)

Ⓐ I do plan to implement changes in my practice based on the information presented.
Ⓑ My current practice has been reinforced by the information presented.
Ⓒ I need more information before I will change my practice.

26. Thinking about how your participation in this activity will influence your patient care, how many of your patients are likely to benefit? (please use a number): ☐
27. If you plan to change your practice behavior, what type of changes do you plan to implement? (Check all that apply.)
28. How confident are you that you will be able to make your intended changes?

Ⓐ Apply latest guidelines
Ⓑ Change in diagnostic methods
Ⓒ Choice of management approach
Ⓓ Change in current practice for referral
Ⓔ Change in vision correction offerings
Ⓕ Change in differential diagnosis
Ⓖ More active monitoring and counseling
Ⓗ Other, please specify: _____

Ⓐ Very confident Ⓑ Somewhat confident Ⓒ Unsure Ⓓ Not confident

29. Which of the following do you anticipate will be the primary barrier to implementing these changes?
30. Additional comments on this course: _____

Ⓐ Formulary restrictions
Ⓑ Time constraints
Ⓒ System constraints
Ⓓ Insurance/financial issues
Ⓔ Lack of interprofessional team support
Ⓕ Treatment related adverse events
Ⓖ Patient adherence/compliance
Ⓗ Other, please specify: _____

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Rate the quality of the material provided:

1=Strongly disagree, 2=Somewhat disagree, 3=Neutral, 4=Somewhat agree, 5=Strongly agree

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

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

Signature _____ Date _____ Lesson 121330 RO-OSC-0621

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Through Thick and Thin

Drug improves endothelial function but also yields corneal edema.

Q The effect of Rhopressa (netarsudil, Aerie) on endothelial function is exciting, yet some patients have shown significant epithelial edema. What is the exact mechanism of this drop?

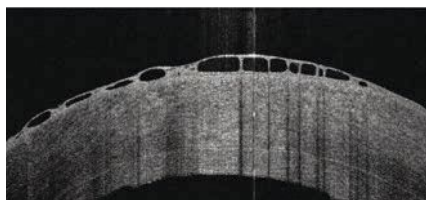
A "The use of Rhopressa in the management of pathology of corneal endothelial cells (CECs) is a therapy in its infancy, but also one of the most exciting and active areas of publication in cornea literature," according to Aaron Bronner, OD, of Pacific Cataract and Laser Institute in Kennewick, WA. Most of this research has been retrospective and case-based, but as of March, the first randomized clinical trial studied the use of Rhopressa in Fuchs' dystrophy patients and reported an improvement in corneal thickness and visual acuity over three months.[1]

A medical approach to endothelial decompensation would be a revolutionary treatment, says Dr. Bronner, but notes the importance of having realistic expectations and recognizing potential complications, the most common of which is honeycomb epithelial edema.

The Nitty-Gritty

Honeycomb epithelial edema is concerning both in that it is extremely common in patients with corneal endothelial disease treated with netarsudil and that its presence seemingly flies in the face of the proposed benefit of the therapy, says Dr. Bronner, as these two outcomes are distinctly at odds.

The field is still in the early stages of research into this question, but based on available data and augmented by his own use of netarsudil in this population, Dr. Bronner believes that honeycomb epithelial edema counterintuitively does not cause an increase in corneal edema or have a negative influence on CECs. Instead, he thinks it's probably associated with a change in the distribution of edema within corneas altered by ROCK inhibition. He notes that these eyes have not shown a consistent worsening of edema as measured by pachymetry.[2] In fact, the eyes he has seen with honeycomb edema have surprisingly exhibited thinning. So, what is going on?



Honeycomb edema in a patient treated with Rhopressa who underwent a failed DSAEK.

When establishing a cause of honeycomb edema, pinpoint where the problem is localized, who will develop it and the conditions that will help it clear. Although ROCK inhibitors are gaining attention for their role in endothelial healing, the endothelium isn't the only cellular layer within the cornea that they impact. They also play a role in epithelial healing, cellular and intracellular junctions and maintenance of membrane permeability of epithelial cells.[3,4]

The at-risk patient profile is very specific. According to one review, honeycomb epithelial edema is nearly universal in patients treated with netarsudil who also have corneal edema or at least a significant risk factor for edema, but it doesn't seem to occur in those treated with netarsudil who have a healthy endothelium.[5] Therefore, both substantial CEC disease and netarsudil are necessary ingredients for the development of honeycomb edema. If either of these conditions change—the edema clears or the medication is discontinued—the honeycomb appearance will dissipate.[2,5,6]

Takeaways

The anterior localization of the problem, influence of ROCK inhibitors on cellular permeability of epithelial cells and requirement of pre-existing edema for the development of honeycomb edema make it very likely that this adverse effect reflects a change in the fluid balance in how *epithelial* cells respond to edema, rather than an impact on endothelial cells or a worsening of edema, Dr. Bronner notes.

Of course, this is all speculative. Time and research will shed light on the full picture, but for now, Dr. Bronner says he feels comfortable placing his patients with corneal edema on Rhopressa as an off-label way to attempt to improve corneal edema. In the event that honeycomb edema develops, he gives the medication up to a month longer. If it continues to persist, simply discontinuing the medication should result in its resolution with less of a risk for long-lasting harm. ■

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4. Yin J, Yu FSX. Rho kinases regulate corneal epithelial wound healing. Am J Physiol Cell Physiol. 2008;295(2):C378-87.

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About Dr. Shovlin

Dr. Shovlin, a senior optometrist at Northeastern Eye Institute in Scranton, PA, is a fellow and past president of the American Academy of Optometry and a clinical editor of *Review of Optometry* and *Review of Cornea & Contact Lenses*. He consults for Kala, Aerie, AbbVie, Novartis, Hubble and Bausch + Lomb and is on the medical advisory panel for Lentechs.

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REVIEW Education GROUP

BY JAMES L. FANELLI, OD

Don't Feed the Hand that Bites You

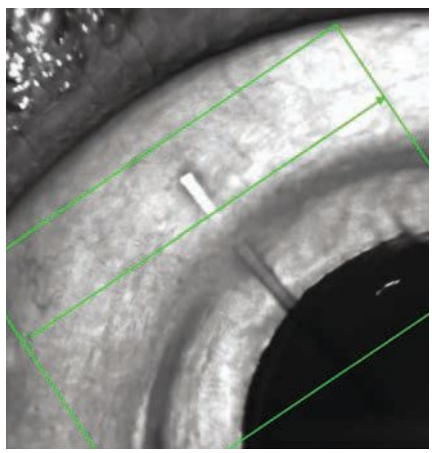
Manage your glaucoma patients to the best of your ability before initiating a referral that may ultimately do more harm than good.

Second-guessing another doctor's clinical care should not be taken lightly, but if you do, be judicious in your approach. Here are several takeaways from when I found myself in this position.

Case

I was examining a 55-year-old Caucasian female a couple of weeks ago. During the evaluation, she mentioned her mother and how hard of a time she was having with her glaucoma. Alarm bells went off in my head while she was giving me her version of events. We should always take a secondhand impression of events with a grain of salt, but there were several items mentioned that were, unfortunately, all too familiar. One thing led to another and I agreed to see the patient's mother for a second opinion.

She was an 81-year-old woman who presented anxiously about her "failed glaucoma surgery" in her left eye. She had been treated for approximately 10 years for bilateral glaucoma, which she reported was now well controlled with daily Rhopressa (netarsudil, Aerie). After moving to the area, she noted a decline in vision in the left eye and sought the care of a local OD who immediately referred her to a glaucoma surgeon. The surgeon changed her glaucoma medications and scheduled her for surgery the following week.



This MIGS device is designed to facilitate drainage of aqueous from the anterior chamber into the subconjunctival space.

Post-op, the patient was told the procedure had failed, there was some bleeding and there was nothing more that could be done. After her third follow-up, she sought my care.

At her first visit with me at the end of April, entering visual acuities were 20/25 OD and 20/200 OS. Best-corrected acuities were 20/25+ OD and 20/80 OS. Pupils were round, equal and without an afferent pupillary defect, and extraocular muscles were full in all positions of gaze. Current medications included Xelpros (latanoprost, Sun Pharma) QHS OU, Cosopt (dorzolamide/timolol, Akorn) TID OU and prednisolone acetate QD OS, along with oral diazepam, trazodone and prednisone. She reported an allergy to indomethacin.

Slit lamp examination of her anterior segments was remarkable for bilateral LASIK flaps, with a clear interface and no striae or epithelial ingrowth bilaterally. She had undergone LASIK approximately 15 years earlier. Pachymetry readings were 522µm OD and 557µm OS. Applanation tensions at 11:06am were 12mm Hg OD and 10mm Hg OS. There were scattered guttatae, and the patient was pseudophakic bilaterally. She'd had cataract surgery about seven years earlier.

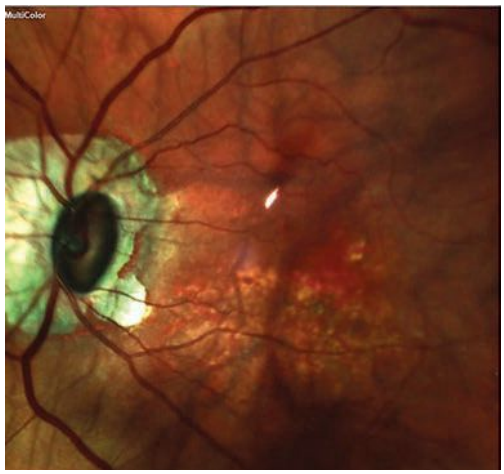
In the right eye, there was a tube in the anterior chamber extending to the visual axis on one end, and to about 2mm behind the limbus and beneath the conjunctiva at the other. There was no valve present, nor was the tube cut from a valve that had failed. Close examination of the conjunctiva distal to the tube demonstrated no explanted valve or disrupted conjunctiva. There was no bleb.

The patient was dilated in the usual fashion. Through dilated pupils, her intraocular lenses were clear and centered in their capsular bags. The posterior capsules were clear and intact OU. Bilateral posterior vitreous detachments were present. Posterior pole imaging demonstrated bilateral tilted discs with peripapillary atrophy and myopic stretching. The left disc was tilted much more than the right, and consequently, both cup-to-disc appearances were vertically elongated due to the oblique nasal insertion of the optic nerves OS>OD.

There was concurrent glaucomatous damage present OU. But my initial impression was that, although damage from glaucoma was evident, neither nerve was at the threshold of complete neuroretinal rim loss. Prior to her LASIK surgery, the patient said she

About Dr. Fanelli

Dr. Fanelli is in private practice in North Carolina and is the founder and director of the Cape Fear Eye Institute in Wilmington, NC. He is chairman of the EyeSki Optometric Conference and the CE in Italy/Europe Conference. He is an adjunct faculty member of PCO, Western U and UAB School of Optometry. He is on advisory boards for Heidelberg Engineering and Glaukos.



The patient's left eye demonstrated an oblique insertion of the optic nerve, along with macular changes and thinning of the neuroretinal rim.

was a -9.00 OU myope. This would certainly account for her myopic optic nerve characteristics.

The right macula was characterized by fine retinal pigment epithelial mottling; that of the left was characterized by more advanced retinal pigment epithelial disruption as well as scattered drusen. Given the symmetric pre-LASIK refractive error and the lack of myopic stretching in the right eye, it is possible that some of the macular changes in her left eye were related to myopic stretching. When asked if she was told about the changes in her maculae, OS>OD, she looked surprised and said no.

The retinal vascular examination was essentially unremarkable, except for expected mild arteriolar changes associated with her age. Her peripheral retinal evaluations were remarkable for 360° cystoid and scattered areas of pavingstone degeneration. I obtained optic nerve images and anterior segment OCTs of the tube/shunt.

Discussion

Examining this patient raised a few questions and brought home several points. First, the patient sought care due to decreased vision OS. Was her vision decreased because of progressive field loss secondary to the glaucoma? Or, was it decreased due to progression of the macular changes in the left eye? The only way to determine the answer prior to glaucoma surgery would have been through completing a visual field study and assessing previous records, neither of which was done.

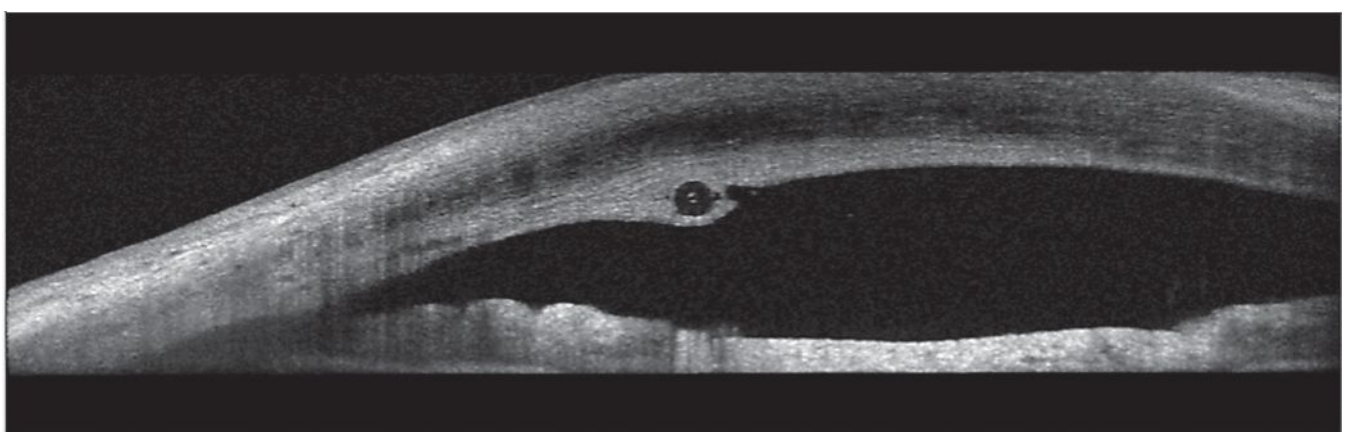
Second, we must be careful about abruptly changing the meds of a new patient who had been managed by another provider for years beforehand. Obtaining prior records helps tremendously in determining what, if anything, needs to be done differently. Our patient may not have required a medication change. At the very least, the efficacy of the change should have been evaluated prior to surgery. It is certainly possible that her glaucoma was completely stable and her vision changes were related to her macular issues instead. But in any event, the glaucoma surgeon didn't think twice about proceeding with surgery.

The device implanted was a Xen Gel Stent (Allergan), which is a flexible stent inserted perpendicular to and through the angle and extends into the subconjunctival space. It works to reduce intraocular pressure by facilitating aqueous movement out of the anterior chamber and into the subconjunctival space, similar to a trabeculectomy. The problem is, the stent was not properly placed in this patient, who may not have even needed it in the first place.

Takeaways

It's important to avoid throwing another provider under the bus; instead, give them the benefit of the doubt. However, when a provider actively works against optometry in scope expansion issues, citing our inability to properly manage glaucoma, and leaves a trail of excessive and unnecessary surgeries in multiple patients over years, you begin to realize that involving this provider in your patients' care is not in their best interest.

We as ODs can and should facilitate better glaucoma care by becoming more active in patient management, as opposed to immediately referring to a glaucoma surgeon. Lean on the ODs in your area who manage glaucoma for help, if needed, as most glaucoma patients do not require surgery. Becoming more involved in patient care can reduce unnecessary referrals that may ultimately end up doing more harm than good, as well as improve overall outcomes. ■



The stent was improperly placed. Here, it runs through the cornea, anterior to the anterior chamber angle.

BY JOSEPH W. SOWKA, OD

Not a BRITE Idea

Beware this severe complication from a rare cosmetic procedure.

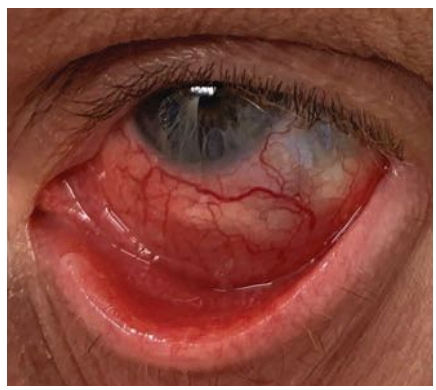
A 59-year-old man presented urgently with a red, painful, photophobic left eye of approximately 10 days duration. His uncorrected visual acuity was 20/40 OD and finger counting OS with no pinhole improvement. His left eye manifested profound deep injection throughout his bulbar conjunctiva. Additionally, there was a mixed papillary and follicular response of the palpebral conjunctiva. There was a grade 3 cell and flare reaction of the left eye with stromal corneal edema and endothelial keratic precipitates.

His crystalline lens manifested an age-appropriate nuclear sclerosis in the right eye but a dense nuclear cataract in the left. His intraocular pressures (IOP) were 18mm Hg OD and 34mm Hg OS. There was also temporal conjunctival and scleral thinning OS and a calcific scleral plaque. His right eye was normal. Cataract and posterior synechia prevented any views of the left fundus.

Based upon signs and symptoms, he was diagnosed with anterior scleritis OS. He was prescribed topical difluprednate 0.05% QID, atropine 1% BID, Combigan (brimonidine/timolol, Allergan) BID and oral ibuprofen 800mg QID PO. His medical history was significant only for diabetes, with no suggestion of autoimmune or rheumatologic diseases. He was referred for medical evaluation with a rheumatologist to find a potential underlying cause.

Painful Reaction

Scleritis is an inflammation of the sclera.[1-4] Patients often report ocular pain that may radiate to involve the adjacent head and facial regions. Photophobia and lacrimation are common. Depending on the involvement of the cornea and severity of inflammation, vision can be reduced. While scleritis may be local and idiopathic, most cases are secondary to systemic disease as arthritis, medication side effects or as a complication of ocular surgery.[5-7]



Anterior scleritis in a patient post-surgery.

Although the pathogenesis of scleritis is not entirely understood, evidence points to a deposition of immune complexes within the sclera, leading to a vasculitis with associated inflammatory cell infiltration and edema.[8] There typically will be dilation of the scleral vessels as well as the overlying vasculature of the episclera and bulbar conjunctiva.[1-4] The affected eye may assume a deep red, almost hemorrhagic appearance.[4-6] The presentation may be sectorial but is usually diffuse, differentiating it from the more common and benign episcleritis. Scleritis is bilateral in many cases but is often asymmetric. Corneal involvement in the form of infiltrative stromal keratitis, non-inflammatory corneal thinning or peripheral ulcerative keratitis is possible.[9] Glaucoma can occur from inflammation or angle closure secondary to choroidal effusion.[10]

Necrotizing scleritis is a particularly severe form where the sclera thins to the point that the underlying dark hue of the choroid is visible.[11] The most destructive form of necrotizing scleritis is scleromalacia perforans, presenting insidiously without substantial pain or visible inflammatory signs with uveal herniation through a perforated scleral wall.[12]

Scleritis can be associated with both infectious and non-infectious causes. While the etiology remains idiopathic for many cases of scleritis, most are associated with a causative systemic disease. The most common related disorders are rheumatoid arthritis, polyarteritis nodosa, systemic lupus erythematosus, inflammatory bowel disease, sarcoidosis, granulomatosis with polyangiitis, tuberculosis, herpes zoster and syphilis.[13] Assume that there is an underlying systemic disease until proven otherwise. Patients should undergo a comprehensive medical evaluation. A rheumatologist is the best comanagement source.

Treatment

Topical therapy alone is typically insufficient to manage most cases of scleritis and should be considered adjunctive to ameliorate initial acute symptoms. Initial topical therapy involves potent cycloplegia with atropine 1% BID and topical steroids such as prednisolone acetate 1% Q2h to QID or difluprednate TID-QID.

Systemic treatment begins with oral nonsteroidal anti-inflammatory drugs (NSAIDs) for mild to moderate, non-necrotizing anterior scleritis.[1] Therapy may include ibuprofen 600mg to 800mg QID or naproxyn sodium

About Dr. Sowka **Dr. Sowka** is an attending optometric physician at Center for Sight in Sarasota, FL, where he focuses on glaucoma management and neuro-ophthalmic disease. He is a consultant and advisory board member for Carl Zeiss Meditec and Bausch Health.

250mg to 500mg TID. If insufficient, oral prednisone 60mg to 80mg PO QD can be given for two to three days and then slowly tapered to 10mg to 20mg daily. Immunosuppressive agents such as cyclophosphamide, cyclosporine or methotrexate are sometimes necessary in the most severe or recalcitrant cases.[14,15]

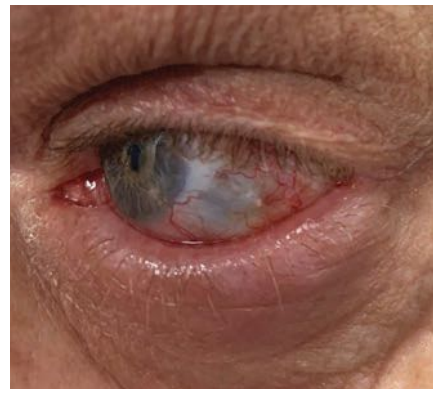
Based upon lack of insurance, the patient was resistant to rheumatologic consultation or any systemic medical testing. During the course of his therapy and follow-up, he casually mentioned that he had undergone a cosmetic conjunctival whitening procedure called "I-BRITE" approximately five years prior in another state. Initially, this elicited no recollection, but later investigation of this procedure proved very informative and ultimately the likely cause.

Dangers of Whitening

Eye whitening procedures were introduced around 2008 and have been offered as a treatment of chronic conjunctival hyperemia. Patients variably undergo conjunctivectomy with topical mitomycin C (MMC) 0.02% application to achieve a whitened appearance from bleaching of the avascular sclera. In some procedures, MMC is combined with antiangiogenic bevacizumab during the procedure.

The literature has noted complications of cosmetic eye whitening, including chronic conjunctival epithelial defects, scleral thinning, avascular zones in the sclera, dry eye syndrome and diplopia requiring strabismus surgery.[16] One review of 1,713 patients undergoing cosmetic whitening procedures noted an overall complication rate of 83%, of which 55.6% cases were considered severe. These severe complications included fibrovascular conjunctival tissue proliferation, scleral thinning with calcified plaques, IOP elevation, diplopia and recurrence of hyperemic conjunctiva.[17]

Another study found that the average time from the procedure to diagnosis was 51 months, and all patients had unilateral findings. There was no



Scleral thinning in a patient with scleritis.

underlying systemic autoimmunity or infectious etiology found. The authors noted that, because of the large area of the ocular surface that is treated in eye whitening with MMC, the necrotizing scleritis that can ensue may be more extensive and severe than the surgically induced necrotizing scleritis following other periocular surgeries.[18]

One study reported on a patient who developed bilateral necrotizing scleritis within the nasal region of both eyes. The patient also developed calcified plaques within the areas of scleromalacia, along with an epithelial corneal defect four years after undergoing I-BRITE. It found a delayed development of complications.[19]

In an attempt to raise awareness of the complications for surgical conjunctival eye whitening procedures, one study reviewed the medical records of patients who received cosmetic conjunctivectomy plus postsurgical topical MMC treatment to eliminate conjunctival injection in a single facility. They found that of the 48 patients undergoing the procedure, 44 had complications related to the procedure. These complications included fibrovascular conjunctival adhesion at the muscle insertion site, chronic dysfunctional tear syndrome, abnormal vessel growth, lymphangiectasis, adhesions of Tenon's capsule and the conjunctiva at the extraocular muscle insertion site, extraocular muscle fiber exposure and diplopia.[20]

For this patient, topical and oral anti-inflammatory therapy, physical removal of the calcific plaque and cataract extraction ultimately were able to restore ocular health and visual function after several months. He was informed that a similar situation could still develop in his fellow eye as the I-BRITE procedure was done bilaterally.

Be aware of cosmetic surgical conjunctival whitening procedures that can have an unacceptably high attendant complication rate and risk of serious vision-threatening outcomes, even years after the procedure. ■

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BY PAUL M. KARPECKI, OD
CHIEF CLINICAL EDITOR

Five Easy Pieces

Follow these steps to increase your dry eye diagnostic accuracy.

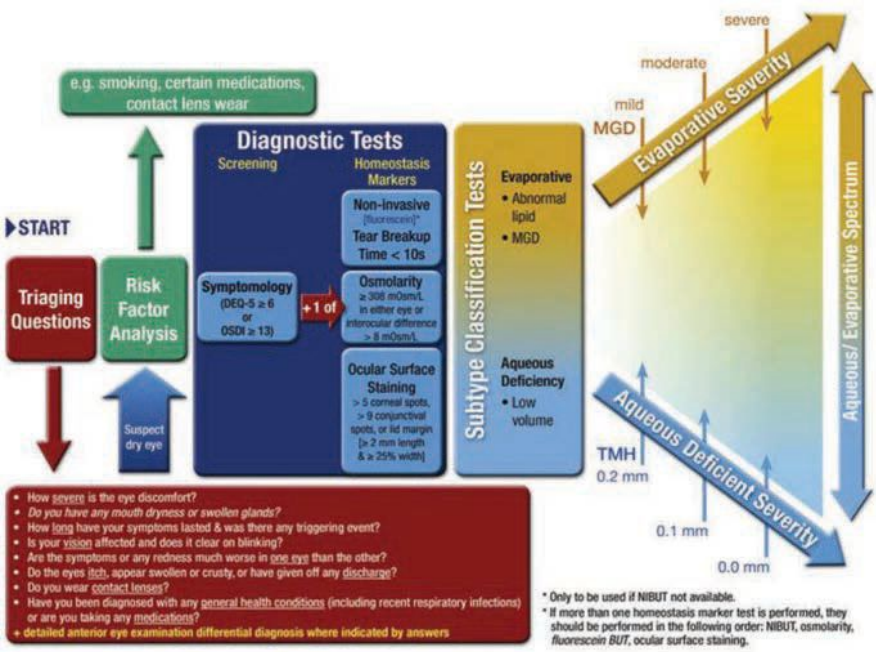
It seems impossible that an algorithm for dry eye disease (DED) diagnosis can have almost 600 scientific references and yet still be so simple every clinician can implement it, but that's what the TFOS DEWS II Diagnostic Methodology Subcommittee came up with—and it works! Having been fortunate to serve on this committee, I can tell you firsthand it's a practical approach that streamlines your efforts and increases diagnostic accuracy.

The first thing you have to recognize is that DED requires both a sign and a symptom to make the diagnosis. For example, an extreme sign like punctate epithelial keratitis without symptoms isn't dry eye—it's likely neurotrophic keratitis. Let's look at the five key steps in this diagnostic methodology.

1. Ask Triaging Questions

The first action in identifying DED is to ask a series of triaging questions. My favorite ones came from the Optometric Dry Eye Summit, which took place in Denver in 2014, although the TFOS DEWS II report, released in 2017, has far more examples. Somewhat paraphrased, these questions are:

- How do your eyes feel? (*i.e.*, are they dry, gritty, light sensitive, burning or stinging?)
- How do your eyes look? (*i.e.*, are they red or look irritated?)
- Do you experience fluctuating vision?
- Do you use, or have the urge to use, artificial tears or rewetting drops?
- How much time do you spend on digital devices?



The TFOS DEWS II dry eye diagnostic algorithm at a glance.

2. Assess Risk Factors

Next, look at the risk factors. The list includes use of various medications like oral antihistamines and topical glaucoma medications, as well as history of contact lens wear, previous ocular surgery and autoimmune conditions such as thyroid disease, diabetes, arthritis, smoking and others.

3. Inquire about Symptoms

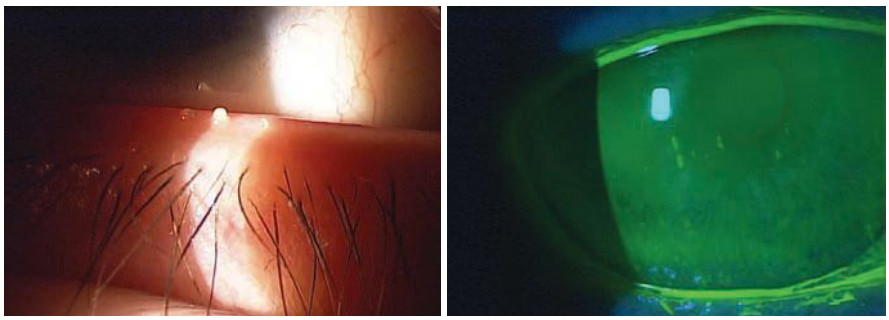
Essential to a dry eye diagnosis is how the patient feels. Any of the following could indicate DED: dryness, grittiness, burning or stinging, tearing, foreign body sensation, itching, contact lens intolerance, fluctuating or blurred vision, hyperemia, photophobia and pain/discomfort.[2]

Although eye dryness is likely the best single indicator—after all, it's right there in the name of the condition—a few others that stand out are fluctuating vision, tearing (epiphora) and hyperemia. In fact, if you are refracting a patient and the image clears and then blurs with each blink, consider DED. Tearing is difficult for patients to assess, as they can't understand how "dry" eye could cause excess tears. What typically occurs is meibomian gland dysfunction (MGD) and the body's response to it is reflex tearing, so an explanation of mechanisms to the patient is warranted. The last indicator is hyperemia, which is often noted by patients and indicates inflammation, also strongly associated with DED.

An easier way to assess symptoms is having the patient take a validated questionnaire before examination. The TFOS DEWS II Diagnostic Methodology Committee recommends the DEQ-5, and other options include the SPEED or OSDI questionnaires. The benefit of this step is that it provides a score that can be monitored for changes over time.

About Dr. Karpecki

Dr. Karpecki is medical director for Keplr Vision and the Dry Eye Institutes of Kentucky and Indiana. He is the Chief Clinical Editor for *Review of Optometry* and chair of the New Technologies & Treatments conferences. A fixture in optometric clinical education, he consults for a wide array of ophthalmic clients, including ones discussed in this article. Dr. Karpecki's full disclosure list can be found in the online version of this article at www.reviewofoptometry.com.



Evaporative DED, a consequence of MGD (left), and aqueous-deficient (right) and are the two primary types of dry eye.

4. Identify Signs

To help in the diagnostic flow, the committee broke out diagnostic tests between global homeostasis testing and subtyping of DED. In other words, a failed homeostasis test indicates the presence of DED (if symptoms are also present) but classifying dry eye further is still required. The three homeostasis tests to consider are osmolarity testing, ocular surface staining and tear film break-up time (TFBUT). Although the paper suggests using one of the three, I think two of three is still rather efficient and increases accuracy.

To assess osmolarity, bear in mind that if the patient has a reading under 300mOsmol/L and each eye is within 6mOsmol/L of the other (*e.g.*, osmolarities of 291 OD and 293 OS), the patient likely doesn't have dry eye disease.[3] A reading over 308mOsmol/L or significant instability between eyes is indicative of DED.[4,5]

Ocular surface staining involves observing both the cornea and conjunctiva for superficial punctate keratitis, and the recommended approach is a noninvasive TFBUT.

Once one of these is positive, you have signs to go with symptoms and a diagnosis of DED is made.

5. Find the Subtype

Finally, we need to determine the type of dry eye so that you can begin appropriately targeted treatment. There are two primary types—evaporative and aqueous-deficient—but overlap can occur. Evaporative DED is a consequence of MGD, while aqueous-deficient DED refers to issues involving the lacrimal glands and/or mucin producing goblet cells.

To determine if a patient has evaporative DED, simply express the meibomian glands. While at the slit lamp, use a Mastrota Meibomian Paddle, Collins forceps, wet Q-tip or your fingers to gently press on the lower nasal to central eyelid. Normal expression should be clear and thin like olive oil. Abnormal is turbid, thickened, paste-like or non-expressive.

To determine if it is aqueous-deficient DED, look at the lower tear meniscus height while the NaFl is present. Any measurement under 0.2mm is considered abnormal.

Over to You

There, you've just saved yourself from reading more than 50 pages of the TFOS DEWS II report—although I do believe it is well worth reading for those who want a deeper dive! ■

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Dredging Up the Past

Evident scarring and a telltale history point to the diagnosis.

BY MARK T. DUNBAR, OD, AND STÉPHANE FITOUSSI, OD
MIAMI

A 59-year-old African-American male presented with blurry vision and a central spot in his right eye for the past four years. He reported seeing floaters that began two years prior; his past ocular history was also significant for a motor vehicle accident that occurred four years earlier. In the accident, he was hit on the right side of his body and reported having sudden vision loss in the right eye.

His medical history was significant for a stroke involving the left hemisphere, also four years prior, as well as Type 2 diabetes, which was diagnosed nine years ago and is being treated with metformin and insulin injections.

Upon exam, best-corrected visual acuity was 20/200 OD and 20/20 OS. Confrontation visual fields were full-to-careful finger counting OU. Pupils were equal, round and reactive, but there was an afferent pupillary defect in the right eye. The anterior segment exam was unremarkable except for mild cataracts OU, and there were no cells or flare in either eye.

On dilated fundus exam of the right eye, a posterior vitreous detachment was present. There was a large fibrotic scar with areas of pigment surrounding in the macula (*Figure 1*). Upon careful examination, a suspicious orange area was noted temporal to that lesion (*see yellow arrow in Figure 1*). An OCT was performed and is available for review (*see red arrow in Figure 2*). The retinal exam of the left eye was completely normal.

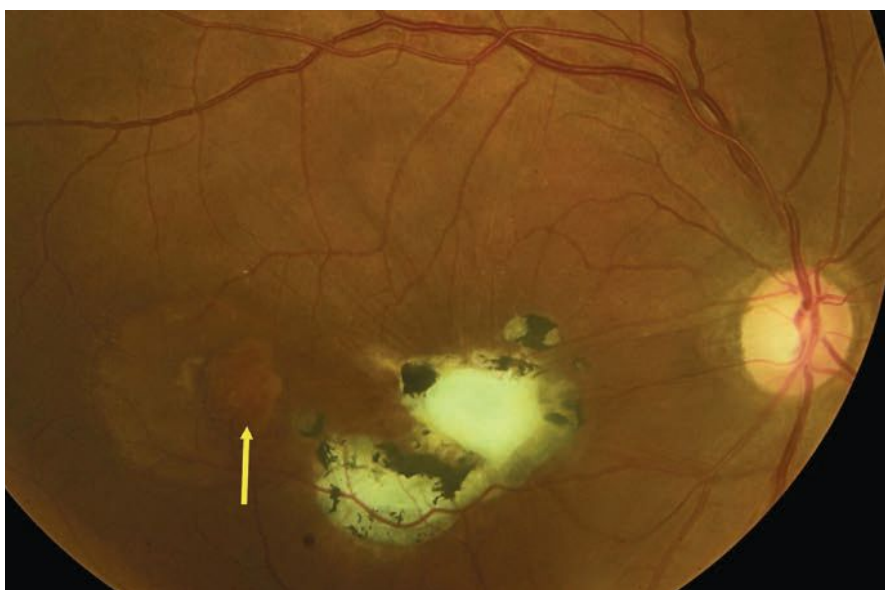

Fig. 1. Fundus photo of the right eye. What might that central fibrotic lesion represent?

Take the Retina Quiz

1. Which element of the patient's history could have caused this retinal finding?
a. Type 2 diabetes.
b. Stroke of the left hemisphere.
c. Car accident injury to the right side of his body.
d. Onset of floaters two years prior.

2. What does the orange lesion adjacent to the fibortic area represent?
a. A retinal detachment.
b. A subretinal hemorrhage.
c. A drusenoid PED.
d. A PED secondary to choroidal neovascularization (CNV).

3. What is the correct diagnosis for this fundus presentation?
a. Chorioretinal scar from toxoplasmosis.
b. CHRPE.
c. Choroidal rupture with secondary CNV.
d. Proliferative diabetic retinopathy.

4. Which of the following is not a treatment option for this condition?
a. Observation.
b. Injection of anti-VEGF agent.
c. Azithromycin PO.
d. Combination of A and B.

5. Which imaging technique would be helpful in order to get a definitive diagnosis?
a. B-scan ultrasound.
b. Fluorescein angiography/ OCT angiography.
c. Electroretinogram.
d. All of the above.

For answers, see page 98.

Discussion

There is an obvious chorioretinal scar involving the macula of right eye in our patient. In the absence of history, there are a lot of possible etiologies,

About Dr. Dunbar **Dr. Dunbar** is the director of optometric services and optometry residency supervisor at the Bascom Palmer Eye Institute at the University of Miami. He is a founding member of the Optometric Glaucoma Society and the Optometric Retina Society. Dr. Dunbar is a consultant for Carl Zeiss Meditec, Allergan, Regeneron and Genentech.

including an old toxoplasmosis scar which might be the immediate conclusion given the location and characteristic appearance. However, the history offers a critical clue to the actual etiology. Our patient describes having an motor vehicle accident four years prior that resulted in a sudden loss of his central vision. So, how does the clinical presentation fit into our patient having a traumatic event?

Ocular trauma can present in myriad ways depending on the type and location of the injury. Blunt force trauma can cause a contrecoup injury in which direct displacement and deformation of the globe can occur. In commotio retina, the hydraulic forces on the eye as a result of the injury can cause disruption and swelling of the photoreceptors, which can even lead to death of these cells.[1] The shock waves from the trauma coursing through the eye can result in hemorrhagic or non-hemorrhagic posterior vitreous detachments, retinal breaks and even retinal detachment, as well as traumatic optic neuropathy and choroidal rupture, to name a few.

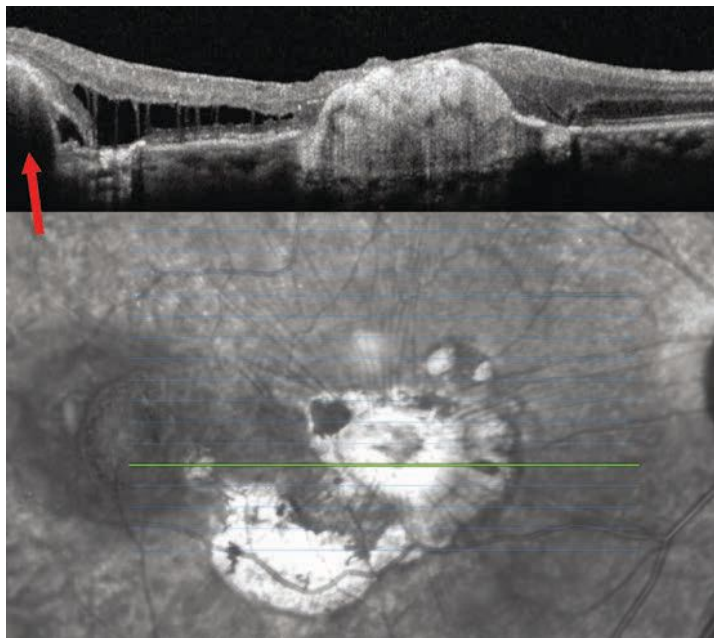

Fig. 2. OCT of the right eye. What could the area of RPE elevation possibly correspond to?

A choroidal rupture happens when there is disruption of the choriocapillaris, Bruch's membrane and the retinal pigment epithelium (RPE) from the direct result of the trauma or indirectly from the shock waves that are transferred through the vitreous and/or eye walls of the globe as a result of the trauma. When this occurs, stretching and folding of these structures leads to a break in Bruch's membrane and the RPE.

Choroidal ruptures are typically located in the posterior pole and most often concentric to the optic disc.[2] Sports injuries and car accidents involving the deployment of airbags are common causes, although systemic conditions such as pseudoxanthoma elasticum, Ehlers-Danlos syndrome, Paget's disease, sickle cell anemia or virtually any condition involving angioid streaks may predispose a patient to choroidal ruptures during an impact of even minimal intensity, as these represent breaks in Bruch's membrane as well.

In the acute phase, these breaks are commonly associated with subretinal and sub-RPE hemorrhages and may also be associated with other traumatic findings. In the chronic phase, CNV may develop and can be found at the edges of the rupture.[2] CNV may occur months to years after the traumatic event and can result in fibrotic scarring.[3]

The prognosis for choroidal rupture is highly dependent on location and whether it involves the foveal area, and also if subretinal or sub-RPE hemorrhages are present. Patients with choroidal rupture should be followed carefully for potential secondary CNV post-trauma, which may further decrease visual acuity.[3]

The treatment of choroidal rupture often involves observation and close follow-up to monitor for spontaneous improvement of vision, as there is no medical or surgical therapy indicated to treat acute episodes. Patients are often provided with an Amsler grid as a way to self-monitor for any changes in vision, as they carry the risk of developing CNV in the future. In cases where it develops, intraocular injections of anti-VEGF can improve visual outcomes.

Concluding Thoughts

No doubt the vision loss that our patient suffered is from his motor vehicle accident. The large chorioretinal scar involving the macula is due to a large choroidal rupture. Unfortunately, the rupture involves his macula, which explains the sudden loss of vision.

Regrettably, that's not the end of it. The OCT and clinical finding temporal to the macula are quite revealing. The orange area that was noted with the arrow is elevated and confirmed on the OCT scan where we can see an RPE detachment. There is also intra- and subretinal fluid adjacent to the RPE detachment, all consistent with an active CNV.

Our patient was referred to a retina specialist for possible treatment; ultimately, the MD decided to monitor him without treatment because of his poor visual prognosis. ■

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IOP Control: Time to Play Offense?

Interventional glaucoma allows optometrists to minimize the treatment burden for our patients.

BY MACKENZIE MACINTYRE, OD
SPARKS, NV

We are all too familiar with the constraints of the classic glaucoma treatment progression of drops-laser-surgery. Whether it is the cost of medications, compliance with administration of drugs or the need to tailor treatment to the stage of glaucoma, we routinely take a customized approach to this condition already. But there is an emerging, potentially disruptive treatment philosophy to consider: interventional glaucoma. It expands our arsenal of treatment options to further reduce the burden on our patients, allowing us to take earlier, more aggressive steps to treat glaucoma before it becomes uncontrollable.

In 2019, the interventional glaucoma philosophy received a major boost when the LiGHT trial outlined the benefits of selective laser trabeculoplasty (SLT) over topical meds as an initial treatment for glaucoma. This shift allows us to identify suboptimally controlled patients who are being treated with topical glaucoma medications and recommend reducing those agents in favor of early SLT, yielding improved ocular comfort and quality of vision, while also maintaining good glaucoma control.

The interventional glaucoma philosophy encourages a proactive approach and a more aggressive posture toward achieving IOP targets and disease control, relying on the numerous less invasive surgical options. We have become accustomed to thinking that minimally invasive glaucoma surgeries (MIGS) should occur at the time of cataract surgery; however, some experts are of the opinion that at least a few of these procedures could be considered as standalone treatment options as well in appropriately selected patients.

For a video of the procedure, read this article online at www.reviewofoptometry.com.

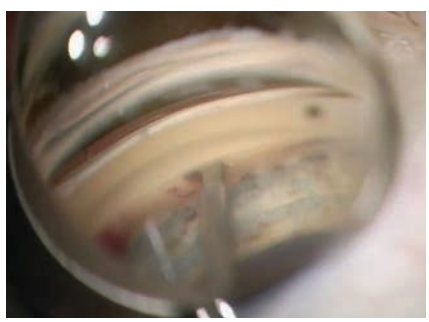

Photo: New World Medical

The Kahook Dual Blade excises a strip of trabecular meshwork to aid aqueous outflow.

A few approaches are especially well-suited to consideration when adopting an interventional glaucoma mindset:

• ***Durysta.*** In 2020, Allergan received FDA approval for this agent—a biodegradable, intracameral bimatoprost implant that can be administered either at the slit lamp or in the operating room during the time of cataract surgery.

• ***Omni.*** This MIGS technique, from Sight Sciences, combines a canaloplasty with a trabeculotomy and can be performed with cataract surgery or in pseudophakic patients as an alternative to filtering surgery.

• ***Trabectome (Microsurgical Technology).*** This device creates a partial trabeculotomy, cauterizing and aspirating the nasal trabecular meshwork tissue roughly 90 to 180 degrees.

• ***Kahook Dual Blade (New World Medical).*** Similar to the Trabectome procedure, this method also removes tissue from the trabecular meshwork; however, it uses a dual-blade scalpel rather than cautery.

• ***Ab interno canaloplasty (iTrack, Ellex).*** Viscodilation of Schlemm's canal is the method here, with the added advantage of breaking adhesions in the trabecular meshwork and irrigation of the collector channels.

We know that better IOP control in earlier stages of glaucoma decreases the risk of vision loss. Therefore, it is our responsibility to reach out to our ophthalmological colleagues and recommend less invasive, cost-saving and likely more efficient and effective treatment options for our patients. ■

CANDIDATES FOR INTERVENTIONAL GLAUCOMA PROCEDURES

Cataract Patients:

- POAG patient on two drops with blurry vision
- IOP has increased the past three months due to noncompliance with regimen
- Candidate for cataract surgery

Pseudophakic Patients:

- Pseudophakic POAG patient on two drops
- Underwent SLT two years ago
- Visual fields reflect evidence of progression
- Needs an additional drop

ABOUT THE AUTHOR

Dr. Macintyre is an optometrist at Eye Care Associates of Nevada. He specializes in cataract surgery comanagement and perioperative care of cataract patients. He has no financial interests to disclose.

About Drs. Cunningham and Whitley

Dr. Cunningham is the director of optometry at Dell Laser Consultants in Austin, TX. He has no financial interests to disclose. **Dr. Whitley** is the director of professional relations and residency program supervisor at Virginia Eye Consultants in Norfolk, VA. He is a consultant for Alcon.

PRODUCT REVIEW

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▶ DIAGNOSTICS

Genetic Test Assesses Risk of Keratoconus

Now that collagen crosslinking has proved its mettle in slowing keratoconus progression, doctors need to identify candidates right away so that the option can be considered early. Those looking for help may wish to offer keratoconus suspects a genetic test that will assess their risk. AvaGen, by Avellino, examines 75 keratoconus-related genes, more than 2,000 gene variants and data on ethnic predispositions to the disease to come up with a keratoconus genetic risk score, a company press release explains. These results will allow you to offer more customized care to your patients based on their individual odds of developing keratoconus, the company suggests.

The test can also measure susceptibility to a variety of corneal dystrophies (*e.g.*, epithelial basement membrane, granular, lattice, Reis-Bucklers, Schnyder, Theill-Behnke), allowing for more conclusive diagnoses and more effective treatment plans, Avellino says. The test results may also influence your decisions about a patient's viability for refractive surgery given that some options are contraindicated in patients with certain corneal dystrophies, the company notes.

An in-office cheek swab yields a sample that the practice sends to Avellino's lab for analysis; the company says results should arrive in a few days. To ensure that the doctor and patient both understand the results, the company also provides genetic counseling, the press release explains.

Big Screen Camera Handles Small Pupil Patients

A new retinal camera introduced by Coburn Technologies may be just what your practice needs to take your imaging capabilities to the next level. The HFC-1 non-mydriatic fundus camera uses automated pupil detection and eye tracking to simplify image capture while producing sharp and reliable retinal photos, the company says. Fixation targets can also be set manually for greater flexibility in recording the desired images.

The camera is able to quickly shift between its five imaging modes—color, blue, red, red-free and cobalt—while adapting to varying pupil sizes, saving you the time of making manual adjustments during an exam. The device works even with pupils as small as 3.3mm, company literature explains. Its 20-megapixel sensor reduces motion artifacts and captures images large enough to allow for closer examination of fine details, according to Coburn.

The company says the range of imaging modes makes the device versatile enough to document glaucoma (including fine detail of the RNFL using the cobalt filter), macular edema, epiretinal membranes, diabetic retinopathy, pigmentary abnormalities and much more.

The DICOM-compatible HFC-1 also features a built-in PC with web browsing capabilities and an LCD touch screen to enable image analysis and sharing from the same device, the company points out.

▶ CONTACT LENSES

New Ortho-K Option from J&J on the Way

The recent announcement that Johnson & Johnson Vision's first product for myopia management is an ortho-K lens brings renewed attention—and the only on-label indication—to this longstanding modality. The company says its Acuvue Abiliti Overnight contact lenses have been shown to reduce axial elongation in myopic children by 0.28mm on average over a two-year period.

The lens will be available in spherical and toric options. Practitioners will use custom software that draws on corneal topography, refractive error and other data to create a lens fit that temporarily reshapes the cornea during overnight wear, a press release explains.

Details of the lens design are not yet available, but studies cited in the press release reference clinical trials conducted using the Menicon Z Night ortho-K lens. The company previously announced a collaboration with Menicon and says this new lens is part of that effort.

J&J also promised "additional products and services to address the progression of myopia" down the road.

Acuvue Abiliti Overnight will be available by the end of the year, according to the company.

More Options for Astigmats

If you like fitting the Biofinity XR toric from CooperVision but sometimes find a lens power isn't available, take heart: the company says it has nearly doubled the prescription options. The lens can now be ordered in sphere powers from -20.00D to +20.00D, a press release explains. Cylinder powers vary based on sphere, but options begin at -0.75D and go up to -5.75D for some lenses. Axis options are available in 5° increments, the press release notes. ■

Contact Lenses

Practice For Sale

Equipment & Supplies

BY ANDREW S. GURWOOD, OD

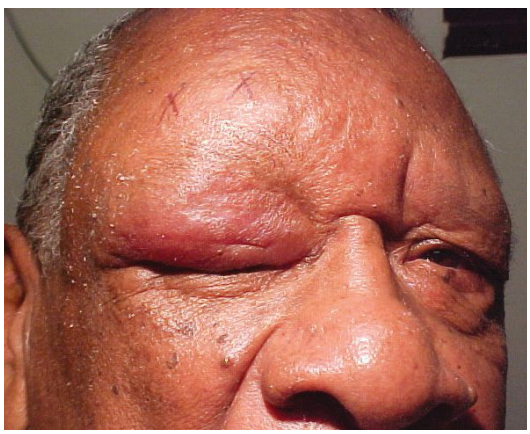
DIAGNOSTIC QUIZ

The Plot Thickens

When a patient's clinical outlook changes radically, so too should your approach to differential diagnosis.

A 69-year-old Black male presented for a routine eye exam with a nebulous complaint of ocular discomfort and itching, OD>OS of one month's duration. He reported no visual loss, pain or diplopia.

His pertinent medical history included reports of hypertension and gout. Current medications included clopidogrel (Plavix, Bristol-Myers Squibb), hydrochlorothiazide and aspirin. The patient denied allergies of any kind.



What seemed like a routine case of contact dermatitis actually turned out to be far less commonplace.

Diagnostic Data

Ocular examination revealed best-corrected visual acuities to be 20/20 OU through -1.00/+2.50 DS spectacles. External evaluation uncovered normal motilities and visual fields, normal color and stereo with no evidence of afferent pupillary defect. Refraction was negligibly different between eyes.

The pertinent anterior segment finding OD is demonstrated in the photograph.

Goldmann applanation tonometry was 15mm Hg. Dilated fundus examination revealed normal and quiet posterior segments with no peripheral pathology OU.

Additional Testing

Other efforts might include palpation of the region to ensure the absence of cellulitis. Also, topical sodium fluorescein could be used to assess the corneal surface for damage and to evaluate the status of the lacrimal lake.

Back for More

The patient was diagnosed with acute contact dermatitis and allergic blepharoconjunctivitis OD. He was educated to remove possible triggers, use supportive measures (*e.g.*, artificial tears, cold compresses) and was prescribed a topical mast cell stabilizing/antihistamine agent. He was scheduled to return for follow-up in five to 14 days.

The patient did not return for his scheduled follow-up but did come in within one month with a recurrent acute episode. His chief complaint at the return visit was worsening eyelid edema OD and onset of horizontal diplopia accompanied by constant mild pain behind the right eye.

Forced duction testing was positive OD, suggesting a restrictive etiology. Exophthalmometry (base 103) measured 25mm OD and 21mm OS. There was a 3mm ptosis OD produced by the weight of the edematous lid tissue. There was no afferent defect. Slit lamp exam confirmed upper lid edema and revealed possible lacrimal gland enlargement OD. Mild bulbar conjunctival injection OD was also observed. Posterior segment findings remained unchanged.

Your Diagnosis

What would be your diagnosis in this case? To find out, read the online version at www.reviewofoptometry.com. ■

About Dr. Gurwood Dr. Gurwood is a professor of clinical sciences at The Eye Institute of the Pennsylvania College of Optometry at Salus University. He is a co-chief of Primary Care Suite 3. He is attending medical staff in the department of ophthalmology at Albert Einstein Medical Center, Philadelphia. He has no financial interests to disclose.

Retina Quiz Answers (from page 92)—Q1: c, Q2: d, Q3: c, Q4: c, Q5: b

NEXT MONTH IN THE MAG

In July, we present our annual glaucoma report. Articles will include:

- How to Make Sense of OCT Scans for Glaucoma
- Beware These Diagnostic Pitfalls in Glaucoma
- Optic Nerve Head Dynamics in Glaucoma and Beyond
- Comanagement Series: Keep Glaucoma Care Close to Home

Also included in July:

- The Results-oriented Neuro Work-up
- Cataract Q&A: Expert Answers to Common Dilemmas
- The Optometric Workforce: Changes and Challenges

BAUSCH + LOMB ULTRA® MULTIFOCAL FOR ASTIGMATISM

FIT FOR SUCCESS

The results are in. Eye Care Professionals gave **Bausch + Lomb ULTRA® Multifocal for Astigmatism** high marks across the board thanks to its easy fitting process.

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agreed that it is easy to get a **successful** fit during the **first visit** with this lens.*

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80% agreed that it took **no more than 5 minutes longer** to fit this lens compared to toric lenses.†

95%

agreed that this lens allows them to **fit patients who have previously dropped out** of contact lenses.‡

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*Results of an online survey with Eye Care Professionals who completed an evaluation program for Bausch + Lomb ULTRA® Multifocal for Astigmatism contact lenses (n=219). Survey results include Eye Care Professionals who reported that successful fitting was extremely easy, easy, or somewhat easy (on a 6-point scale) during the first visit, with a margin of error ±4.1%.

†Results of an online survey with Eye Care Professionals who completed an evaluation program for Bausch + Lomb ULTRA® Multifocal for Astigmatism contact lenses (n=219). Survey results include Eye Care Professionals who reported that it took less chair time, no extra chair time, 1-2 minutes of additional chair time, or 3-5 minutes of additional chair time to fit Bausch + Lomb ULTRA® Multifocal for Astigmatism with a margin of error ±5.3%.

‡Results of an online survey with Eye Care Professionals who completed an evaluation program for Bausch + Lomb ULTRA® Multifocal for Astigmatism contact lenses (n=219). Survey results include Eye Care Professionals who strongly agreed, agreed, or slightly agreed (on a 6-point agreement scale) with the surveyed statement, with a margin of error ±2.8%.

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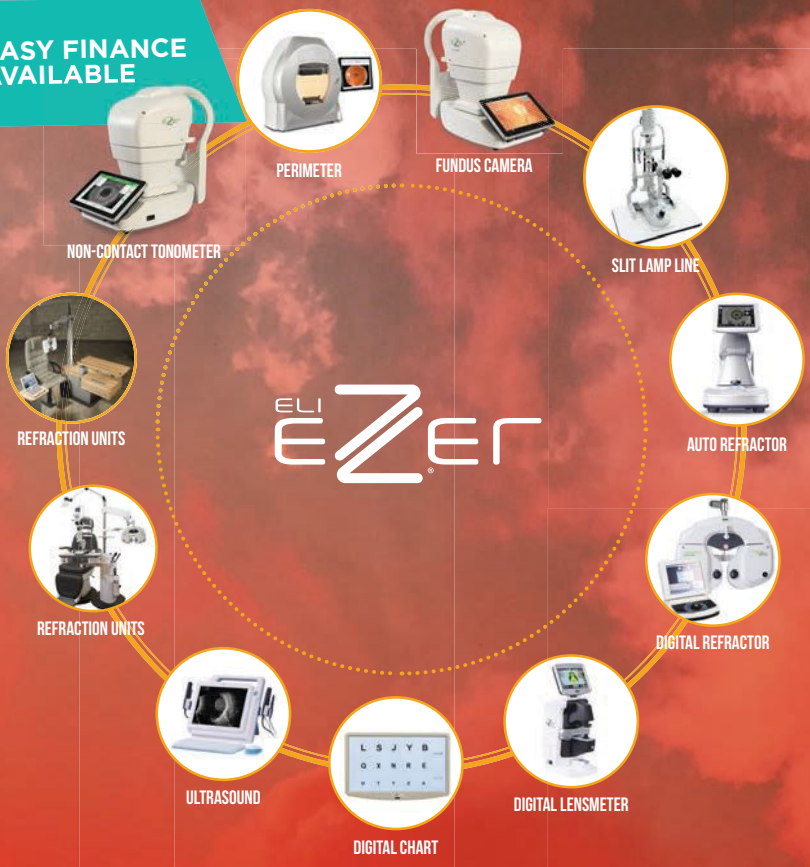

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