Lifelong Contact Lens Success: ***Keep Allergy and Dry Eye at Bay,*** *Page 48*

REVIEW® OF OPTOMETRY

June 15, 2017

www.reviewofoptometry.com

Can an Eye Drop Eliminate PRESBYOPIA?

New therapies under investigation have the potential to radically alter your approach to this age-old problem. ***Page 42***

8TH ANNUAL RETINA REPORT

- Diabetes Care in the Age of Anti-VEGF, *Page 56*
- Advanced Imaging Techniques for Choroidal Disease, *Page 62*
- Case Report: Acute Syphilitic Posterior Placoid Chorioretinitis, *Page 70*
- AMD Mimickers: When to Suspect Macular Dystrophy, *Page 81*

INSIDE – EARN 2 FREE CE CREDITS: Advanced Refractive Solutions for Today's Presbyopic Patient *Page 34*

EYE-INSPIRED™
PATIENT-FOCUSED

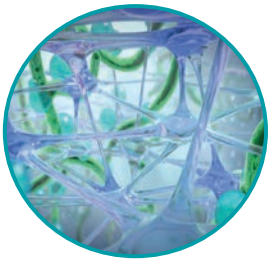
INDUSTRY LEADING UV BLOCKING†

Because I know Eric leaves no detail unchecked, I prescribe NEW ACUVUE OASYS® 1-Day for ASTIGMATISM.

*Helps protect against transmission of harmful UV radiation to the cornea and into the eye.

†WARNING: UV-absorbing contact lenses are NOT substitutes for protective UV-absorbing eyewear such as UV-absorbing goggles or sunglasses because they do not completely cover the eye and surrounding area. You should continue to use UV-absorbing eyewear as directed. NOTE: Long-term exposure to UV radiation is one of the risk factors associated with cataracts. Exposure is based on a number of factors such as environmental conditions (altitude, geography, cloud cover) and personal factors (extent and nature of outdoor activities). UV-blocking contact lenses help provide protection against harmful UV radiation. However, clinical studies have not been done to demonstrate that wearing UV-blocking contact lenses reduces the risk of developing cataracts or other eye disorders. Consult your eye care practitioner for more information.

Two innovations combined for stable vision and exceptional comfort.



HydraLuxe™ Technology

Tear-like molecules and highly breathable hydrated silicone integrate with the patient's own tear film.

BLINK STABILIZED® Design

This design works naturally with the eyelids, helping to keep the lens in the correct position.

For your practice...

99%

FIRST-FIT SUCCESS[1]
for great practice efficiency

For your patients...

ACHIEVE

20/20

VISUAL ACUITY OR BETTER‡[1]

To learn more about this and other EYE-INSPIRED™ PATIENT-FOCUSED innovations, visit acuvueprofessional.com

From the contact lens family **unbeaten on comfort***

NEW ACUVUE® oasys 1-Day WITH HydraLuxe™ BRAND CONTACT LENSES ASTIGMATISM

* In 15 clinical trials posted on www.clinicaltrials.gov, a website maintained by the NIH. The 15 clinical studies evaluated subjective comfort as a primary or secondary endpoint for ACUVUE OASYS® Brand with HYDRACLEAR® PLUS Technology. Review conducted as of December 31, 2016.

‡ In a clinical trial, 97% of patients achieved a monocular VA 20/20 or better at the fitting visit with 100% achieving 20/25 or better.

1. Straker B, Hamada W, Sulley A, Olivares G. Fitting performance and efficiency with a low silicone hydrogel daily disposable toric contact lens. Poster presented at: GSLS Conference 2017.

ACUVUE® Brand Contact Lenses are indicated for vision correction. As with any contact lens, eye problems, including corneal ulcers, can develop. Some wearers may experience mild irritation, itching or discomfort. Lenses should not be prescribed if patients have any eye infection, or experience eye discomfort, excessive tearing, vision changes, redness or other eye problems. Consult the package insert for complete information. Complete information is also available by visiting acuvueprofessional.com, or by calling Johnson & Johnson Vision Care, Inc. at 1-800-843-2020.

VOL. 154 NO. 6 ■ JUNE 15, 2017

IN THE NEWS

Bausch + Lomb recently announced **updated results** from the **ARMOR surveillance study**, including preliminary 2016 data on **antibiotic resistance levels** and an eight-year trend analysis of antibiotic resistance among staphylococcal isolates. They found non-susceptibility to fluoroquinolones more than doubled from 2015, and methicillin resistance is decreasing among *S. aureus*, but not among coagulase-negative staphylococci. While resistance is decreasing, **resistance to several commonly used antibiotics is still a challenge**, according to Penny Asbell, MD, lead author.

Bausch + Lomb Reports Updated Results of the Antibiotic Resistance Monitoring in Ocular MicRoorganisms (ARMOR) Study. May 10, 2017. www.bausch.com/our-company/recent-news/id/2374/5102017-Wednesday. Accessed May 16, 2017.

The Optometric Glaucoma Society recently established the **Optometric Glaucoma Foundation (OGF)** to support glaucoma education for the optometric profession, including students, residents, educators and practitioners. The 501(c)(3) not-for-profit organization is designed to promote excellence in the care of glaucoma patients, support research and help optometrists become involved in research. The OGF is led by Murray Fingeret, OD, president; Leo Semes, OD, vice president; John McSoley, OD, secretary; and Austin Lifferth, OD, treasurer.

Researchers have found that roughly 15% of **Ebola survivors** in Sierra Leone who had previously reported ocular symptoms have a **retinal scar that seems unique to the disease.** Researchers now speculate the virus enters the eye through the optic nerve to reach the retina, similarly to West Nile Virus.

Steptoe PJ, Scott JT, Baxter JM, et al. Novel retinal lesion in Ebola survivors, Sierra Leone, 2016. Emerging Infectious Diseases. 2017;23(7). [Epub ahead of print].

Systemic Therapy vs. Implant for Uveitis

Long-term follow up reveals systemic therapy may be better for chronic uveitis patients.

By Rebecca Hepp, Managing Editor

Patients with chronic uveitis may consider systemic therapy with corticosteroids and immunosuppressants in lieu of a long-term corticosteroid intraocular implant, according to new research. The study, funded by the National Eye Institute, found that, after seven years of treatment, patients on systemic therapy had stable visual acuity, while those with the implant saw a decline of roughly six letters.

The study looked at 255 patients with uveitis randomly assigned either a fluocinolone intraocular implant, or systemic therapy consisting of prednisone and immunosuppressants such as methotrexate or mycophenolate mofetil. While visual acuity remained about the same in the two groups through two years, researchers noted reactivations of uveitis after roughly five years in the implant-treated eyes. This coincided with a decline in visual acuity, which the researchers speculate may be due to increased damage in the retina and choroid.

In addition to the long-term changes in treatment efficacy, patients with the implant were more likely to experience negative ocular effects, such as cataracts, elevated intraocular pressure and glaucoma. Patients receiving systemic therapy, although they had an increased risk of needing treatment with antibiotics, did not have large increases in the risk of adverse effects common with systemic corticosteroids such as high blood pressure or diabetes.

"This study now clearly states that systemic oral therapy is just as good, and in fact better, than a steroid implant," says Nathan Lighthizer, OD, an associate professor at Oklahoma College of Optometry. "This may save the patient another visit or a referral to a specialist since optometrists in some states can prescribe oral steroids."

But Dr. Lighthizer still recommends ODs consult with an ophthalmologist or uveitis specialist for some refractory cases, which may lead to a team-based care approach for some patients. "It is good to know that these specialists now may be more inclined to treat with systemic therapy rather than surgery, and we may need to participate in the comanagement of these patients and potentially follow them long-term."

"Anytime a surgical implant into the eye can be avoided, it's potentially a good thing, especially when oral therapy in this study proved to preserve more vision, have fewer long-term side effects and was more cost-effective," Dr. Lighthizer adds. "That is a win all around."

The leading cause of ocular discomfort and contact lens dropout is dryness.

Meibomian Gland Function, the foundation of a healthy tear film.

Gland structure captured with Dynamic Meibomian Imaging (DMI).

"Evaluation of meibomian glands is now standard in my patient workups"

Steven J. Ferguson, OD
Dunes Eye Consultants

LipiScan™

MKT-ADV 019 Rev A

tearscience.com | 919-459-4880 | sales@tearscience.com

Bill Leaves Essential Vision Benefits Uncertain

The American Health Care Act (AHCA) that passed the House on May 4 is facing criticism from patient groups such as the American Medical Association and the American Association of Retired Persons for potentially rolling back popular health care benefits.[1,2] For optometry specifically, care for the youngest patients may soon be in flux.

As it currently stands, the bill provides an option for states to independently decide whether to maintain, suspend or make changes to the essential benefits previously protected by the Affordable Care Act (ACA)—including pediatric vision care.[3] The change is an attempt to "encourage fair health insurance premiums," according to the bill.[3]

When the ACA was passed in 2010, pediatric vision was included as one of the 10 essential benefits that must be covered by all providers.[4] That coverage included a yearly eye exam with a materials benefit for every patient younger than 19.[5]

The legislation passed the House after an amendment written by Rep. Tom MacArthur (NJ) and Rep. Mark Meadows (NC) resolved intraparty disagreements.[3] The bill still faces Senate scrutiny and is now in limbo after a Congressional Budget Office report suggested 24 million could lose coverage.[5]

1. Gurman A. AMA statement on cbo score of american health care act. American Medical Association. May 24, 2017. www.ama-assn.org/ama-statement-cbo-score-american-health-care-act. Accessed May 30, 2017.
2. Frank D. Health care bill endangers coverage. AARP. May 24, 2017. www.aarp.org/politics-society/advocacy/info-2017/aarp-response-cbo-score-health-care-bill-fd.html. Accessed May 30, 2017.
3. MacArthur T. Amendment drafted to H.R. 1628. April 24, 2017. www.politico.com/f/?id=0000015b-a790-d120-addb-f7dc0ec90000. Accessed May 30, 2017.
4. American Optometric Association. AOA's Frequently Asked Questions on the Essential Health Benefit and Insurance Marketplaces. www.aoa.org/Documents/advocacy/FAQ_on_EHB.PDF. Accessed May 24, 2017.
5. Congressional Budget Office. American Health Care Act. www.cbo.gov/publication/52486. March 13, 2017. Accessed May 27, 2017.

A Legislative Win in Georgia

Optometrists in Georgia can now perform certain injections, thanks to the passage of SB 153, which was signed into law by Governor Nathan Deal on May 9, 2017. ODs ready to take on the scope of practice expansion must first attend a 30-hour injectables training program approved by the board and be under the direct supervision of a board-certified ophthalmologist, according to the bill. The bill also includes a provision allowing optometrists to treat ocular pain with non-narcotic oral analgesics, hydrocodone administered orally and Schedule III or Schedule IV oral analgesics.

Georgia General Assembly. 017-2018 Regular Session - SB 153. www.legis.ga.gov/Legislation/en-US/display/20172018/SB/153. Accessed June 2, 2017.

Get Ready For DEWS II

Attendees at this year's Association for Research in Vision and Ophthalmology (ARVO) Annual Meeting, held in Baltimore from May 6-11, were the first to get a glimpse of The Tear Film & Ocular Surface Society's (TFOS) forthcoming Dry Eye Workshop II (DEWS II) recommendations. The full report sets out to update the definition, classification and diagnosis of dry eye, as well as evaluate its impact, address management and therapy and develop recommendations for clinical trials to better assess treatment options.[1]

DEWS II took two years to complete and involved 150 experts from around the world, who used an evidence-based approach to increase our understanding of dry eye disease, said organizer David Sullivan, PhD, in a press release.[1]

While the report's updated definition for dry eye—which adds a focus on homeostasis—is a significant change today, its call for better research on treatment outcomes is a welcome addition for the future, according to Lyndon Jones, OD, chair of the DEWS II Management and Therapy Subcommittee and a professor at the Centre for Contact Lens Research, School of Optometry and Vision Science, University of Waterloo.

"What really shocked us was what little high-level evidence there is to support many of the things we do or we prescribe on a day-to-day basis," said Dr. Jones at the ARVO briefing. "And even when we have decided whether the patient has aqueous deficient or evaporative dry eye, how we then tailor that management and therapy to specifically treat those two just simply is missing. We need more evidence. We knew [dry eye treatment] was complex before, and what we really need is a huge number of new studies." Noting that a decade has passed since the first DEWS, Dr. Jones joked that "the good thing is, we'll be busy for the *next* decade."

After two years of work, DEWS II will be published July 1 by *The Ocular Surface* and will be available at www.tearfilm.org, according to the ARVO presentation.

1. Tear Film & Ocular Surface Society. www.tearfilm.org/dettnews-tfos_dews_ii_report_announced/101_16/eng. Accessed May 30, 2017.

Introducing the Keeler Z Series Slit Lamp & Digital Tonometer

Keeler Optics...says it all.

The Z Series Slit Lamp is the latest line from Keeler featuring legendary Keeler optics housed in a stylish, contemporary design.

Your choice of 3 or 5 step magnification option in a standard, digital ready, or comprehensive digital capture system.

Check out some of these great features:

- Converging or parallel binoculars
- 14mm Slit length / maximum aperture
- X 6 to x 40 (5 step) or X 10 to X 25 (3 step) magnifications
- Blue, red-free, clear, neutral density and diffuser filters
- 360 degree continuous slit rotation
- Integrated yellow barrier filter
- 1m Square aperture for Uveitis assessment
- LED illumination
- Illumination control mounted next to joystick
- 3 year warranty

Order now and receive a limited edition bronze D-KAT tonometer.

Contact your preferred Keeler distributor for details.

Keeler

Learn more at www.keelerusa.com

Keeler Instruments, Inc. • 3222 Phoenixville Pike #50 • Malvern, PA 19008
Tel: (800) 523-5620 • Fax: (610) 353-7814 • email: keeler@keelerusa.com

Cancer Drug Combats Thyroid Eye Disease

A drug targeting the insulin-like growth factor 1 receptor may help reduce symptoms associated with thyroid eye disease, according to a new report.[1]

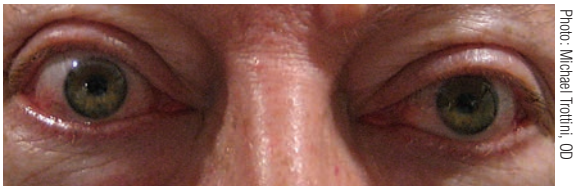


Photo: Michael Trotfini, OD

This patient exhibits proptosis from thyroid orbitopathy.

Teprotumumab, originally tested as a cancer treatment, was investigated in a multicenter study for its ability to reduce the severity of thyroid-associated ophthalmopathy.[1] Researchers randomly assigned 87 patients, diagnosed nine months or less prior to the onset of symptoms, to receive placebo or intravenous teprotumumab once every three weeks, over a 24-week period, or for eight injections total.[1]

The study defined a response as a reduction of two points on a seven-point clinical activity score and a reduction of at least two millimeters in proptosis at the end of week 24.[1]

In patients who received teprotumumab, 69% achieved a response vs. 20% of those who received placebo at week 24, and 43% of patients in the teprotumumab group achieved relief of their symptoms within six weeks.[1] The drug was also well tolerated, and the hyperglycemia some patients experienced was well-controlled after adjusting the dosage.[1]

However, only patients recently diagnosed with active, moderate to severe disease were enrolled in the study, so further investigation is needed to asses teprotumumab's ability to provide relief in patients with stable or milder forms of the disease. Also, orbital imaging was not performed to determine the specific tissues affected by teprotumumab therapy. A one-year follow up is currently underway to determine the drug's long-term therapeutic effect.

"The results of the study are quite impressive—there is a marked clinical difference in response in the group receiving the active drug compared with placebo, and the onset of improvement was rapid," says Tammy Than, OD, a professor at the University of Alabama at Birmingham School of Optometry. "FDA was impressed as well, as last year it granted this drug 'breakthrough therapy' designation."

However, a few questions remain, and Dr. Than hopes further research will help uncover whether regression of clinical improvement occurs once the infusions stop.

The researchers say teprotumumab may also help patients with other autoimmune conditions with ocular manifestations.[1]

"Since other applications may exist for teprotumumab in rheumatoid arthritis and other autoimmune diseases, this drug may offer an avenue of high impact for managing numerous diseases that manifest with debilitating ocular sequelae," Dr. Than concludes. ■

1. Smith TJ, Kahaly GJ, Ezra DG, et al. Teprotumumab for thyroid-associated ophthalmopathy. N Eng J Med. 2017;376(18):1748.

REVIEW® OF OPTOMETRY

BUSINESS OFFICES
11 Campus Blvd., Suite 100
Newtown Square, PA 19073

CEO, INFORMATION SERVICES GROUP
Marc Ferrara
(212) 274-7062 • mferrara@jobson.com

PUBLISHER
James Henne
(610) 492-1017 • jhenne@jobson.com

REGIONAL SALES MANAGER
Michele Barrett
(610) 492-1014 • mbarrett@jobson.com

REGIONAL SALES MANAGER
Michael Hoster
(610) 492-1028 • mhoster@jobson.com

VICE PRESIDENT, OPERATIONS
Casey Foster
(610) 492-1007 • cfoster@jobson.com

VICE PRESIDENT, CLINICAL CONTENT
Paul M. Karpecki, OD, FAAO
pkarpecki@jobson.com

PRODUCTION MANAGER
Scott Tobin
(610) 492-1011 • stobin@jobson.com

SENIOR CIRCULATION MANAGER
Hamilton Maher
(212) 219-7870 • hmaher@jhihealth.com

CLASSIFIED ADVERTISING
(888) 498-1460

SUBSCRIPTIONS
$56 a year, $88 (US) in Canada,
$209 (US) in all other countries.

SUBSCRIPTION INQUIRIES
(877) 529-1746 (US only)
Outside US Call: (845) 267-3065

CIRCULATION
PO Box 81
Congers, NY 10920
Tel: (Toll Free): (877) 529-1746
Outside US: (845) 267-3065

jmi

CEO, INFORMATION SERVICES GROUP
Marc Ferrara

SENIOR VICE PRESIDENT, OPERATIONS
Jeff Levitz

VICE PRESIDENT, HUMAN RESOURCES
Tammy Garcia

VICE PRESIDENT, CREATIVE SERVICES & PRODUCTION
Monica Tettamanzi

CORPORATE PRODUCTION DIRECTOR
John Anthony Caggiano

VICE PRESIDENT, CIRCULATION
Emelda Barea

Down, Boy.

Help Tame Postoperative Ocular Inflammation and Pain With LOTEMAX® GEL

Indication

LOTEMAX® GEL (loteprednol etabonate ophthalmic gel) 0.5% is indicated for the treatment of post-operative inflammation and pain following ocular surgery.

Important Safety Information about LOTEMAX® GEL

- LOTEMAX® GEL is contraindicated in most viral diseases of the cornea and conjunctiva including epithelial herpes simplex keratitis (dendritic keratitis), vaccinia, and varicella, and also in mycobacterial infection of the eye and fungal diseases of ocular structures.
- Prolonged use of corticosteroids may result in glaucoma with damage to the optic nerve, defects in visual acuity and fields of vision. If this product is used for 10 days or longer, IOP should be monitored.
- Use of corticosteroids may result in posterior subcapsular cataract formation.
- Use of steroids after cataract surgery may delay healing and increase the incidence of bleb formation and occurrence of perforations in those with diseases causing corneal and scleral thinning. The initial prescription and renewal of the medication order should be made by a physician only after examination of the patient with the aid of magnification, and where appropriate, fluorescein staining.
- Prolonged use of corticosteroids may suppress the host response and thus increase the hazard of secondary ocular infection. In acute purulent conditions, steroids may mask infection or enhance existing infection.
- Use of a corticosteroid medication in the treatment of patients with a history of herpes simplex requires great caution. Use of ocular steroids may prolong the course and exacerbate the severity of many viral infections of the eye (including herpes simplex).
- Fungal infections of the cornea are particularly prone to develop coincidentally with long-term local steroid application. Fungus invasion must be considered in any persistent corneal ulceration where a steroid has been used or is in use.
- Patients should not wear contact lenses when using LOTEMAX® GEL.
- The most common ocular adverse drug reactions reported were anterior chamber inflammation (5%), eye pain (2%) and foreign body sensation (2%).

Please see brief summary of Prescribing Information on adjacent page.

LOTEMAX® GEL
loteprednol etabonate
ophthalmic gel 0.5%

BAUSCH + LOMB

BRIEF SUMMARY OF PRESCRIBING INFORMATION

This Brief Summary does not include all the information needed to prescribe Lotemax Gel safely and effectively. See full prescribing information for Lotemax Gel.

Lotemax (loteprednol etabonate ophthalmic gel) 0.5%

Rx only
Initial Rx Approval: 1998

INDICATIONS AND USAGE

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

DOSAGE AND ADMINISTRATION

Invert closed bottle and shake once to fill tip before instilling drops.

Apply one to two drops of LOTEMAX into the conjunctival sac of the affected eye four times daily beginning the day after surgery and continuing throughout the first 2 weeks of the post-operative period.

CONTRAINDICATIONS

LOTEMAX, 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

Intraocular Pressure (IOP) Increase

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

Cataracts

Use of corticosteroids may result in posterior subcapsular cataract formation.

Delayed Healing

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

Bacterial Infections

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

Viral Infections

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

Fungal Infections

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

Contact Lens Wear

Patients should not wear contact lenses during their course of therapy with LOTEMAX.

ADVERSE REACTIONS

Adverse reactions associated with ophthalmic steroids include elevated intraocular pressure, which may be associated with infrequent optic nerve damage, visual acuity and field defects, posterior subcapsular cataract formation, delayed wound healing and secondary ocular infection from pathogens including herpes simplex, and perforation of the globe where there is thinning of the cornea or sclera.

The most common adverse drug reactions reported were anterior chamber inflammation (5%), eye pain (2%), and foreign body sensation (2%).

USE IN SPECIFIC POPULATIONS

Pregnancy

Teratogenic Effects

Loteprednol etabonate has been shown to be embryotoxic (delayed ossification) and teratogenic (increased incidence of meningocele, abnormal left common carotid artery, and limb flexures) when administered orally to rabbits during organogenesis at a dose of 3 mg/kg/day (35 times the maximum daily clinical dose), a dose which caused no maternal toxicity. The no-observed-effect-level (NOEL) for these effects was 0.5 mg/kg/day (6 times the maximum daily clinical dose). Oral treatment of rats during organogenesis resulted in teratogenicity (absent innominate artery at ≥5 mg/kg/day doses, and cleft palate and umbilical hernia at ≥50 mg/kg/day) and embryotoxicity (increased post-implantation losses at 100 mg/kg/day and decreased fetal body weight and skeletal ossification with ≥50 mg/kg/day). Treatment of rats with 0.5 mg/kg/day (6 times the maximum clinical dose) during organogenesis did not result in any reproductive toxicity. Loteprednol etabonate was maternally toxic (significantly reduced body weight gain during treatment) when administered to pregnant rats during organogenesis at doses of ≥5 mg/kg/day.

Oral exposure of female rats to 50 mg/kg/day of loteprednol etabonate from the start of the fetal period through the end of lactation, a maternally toxic treatment regimen (significantly decreased body weight gain), gave rise to decreased growth and survival, and retarded development in the offspring during lactation; the NOEL for these effects was 5 mg/kg/day. Loteprednol etabonate had no effect on the duration of gestation or parturition when administered orally to pregnant rats at doses up to 50 mg/kg/day during the fetal period.

There are no adequate and well controlled studies in pregnant women. LOTEMAX should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers

It is not known whether topical ophthalmic administration of corticosteroids could result in sufficient systemic absorption to produce detectable quantities in human milk. Systemic steroids appear in human milk and could suppress growth, interfere with endogenous corticosteroid production, or cause other untoward effects. Caution should be exercised when LOTEMAX is administered to a nursing woman.

Pediatric Use

Safety and effectiveness in pediatric patients have not been established.

Geriatric Use

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

NONCLINICAL TOXICOLOGY

Carcinogenesis, Mutagenesis, Impairment Of Fertility

Long-term animal studies have not been conducted to evaluate the carcinogenic potential of loteprednol etabonate. Loteprednol etabonate was not genotoxic *in vitro* in the Ames test, the mouse lymphoma tk assay, or in a chromosome aberration test in human lymphocytes, or *in vivo* in the single dose mouse micronucleus assay. Treatment of male and female rats with up to 50 mg/kg/day and 25 mg/kg/day of loteprednol etabonate, respectively, (600 and 300 times the maximum clinical dose, respectively) prior to and during mating did not impair fertility in either gender.

PATIENT COUNSELING INFORMATION

Administration

Invert closed bottle and shake once to fill tip before instilling drops.

Risk of Contamination

Patients should be advised not to allow the dropper tip to touch any surface, as this may contaminate the gel.

Contact Lens Wear

Patients should be advised not to wear contact lenses when using LOTEMAX.

Risk of Secondary Infection

If pain develops, redness, itching or inflammation becomes aggravated, the patient should be advised to consult a physician.

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

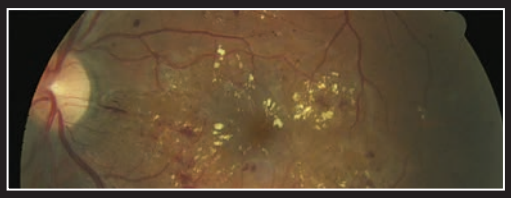
US Patent No. 5,800,807

LGX.0114.USA.16

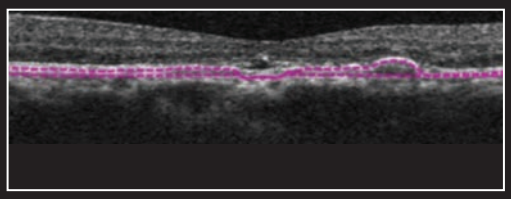
Based on 9269101/9269201 Revised: 08/2016

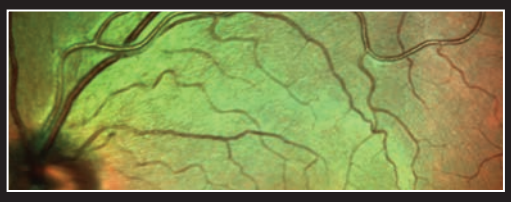
Contents

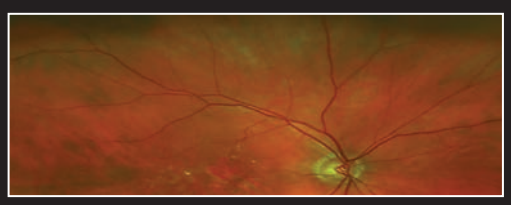
Review of Optometry June 2017

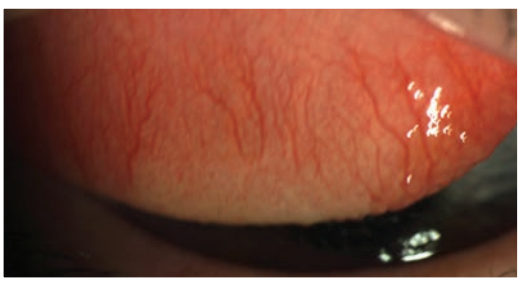


8TH ANNUAL RETINA REPORT



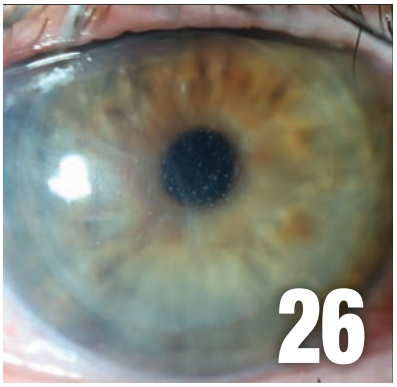


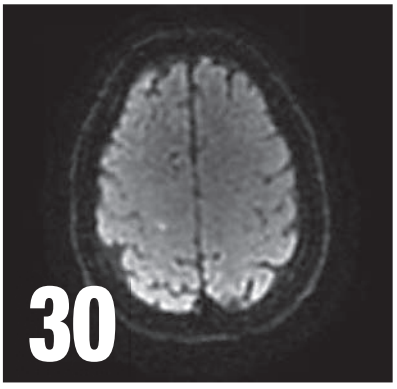


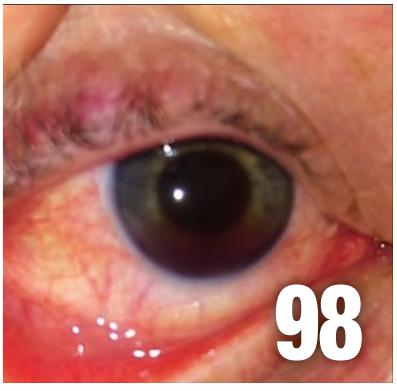


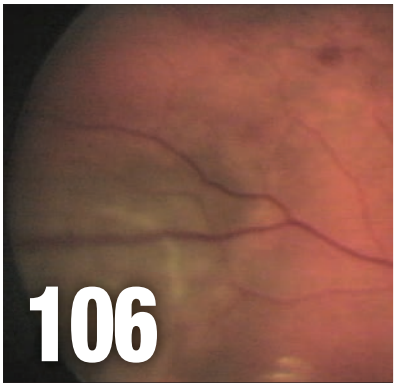
Departments

Review of Optometry June 2017









On The Web »
and more

Check out our multimedia and continuing education online at: *www.reviewofoptometry.com*

Digital Edition

Left your copy of *Review of Optometry* at the office? No problem! Access *Review* on your computer or mobile device!

Go to www.reviewofoptometry.com and click on the digimag link for the current issue.

Facebook and Twitter

For daily updates, "Like" our page on Facebook or "Follow" us on Twitter!

- www.facebook.com/revoptom
- http://twitter.com/#!/revoptom

Look for augmented content and special offers from *Review* and our advertisers. Specified pages work in conjunction with your smartphone or other mobile device to enhance the experience. With Layar, interactive content leaps off the page!

Step1: Download the free Layar app for iPhone or Android.

Step 2: Look for pages with the Layar Logo. layar INTERACTIVE PRINT

Step 3: Open the Layar app, hold the phone above the page and tap to scan it. Hold the phone above the page to view the interactive content.

The first 150 app downloads and completed forms will be entered into a drawing for a complimentary registration to one of *Review*'s 14-hour CE meetings, valued at $495.

THE SCIENCE BEHIND SIMPLE

Topcon's 3D OCT-1 MAESTRO™ has one-touch, auto-alignment making it easy to use and the preferred choice of eye care professionals around the globe. The MAESTRO requires minimal staff training and ensures that you get a high quality OCT and color fundus image every time.

"With the Maestro, generating high-quality images is extremely efficient and almost effortless. I can train my staff to capture images in a day, and its footprint is incredibly small."

—Travis Johnson, OD
Mursfreesboro, TN

MEET THE MAESTRO at AOA Booth #214

TOPCON

YOUR VISION. OUR FOCUS.

Maestro Dimensions: MIN 12.1"W x 18.5"D x 20.4"H | MAX 17.7"W x 26.3"D x 28.4"H

 newsgram.topconmedical.com/maestro MCA# 2002

2018 MEETINGS

NEW TECHNOLOGIES & TREATMENTS IN EYE CARE 2018

REVIEW OF OPTOMETRY®
EDUCATIONAL MEETINGS OF CLINICAL EXCELLENCE

FEBRUARY 16-20, 2018
Winter Ophthalmic Conference
ASPEN, CO
Westin Snowmass Conference Center
Program Chairs: Murray Fingeret, OD and Leo Semes, OD

APRIL 6-8, 2018
NASHVILLE, TN
Nashville Marriott at Vanderbilt
Program Chair: Paul Karpecki, OD

APRIL 26-29, 2018
SAN DIEGO, CA
Program Chair: Paul Karpecki, OD

MAY 17-20, 2018
ORLANDO, FL
Disney's Yacht & Beach Club
Program Chair: Paul Karpecki, OD

NOVEMBER 2-4, 2018
PHILADELPHIA, PA
Program Chair: Paul Karpecki, OD

Visit our website for the latest information:
www.reviewofoptometry.com/events
email: reviewmeetings@jobson.com | call: 866-658-1772

Administered by
Review of Optometry®

COPE *Approval pending

SALUS UNIVERSITY
Pennsylvania College of Optometry

Review of Optometry® partners with Salus University for those ODs who are licensed in states that require university credit. See *Review* website for any meeting schedule changes or updates.

REVIEW OF OPTOMETRY

PRINTED IN USA

FOUNDING EDITOR, FREDERICK BOGER
1891-1913

EDITORIAL OFFICES
11 CAMPUS BLVD., SUITE 100
NEWTOWN SQUARE, PA 19073
WEBSITE • WWW.REVIEWOFOPTOMETRY.COM

SUBSCRIPTION INQUIRIES
1-877-529-1746

CONTINUING EDUCATION INQUIRIES
1-800-825-4696

EDITOR-IN-CHIEF • JACK PERSICO
(610) 492-1006 • JPERSICO@JOBSON.COM

MANAGING EDITOR • REBECCA HEPP
(610) 492-1005 • RHEPP@JOBSON.COM

SENIOR EDITOR • BILL KEKEVIAN
(610) 492-1003 • BKEKEVIAN@JOBSON.COM

ASSOCIATE EDITOR • MICHAEL RIVIELLO
(610) 492-1021 • MRIVIELLO@JOBSON.COM

ASSOCIATE EDITOR • MICHAEL IANNUCCI
(610) 492-1043 • MIANNUCCI@JOBSON.COM

SPECIAL PROJECTS EDITOR • JILL HOFFMAN
(610) 492-1037 • JHOFFMAN@JOBSON.COM

ART DIRECTOR • JARED ARAUJO
(610) 492-1032 • JARAUJO@JOBSON.COM

DIRECTOR OF CE ADMINISTRATION • REGINA COMBS
(212) 274-7160 • RCOMBS@JOBSON.COM

EDITORIAL BOARD

CHIEF CLINICAL EDITOR • PAUL M. KARPECKI, OD

ASSOCIATE CLINICAL EDITORS • JOSEPH P. SHOVLIN, OD; ALAN G. KABAT, OD; CHRISTINE W. SINDT, OD

DIRECTOR OPTOMETRIC PROGRAMS • ARTHUR EPSTEIN, OD

CLINICAL & EDUCATION CONFERENCE ADVISOR
PAUL M. KARPECKI, OD

CASE REPORTS COORDINATOR • ANDREW S. GURWOOD, OD

CLINICAL CODING EDITOR • JOHN RUMPAKIS, OD, MBA

CONSULTING EDITOR • FRANK FONTANA, OD

COLUMNISTS

CHAIRSIDE • MONTGOMERY VICKERS, OD

CLINICAL QUANDARIES • PAUL C. AJAMIAN, OD

CODING CONNECTION • JOHN RUMPAKIS, OD

CORNEA & CONTACT LENS Q+A • JOSEPH P. SHOVLIN, OD

DIAGNOSTIC QUIZ • ANDREW S. GURWOOD, OD

THE ESSENTIALS • BISANT A. LABIB, OD

FOCUS ON REFRACTION • MARC TAUB, OD; PAUL HARRIS, OD

GLAUCOMA GRAND ROUNDS • JAMES L. FANELLI, OD

NEURO CLINIC • MICHAEL TROTTINI, OD; MICHAEL DELGIODICE, OD

OCULAR SURFACE REVIEW • PAUL M. KARPECKI, OD

RETINA QUIZ • MARK T. DUNBAR, OD

REVIEW OF SYSTEMS • CARLO J. PELINO, OD; JOSEPH J. PIZZIMENTI, OD

SURGICAL MINUTE • DEREK N. CUNNINGHAM, OD; WALTER O. WHITLEY, OD, MBA

THERAPEUTIC REVIEW • JOSEPH W. SOWKA, OD; ALAN G. KABAT, OD

THROUGH MY EYES • PAUL M. KARPECKI, OD

URGENT CARE • RICHARD B. MANGAN, OD

JOBSON MEDICAL INFORMATION LLC

Outlook

By Jack Persico, Editor-in-Chief

Puzzling Over Presbyopia

Would a new topical therapy weaken—or strengthen—the standing of corrective lenses?

My wife and I like to do the *New York Times* crossword together every Saturday—the hardest, and most fun, puzzle of the week (Sunday's is too big and boring). It's a weekly ritual we both look forward to, one of the few moments in the hectic lives of new parents when we get to take a break from talking about diapers and day care and actually use our brains. Anyway, we love the crossword.

Trouble is, I can't see the damn thing anymore.

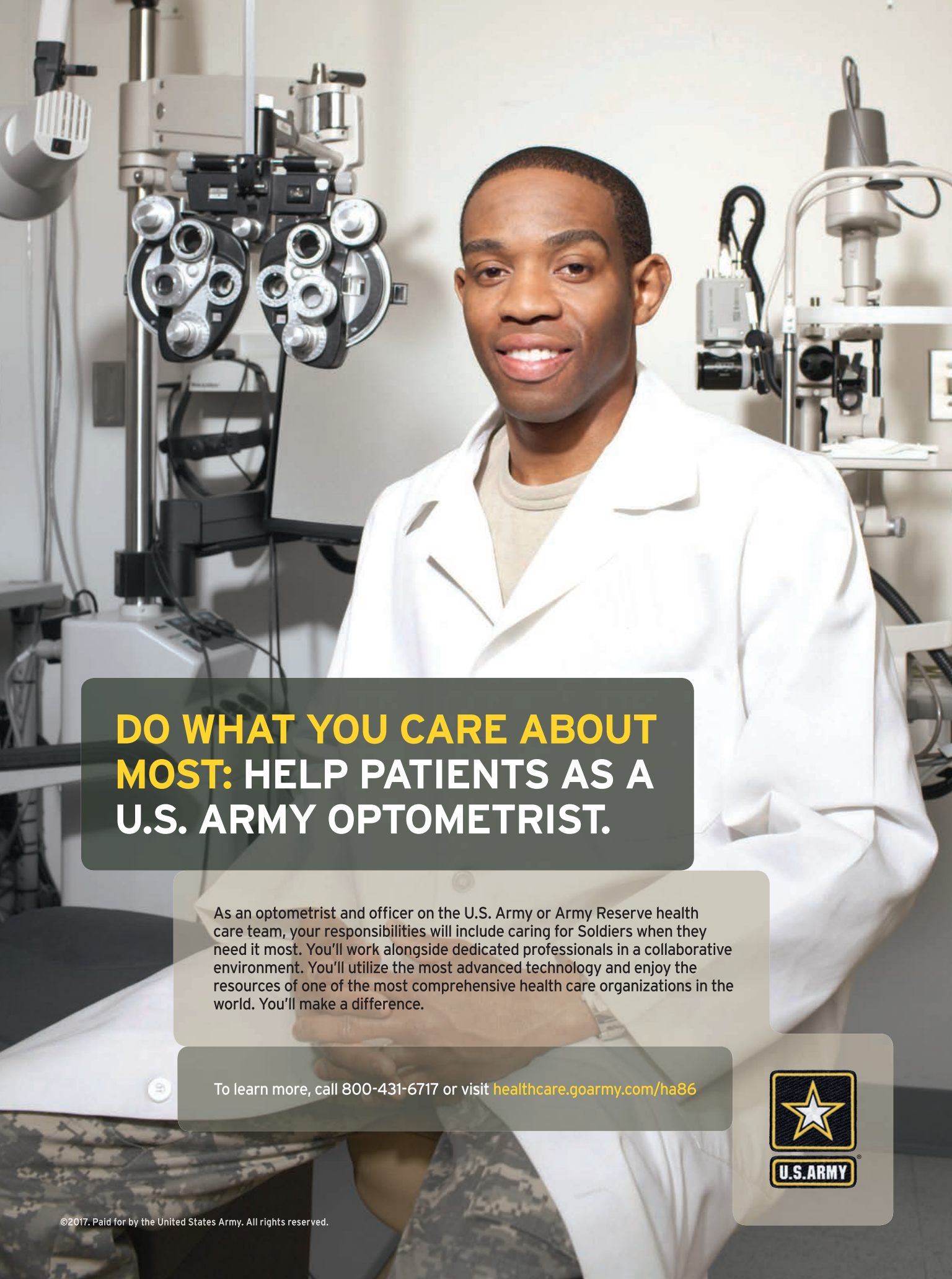
I've been presbyopic for the last few years. Until recently, I took it in stride. Having worn contact lenses since I was 20, I was motivated to stay in the modality. So I got multifocal contacts a few years ago and honestly loved them—for a while. I saw great at distance and just fine at near. Then the inevitable happened: my presbyopia progressed. Reading menus at restaurants got a little harder. I had to maneuver my cell phone a bit to find the sweet spot for viewing. But all in all, I got by.

Until a few months ago when the crossword became unworkable. I would bob and weave like a prizefighter to try to get it in focus, but just couldn't. Maybe the point size of the text is too small or the viewing distance I need for sharing the page with my wife is too awkward for realistic expectations of success (always a thorny issue between doctor and patient). My OD was very generous with time and trial lenses in trying to find a new prescription for me, to no avail; I just had to chalk this one up as a loss.

So, as I wondered who might be a viable candidate for an eye drop to treat presbyopia, well, one name came to mind pretty quickly. It's too early to tell if these drugs will even be approved, let alone perform in practice as anticipated, but I can definitely see the wisdom of an insightful point made by Mile Brujic, OD, in this month's cover story. Dr. Brujic points out that low-add patients are more successful with multifocal contact lenses than high-add ones, "so if we can take advanced presbyopic patients and improve their vision to a level where they really just need a low add, then we can increase their chances of becoming more successful" with multifocal lenses. The same goes for LASIK candidates, notes Chris Freeman, OD.

Wholly new product categories are rare, and I'm as intrigued as everyone we interviewed about the prospect of another option to pursue for such a universal ailment as presbyopia. Maybe these eye drops won't storm into the market and completely overtake the tried-and-true methods; they're still vulnerable to patient noncompliance, an all-too-common check against enthusiasm. But I can see them finding success in cases like mine, where existing modalities fall short and the patient is highly motivated to keep trying.

Don't write off older options in the rush to embrace new ones. If I could administer an eye drop every Saturday morning and be able to enjoy my crossword time with my wife in peace, I'd pay out of pocket for that privilege—and still keep paying for my contact lenses. ■



DO WHAT YOU CARE ABOUT MOST: HELP PATIENTS AS A U.S. ARMY OPTOMETRIST.

As an optometrist and officer on the U.S. Army or Army Reserve health care team, your responsibilities will include caring for Soldiers when they need it most. You'll work alongside dedicated professionals in a collaborative environment. You'll utilize the most advanced technology and enjoy the resources of one of the most comprehensive health care organizations in the world. You'll make a difference.

To learn more, call 800-431-6717 or visit healthcare.goarmy.com/ha86

U.S.ARMY

Letters to the **Editor**

We are committed to continuing the mission President Lincoln promised, the VA mission: "To care for him who shall have borne the battle, and for his widow, and his orphan" by serving and honoring the men and women who are America's veterans.

Eye Care in the VA: Why it Matters

The Department of Veterans Affairs (VA) has a long, successful history of providing care to veterans with complex medical problems.[1] Currently, with estimates of more than 20.1 million veterans in the United States, comprehensive eye exams are provided by more than 930 VA optometrists.[2,3]

However, veterans' eye care services have been under scrutiny lately. A recent *New York Times* article quoted the new Secretary of Veterans Affairs, Dr. David J. Shulkin, as saying, "We make eyeglasses for our veterans. Last time I checked, every shopping mall in America has a place where you can get glasses in an hour. I don't care about making eyeglasses. I care about getting that veteran his prostheses."[4] In the same article, the secretary goes on to acknowledge, "Many of the agency's patients have a complex mix of physical and mental health issues. They can't go get care in the private sector, at least not the comprehensive care the VA gives them."[4] In another report, an unnamed medical center director stated that Secretary Shulkin suggested all medical directors eliminate VA optometry and audiology at their facilities.[5]

But comprehensive eye care goes far beyond an eyeglasses prescription. Optometry provides the majority of the comprehensive eye care encounters at VA, and increasing access and reducing costs isn't as easy as just outsourcing a specialty.

The Long Arm of VA Education

The majority of VA optometrists have completed additional training through hospital-based ocular disease, contact lens or low vision rehabilitation residency programs. Many optometrists pass additional exams designed to document competency in medical-based optometric care. The additional training prepares them to provide the unique care a veteran cohort requires.

The VA also has the largest optometry clinical training program in the United States, with more than 180 accredited resident positions and more than 1,400 student externships each year.[6] Approximately 70% of all optometry graduates depend on VA for some part of their training. Clinical education, one of the pillars of VA, along with clinical care and research, would be lost if VA optometry is outsourced to the private sector.

VIP Care for VA Patients

Refractions are only one part of the comprehensive eye exams VA optometrists routinely perform to evaluate and manage a wide range of ocular diseases, as well as the ocular manifestations of systemic diseases.

VA optometrists also have the unique ability to address veterans' visual needs as part of an integrated system. Optometrists specializing in low vision and vision rehabilitation provide services for veterans who are visually impaired, legally blind or who suffer from duel sensory impairment—no small feat, considering the VA estimates approximately 130,428 veterans are legally blind and more than one million are visually impaired.[7] Integrated care with blind rehabilitation therapists aids our low vision veterans with orientation, mobility training and home skills training. As part of a team, VA optometrists prescribe low vision devices and technologies that help veterans maintain independence and a better quality of life.

Another area of particular significance involves traumatic brain injury (TBI), given an estimated 320,000 soldiers have experienced a TBI during deployment since 2001.[8] Ocular manifestations from this injury can be devastating, and optometrists coordinate care with the subspecialty VA teams that aid in the veteran's recovery or rehabilitation.

Additionally, optometric privileges in a hospital-based setting provide additional diagnostic and ancillary testing. For example, onsite imaging of the head, neck and orbits can save vision, and lives, with prompt diagnosis of conditions such as carotid occlusive disease, space-occupying lesions, aneurysm and stroke.

When it comes to technology, the VA has much to offer that the private sector cannot. The VA's electronic medical record is integral to the instantaneous communication between providers, who can review complete records of patients who have received care at multiple VA facilities and in the Department of Defense.

Further, VA optometrists comprise the majority of providers who are certified in teleretinal imaging. This screening process, most often done at the time of a primary care visit, has expanded access to care and introduced prompt examination and intervention for diabetic and other eye diseases.

The Numbers Don't Add Up

Outsourcing may not even be cost effective. VA, as with other government agencies, can contract for goods through a bidding process, ensuring access to top-of-the-line

technology and goods at the lowest possible price. In addition, optometrists are cost-effective providers. The average VA optometric salary in 2015 was $103,044, compared with an ophthalmologist's base salary of $186,177.[9,10] With optometry providing the majority of eye care, ophthalmology can focus on surgical eye care and treatment of advanced disease—further cutting costs while maintaining exceptional patient care. As the scope of optometry practice widens, optometrists' role will continue to expand, especially in underserved rural communities, where many of our veterans live and access to eye care is lacking.

Most importantly, outsourcing to the private sector creates new challenges in coordinating care delivered by both VA and the community, potentially delaying care while also driving up costs.

VA optometry provides far more than just eyeglasses. We are committed to continuing the mission President Lincoln promised, the VA mission: "To care for him who shall have borne the battle, and for his widow, and his orphan" by serving and honoring the men and women who are America's veterans.

—Jarett Mazzarella, OD,
Salisbury, NC

1. Galea S. Editorial: Veterans' Health. Am J Epidemiol. 2015;181(4):223-4.
2. U.S. Census Bureau. American Community Survey (ACS). www.census.gov/programs-surveys/acs. Accessed May 24, 2017.
3. National Association of Veterans Affairs Optometrists. www.navao.org. Accessed May 24, 2017.
4. Philipps D, Fandos N. New Veterans Affairs Chief: A Hands-On, Risk-Taking 'Standout.' The New York Times. May 9, 2017.
5. Krause B. Shulkin Says Get Rid Of VA Optometry, There Is A LensCrafters On Every Corner. DisabledVeterans.org. May 4, 2017.
6. Department of Veterans Affairs. Education and Training. www.va.gov/OPTOMETRY/Education_and_Training.asp. Updated November 18, 2016. Acessed May 24, 2017.
7. Department of Veterans Affairs. Blind Rehabilitation Services. www.rehab.va.gov/blindrehab. Accessed May 24, 2017.
8. Tanielian T, Lisa H. Jaycox LH, eds. Invisible Wounds of War. Santa Monica, CA: Rand; 2008.
9. Pay Rates for Optometrist. www.federalpay.org. Accessed May 24, 2017.
10. US Department of Veterans Affairs Ophthalmologist Salaries. www.glassdoor.com. Accessed May 24, 2017.

Through **My Eyes**

By Paul M. Karpecki, OD, Chief Clinical Editor

Quarterbacking Retinal Disease

After years of being sidelined, today's optometrist takes the snap.

When I was in my residency in the mid-1990s, the AREDS2 results weren't available to inform our decision-making about intermediate stage AMD, and no anti-VEGF treatments for wet AMD existed. OCT was still only used at research institutions. To a large extent, the only tool we had to monitor AMD patients was our own eyes—and theirs. Patients were typically followed with an Amsler grid, and we referred them to a retina specialist if something changed. Even then, very little could be done to improve their vision if they developed exudation. Likewise for patients with signs of diabetic retinopathy—we sent them off.

But today, innovative diagnostic testing and breakthrough therapies allow optometrists to focus on retinal disease as a key aspect of our practices. And it works best when the optometrist takes the role of quarterback.

Quarterbacks make sure every player is prepared for the next play; likewise, optometrists are tasked with updating the primary care provider or endocrinologist on the status of their patient's ocular health. Most importantly, optometrists make sure all members of the health care team are working in concert and at the top of their game, which ultimately benefits the patient.

Better managing patients with retinal disease begins with establishing a relationship with a retina specialty practice. Clinicians should observe the specialist in clinic and surgery to gauge the skills and demeanor of the specialist caring for your patients. With the right team in place, optometrists can be confident when taking the next step—early diagnosis.

Early Diagnosis for the Win

Newer technologies have upped our game when it comes to diagnosing early retinal disease. For example, optometrists who are equipped with OCT, OCT-A and ultra-widefield imaging can often make a relatively clear diagnosis—even before we sit down with the patient.

For dry AMD, clinicians can now diagnose this condition prior to seeing signs (or with only early minimal signs) through dark adaptation testing with AdaptDx (Maculogix). The test takes about six minutes and has over 90% sensitivity and specificity for a diagnosis of early AMD. Genetic testing will also play a significant role in both the diagnosis and potential treatment options in the future.

In cases of diabetes, technology can help identify the million or more cases of diabetic macular edema (DME) that go undiagnosed or untreated in the United States. Widefield photography, OCT and OCT-A can play a critical role in helping us monitor for retinal changes in moderate to severe nonproliferative diabetic retinopathy, increase the frequency of follow up and discuss the importance of systemic control, smoking cessation and nutrition. All of this further helps us make timely referrals for patients at risk for vision loss due to DME or proliferative disease.

Retina Playbook

The best part of being on the retina team is the expanded playbook. Everything from anti-VEGF therapy to small-gauge surgery allows most patients to have normal vision and lives. Early AMD interventions—such as UV-blocking sunglasses, high-energy visible light-blocking lenses and vitamins with carotenoids (lutein, zeaxanthin, mesozeaxanthin)—can positively impact patients early in the course. AREDS2 supplements may reduce risks for those with more advanced disease. Future therapies directed at previously untreatable causes of vision loss, such as lampalizumab for geographic atrophy, may come to fruition. For end-stage cases, we can refer for an Implantable Miniature Telescope procedure or turn to our own optometric specialists in low vision to provide tangible quality of life benefits.

The features in this month's *Annual Retina Report* show just how far optometry has come. We now take charge of the early identification of posterior segment disease and educate patients on anti-VEGF therapy, then comanage with our retina specialists—exactly the sort of teamwork a good quarterback creates.

The health care system will continue to be stressed over the next decade, but with optometry's higher level of participation in retinal disease, we can make a difference. Optometrists can help slow disease progression and appropriately monitor and refer patients to a multitude of specialists—including those within our own ranks. ■

BRUSH. FLOSS. AVENOVA®.

Avenova. The essential part of any lid hygiene regimen.

Antibiotic and resistance free Avenova with Neutrox®. The one no-sting solution that helps manage the itchy, burning, irritated eyelids often caused by blepharitis, MGD and dry eye. Used alone or concomitantly, Avenova is the essential part of any lid and lash hygiene regimen. Avenova. Every day.

Avenova®
Daily lid and lash hygiene.

Visit NovaBay at AOA – Booth #431

Avenova contains Neutrox, a proprietary pure hypochlorous acid that mimics the body's immune response. It works without creating mammalian cell toxicity or bacterial resistance. It's well tolerated for daily use.

AVENOVA.COM | 1-800-890-0329 | RX ONLY

Testing, Testing, 1, 2, 3…

I haven't had a pop quiz in forever, and never in my optometric crime-fighting life, so here goes. **By Montgomery Vickers, OD**

This month's pop quiz should help you get to know our profession a little better. The category is *States*. I will not give you any answers, so if you are that curious, you can look it up. Also, this is COPE approval pending (except in all 50 states).

1. In what state did an optometrist first decide it was a good business plan to make examinations FREE? (I would have put the Great State of Insanity.)
2. In what state did an optometrist get in trouble for doing scoliosis examinations? (I'll get *back* to you on that.)
3. Name a state that doesn't have two or three new optometry schools in the works?
4. Which is higher, the number of states that allow open carry of assault rifles, or the number of states that accept another state's license to practice optometry? (After all, the government's primary concern is to protect its citizens).
5. Name the state in which your humble Chairside writer was born. (Hint: it's not West Virginia.)
6. Name the state where there are more bears than contact lenses. (You know who you are.)
7. In what state was the Canal of Schlemm studied extensively thanks to a crazy billionaire?
8. True or false: in at least one state, new, healthy contact lenses are more important than new, shiny smartphones.
9. How many states allow optometrists to call themselves physicians?
10. How many states should allow optometrists to call themselves physicians? (That's a gimme.)
11. In which states can specially-trained optometrists use lasers in eye treatments?
12. In which states can anybody with no training and any old MD license use lasers in eye treatments?
13. Are there any states where optometrists can prescribe medical marijuana?
14. Is California still a state?
15. Has Texas seceded from the Union yet?
16. Predicting the future, when will there be more ODs than presbyopes in West Virginia?
17. Newbies, what state houses the American Optometric Association?
18. What state requires the most yearly hours to keep your license?
19. What state flags have human eyes?
20. What state flags have non-human eyes?
21. Any states still not allow glaucoma treatment?
22. Any states still not allow oral medication treatments?
23. Any states you feel like moving out of now?
24. What happens if half your building is in one state and the other is in another state?
25. Can I put an office in Hawaii? Oh, that's right, they don't accept my Texas license and 37 years of experience.

I know you had to look up a few of these, and are now (1) researching moving companies and (2) writing down another New Year's resolution to get involved—for next year. But I digress as usual. Here's the kicker: I don't know the answers to any of these questions either… OK, maybe a couple… so please don't obsess. Go see a patient instead. ■

Hello Miru.

Bye, bye blister pack.

Introducing Miru 1day, the world's thinnest package for daily disposable contact lenses.

Miru's ultra lightweight 1mm thin package is about 1/8th the thickness of a traditional blister pack and was specifically developed to reduce the risk of microbial contamination. When opened, the lens is presented on a special disk, oriented correctly for proper insertion.

To learn more and request trials, please visit: miru.meniconamerica.com

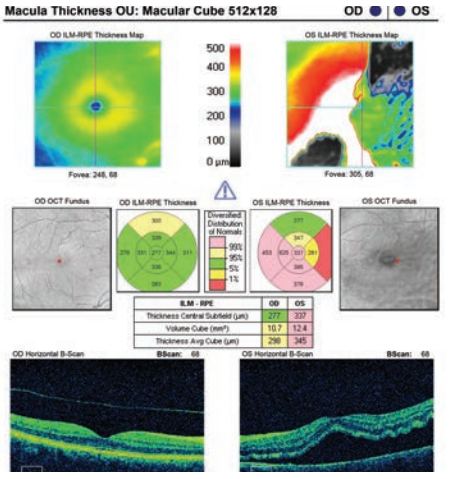
Miru
1day Menicon Flat Pack

Look Before You Leap

Some practitioners are ordering OCTs at the drop of a hat if the visual acuity is reduced. But often, all you need to do is dilate. **Edited by Paul C. Ajamian, OD**

Q I have a post-op cataract patient who, at their one-week visit, had 20/20 vision and a normal dilated fundus exam. At their one-month visit, vision was 20/20-2. Should I get an OCT?

A "It's critical to look at the fundus and let your clinical acumen help determine the presence or absence of clinically significant findings, no matter the scenario," says Jeffry Gerson, OD, of Grin Eye Care in Olathe, KS. "Make sure that it's you, the clinician, who is doing the diagnostic digging—and not the optical coherence tomography (OCT)." The machine is only as good as the optometrist's expertise to determine the need for referral based on the findings of their dilated exam, Dr. Gerson explains.



Not every ancillary test can or should be used on every patient.

The Eyes God Gave You

Not every patient needs extensive testing or OCT imaging—remember, some will never achieve 20/20 vision. "The primary modality for finding pathology has long been, and should continue to be, the good, old-fashioned clinical exam," says Dr. Gerson. Not every ancillary test can or should be used on every patient, he says.

For instance, later in the post-op course, CME is one of the more common complications following cataract surgery.[1] However, this doesn't mean that OCT takes the place of your fundus examination, says Dr. Gerson. "Clinicians should be able to observe conditions—in this particular example, CME—with a condensing lens at the slit lamp," he explains. While OCT can serve as a confirmatory test, it's not likely needed to make a diagnosis.

"In general, if somebody corrects to only 20/25, then clinical examination should rule out such significant pathologies as dry eye and cataracts. Most macular pathology, as well, can be seen on careful clinical examination," says Dr. Gerson. Of course, some subtle changes will be seen with OCT that are not evident on exam, but this should be the exception and not the rule when vision is decreased, he explains. "Visually significant pathologies, whether they are macular edema, macular degeneration or pigmentary changes, should be evident via the clinical examination."

What's the Harm?

Given the utility of OCT in various situations, it's tempting to say there's no harm in doing OCT in any and all contexts; however, clinicians must rely on their clinical skills and remember the value of dilation to reveal the presence or absence of conditions warranting referral.

"There is the possibility of over-referral, in a post-op or any other patient that comes back with an 'abnormal OCT'," says Dr. Gerson. It's imperative clinicians use the OCT in conjunction with the history and clinical exam before sending a patient out to a retina or glaucoma specialist, or back to the cataract surgeon, he explains.

"Sending a patient for a second opinion and giving the patient the impression that a significant, possibly vision-threatening issue exists can put them on an emotional roller coaster," says Dr. Gerson. Worry, despair and anxiety due to a potential vision problem lead to anger and frustration when the patient finds out that their anxiety was ultimately unfounded, explains Dr. Gerson.

So, before ordering an OCT, look at the fundus and put the findings together. OCT is a great ancillary tool, but it's dangerous to stop looking directly at the fundus and let the OCT 'do the thinking.' "Ultimately, the machine is the not clinician—you are," says Dr. Gerson. "Using the eyes God gave you will prove the most fruitful when discerning the need to refer." ■

1. Henderson B, Kim J, Ament C, et al. Clinical pseudophakic cystoid macular edema-Risk factors for development and duration after treatment. J Cataract Refract Surg. 2007;33:1550-8.

EagleVision®

So many advantages for such a small piece of medical technology

Tapered Shaft facilitates better patient retention

Low Profile Cap superb comfort with less foreign body sensation

Soft Pliable Silicone to accommodate any anatomy

Fine Distal Tip for easy insertion

katena
INSTRUMENTS • BIOLOGICS • DEVICES
& now Plugs!

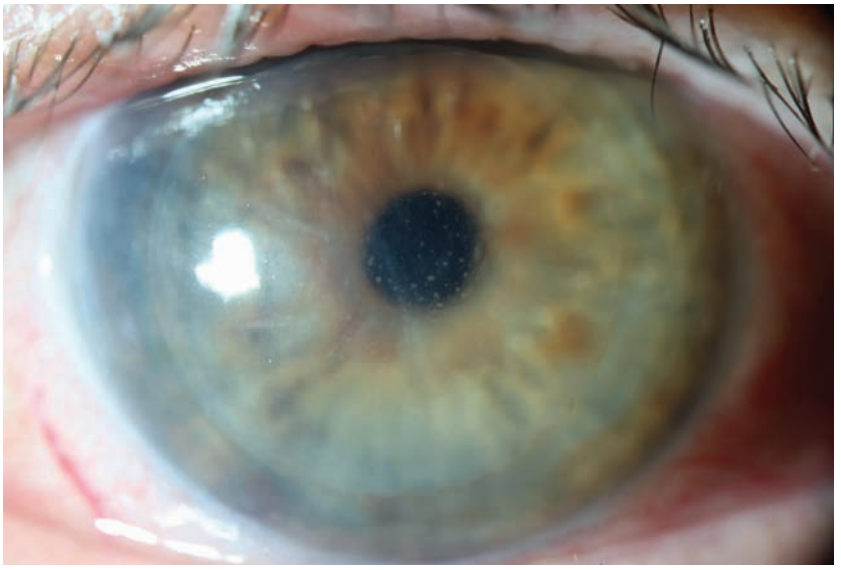
1-800-222-7584 • 973-989-1600 • www.eaglevis.com

LIT200 CID 5324 Rev0

Coping with Rejection

Early recognition is the key to caring for patients with urgent corneal transplant issues. **By Christopher Kuc, OD, and Richard Mangan, OD**

In the past 55 years, more than a million corneal transplants have restored vision for patients, and more than 40,000 patients undergo this procedure every year.[1] Naturally, these patients will turn to their optometrist for primary care concerns. Caring for these patients is well within the OD's scope, but they do have special needs, whether those are fitting a specialty contact lens, an annual exam to evaluate the health and integrity of the graft, or a postoperative visit to monitor the healing process. Additionally, these patients may present urgently with decreased vision, redness or other symptoms. Understanding the hurdles and timing that were overcome while procuring the graft should make any process that threatens the graft urgent and paramount to the examining doctor.



This patient's DSAEK rejection demonstrates microcystic edema and Descemet's folds with keratic precipitates.

Success and Failure

The cornea is an "immune privileged" tissue that is avascular and possesses immune characteristics that allow any suitable cornea to be transplanted from the donor to the recipient, whether ABO or human leukocyte antigen matched.[2] Penetrating keratoplasty was the standard for transplantation for dystrophies, infections, scarring and keratoconus, but with the advent of Descemet's stripping endothelial keratoplasty (DSEK/DSAEK) and Descemet's membrane endothelial keratoplasty (DMEK), endothelial dystrophies can be treated with less risk for failure and shorter recovery time.[3] Although they require greater surgical time, anterior corneal transplants, such as deep anterior lamellar keratoplasty have also significantly diminished rejection rates and enhanced the success rate of corneal transplants.[4]

Clarifying the terms "rejection" and "failure" is the first step to identifying what process is taking place. Graft failure is a term that describes any reason the graft has stopped functioning and has become cloudy, preventing usable vision. This may be due to any number of reasons, such as endothelial pump failure, rejection, infection or ocular surface disease.[5,6] Graft rejection is a specific process whereby the host immune response is directed toward antigens on the corneal donor button.[7] Rejection leads to failure, but failure is not necessarily caused by rejection.

Graft rejection is characterized by one or more of these patient symptoms: redness, pain, photophobia and decreased vision. Common clinical findings that could indicate early rejection are corneal edema, corneal infiltrate, anterior chamber reaction, keratic precipitates (specifically on the graft endothelium and not the host) and limbal injection.[8] A rejection line is one clinical finding that is pathognomonic for graft rejection. These can be epithelial or endothelial (Khodadoust line).

Rejection Types

Differentiating these clinical signs can help distinguish what type of rejection is taking place. Epithelial

The *FIRST* and *ONLY* NSAID indicated to prevent ocular pain in cataract surgery patients[1]

A DROP OF PREVENTION

FOR YOUR CATARACT SURGERY PATIENTS

Defend against ocular pain and combat postoperative inflammation with the penetrating power of BromSite® formulated with DuraSite®[1]

- DuraSite® increases ocular surface retention time, resulting in increased bromfenac absorption[2-5]
- Provides 24-hour coverage with BID dosing[1]
- Available in 5 mL bottle

Visit bromsite.com to find out more.

BromSite®
(bromfenac ophthalmic solution) 0.075%
Formulated with DURASITE® DELIVERY SYSTEM

Indications and Usage

BromSite® (bromfenac ophthalmic solution) 0.075% is a nonsteroidal anti-inflammatory drug (NSAID) indicated for the treatment of postoperative inflammation and prevention of ocular pain in patients undergoing cataract surgery.

Recommended Dosing

One drop of BromSite® should be applied to the affected eye twice daily (morning and evening) 1 day prior to surgery, the day of surgery, and 14 days postsurgery.

Important Safety Information

- **Slow or Delayed Healing:** All topical nonsteroidal anti-inflammatory drugs (NSAIDs), including BromSite®, may slow or delay healing. Topical corticosteroids are also known to slow or delay healing. Concomitant use of topical NSAIDs and topical steroids may increase the potential for healing problems.
- **Potential for Cross-Sensitivity:** There is the potential for cross-sensitivity to acetylsalicylic acid, phenylacetic acid derivatives, and other NSAIDs, including BromSite®. Therefore, caution should be used when treating individuals who have previously exhibited sensitivities to these drugs.
- **Increased Bleeding Time of Ocular Tissue:** With some NSAIDs, including BromSite®, there exists the potential for increased bleeding time due to interference with platelet aggregation. There have been reports that ocularly applied NSAIDs may cause increased bleeding of ocular tissues (including hyphemas) in conjunction with ocular surgery. It is recommended that BromSite® be used with caution in patients with known bleeding tendencies or who are receiving other medications which may prolong bleeding time.
- **Keratitis and Corneal Effects:** Use of topical NSAIDs may result in keratitis. In some susceptible patients, continued use of topical NSAIDs may result in epithelial breakdown, corneal thinning, corneal erosion, corneal ulceration or corneal perforation. Patients with evidence of corneal epithelial breakdown should immediately discontinue use of topical NSAIDs, including BromSite®, and should be closely monitored for corneal health. Patients with complicated ocular surgeries, corneal denervation, corneal epithelial defects, diabetes mellitus, ocular surface diseases (e.g., dry eye syndrome), rheumatoid arthritis, or repeat ocular surgeries within a short period of time may be at increased risk for corneal adverse events which may become sight threatening. Topical NSAIDs should be used with caution in these patients. Post-marketing experience with topical NSAIDs also suggests that use more than 24 hours prior to surgery or use beyond 14 days postsurgery may increase patient risk for the occurrence and severity of corneal adverse events.
- **Contact Lens Wear:** BromSite® should not be administered while wearing contact lenses. The preservative in BromSite®, benzalkonium chloride, may be absorbed by soft contact lenses.
- **Adverse Reactions:** The most commonly reported adverse reactions in 1% to 8% of patients were anterior chamber inflammation, headache, vitreous floaters, iritis, eye pain, and ocular hypertension.

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

NSAID=nonsteroidal anti-inflammatory drug.

References: 1. BromSite® [package insert]. Cranbury, NJ: Sun Pharmaceutical Industries, Inc.; 2016. **2.** Hosseini K, Hutcheson J, Bowman L. Aqueous humor concentration of bromfenac 0.09% (Bromday™) compared with bromfenac in DuraSite® 0.075% (BromSite™) in cataract patients undergoing phacoemulsification after 3 days dosing. Poster presented at: ARVO Annual Meeting; May 5-9, 2013; Seattle, Washington. **3.** ClinicalTrials.gov. Aqueous humor concentration of InSite Vision (ISV) 303 (bromfenac in DuraSite) to Bromday once daily (QD) prior to cataract surgery. https://clinicaltrials.gov/ct2/show/results/NCT01387464?sect=X70156&term=insite+vision&rank=1. Accessed March 2, 2017. **4.** Si EC, Bowman LM, Hosseini K. Pharmacokinetic comparisons of bromfenac in DuraSite and Xibrom. *J Ocul Pharmacol Ther.* 2011;27(1):61-66. **5.** Bowman LM, Si E, Pang J, et al. Development of a topical polymeric mucoadhesive ocular delivery system for azithromycin. *J Ocul Pharmacol Ther.* 2009;25(2):133-139.

SUN OPHTHALMICS

BromSite® (bromfenac ophthalmic solution) 0.075%
Brief Summary

INDICATIONS AND USAGE
BromSite® (bromfenac ophthalmic solution) 0.075% is indicated for the treatment of postoperative inflammation and prevention of ocular pain in patients undergoing cataract surgery.

CONTRAINDICATIONS
None

WARNINGS AND PRECAUTIONS
Slow or Delayed Healing
All topical nonsteroidal anti-inflammatory drugs (NSAIDs), including BromSite® (bromfenac ophthalmic solution) 0.075%, may slow or delay healing. Topical corticosteroids are also known to slow or delay healing. Concomitant use of topical NSAIDs and topical steroids may increase the potential for healing problems.

Potential for Cross-Sensitivity
There is the potential for cross-sensitivity to acetylsalicylic acid, phenylacetic acid derivatives, and other NSAIDs, including BromSite® (bromfenac ophthalmic solution) 0.075%. Therefore, caution should be used when treating individuals who have previously exhibited sensitivities to these drugs.

Increased Bleeding Time of Ocular Tissue
With some NSAIDs, including BromSite® (bromfenac ophthalmic solution) 0.075%, there exists the potential for increased bleeding time due to interference with platelet aggregation. There have been reports that ocularly applied NSAIDs may cause increased bleeding of ocular tissues (including hyphemas) in conjunction with ocular surgery.

It is recommended that BromSite® be used with caution in patients with known bleeding tendencies or who are receiving other medications which may prolong bleeding time.

Keratitis and Corneal Reactions
Use of topical NSAIDs may result in keratitis. In some susceptible patients, continued use of topical NSAIDs may result in epithelial breakdown, corneal thinning, corneal erosion, corneal ulceration or corneal perforation. These events may be sight threatening. Patients with evidence of corneal epithelial breakdown should immediately discontinue use of topical NSAIDs, including BromSite® (bromfenac ophthalmic solution) 0.075%, and should be closely monitored for corneal health.

Post-marketing experience with topical NSAIDs suggests that patients with complicated ocular surgeries, corneal denervation, corneal epithelial defects, diabetes mellitus, ocular surface diseases (e.g., dry eye syndrome), rheumatoid arthritis, or repeat ocular surgeries within a short period of time may be at increased risk for corneal adverse events which may become sight threatening. Topical NSAIDs should be used with caution in these patients.

Post-marketing experience with topical NSAIDs also suggests that use more than 24 hours prior to surgery or use beyond 14 days postsurgery may increase patient risk for the occurrence and severity of corneal adverse events.

Contact Lens Wear
BromSite® should not be administered while wearing contact lenses. The preservative in BromSite®, benzalkonium chloride, may be absorbed by soft contact lenses.

ADVERSE REACTIONS
The following serious adverse reactions are described elsewhere in the Brief Summary:
- Slow or Delayed Healing
- Potential for Cross-Sensitivity
- Increased Bleeding Time of Ocular Tissue
- Keratitis and Corneal Reactions
- Contact Lens Wear

Clinical Trial 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 clinical practice.

The most commonly reported adverse reactions in 1–8% of patients were: anterior chamber inflammation, headache, vitreous floaters, iritis, eye pain and ocular hypertension.

USE IN SPECIFIC POPULATIONS
Pregnancy
Risk Summary
There are no adequate and well-controlled studies in pregnant women to inform any drug associated risks. Treatment of pregnant rats and rabbits with oral bromfenac did not produce teratogenic effects at clinically relevant doses.

Clinical Considerations
Because of the known effects of prostaglandin biosynthesis-inhibiting drugs on the fetal cardiovascular system (closure of ductus arteriosus), the use of BromSite® during late pregnancy should be avoided.

Data
Animal Data
Treatment of rats with bromfenac at oral doses up to 0.9 mg/kg/day (195 times a unilateral daily human ophthalmic dose on a mg/m^2 basis, assuming 100% absorbed) and rabbits at oral doses up to 7.5 mg/kg/day (3243 times a unilateral daily dose on a mg/m^2 basis) produced no structural teratogenicity in reproduction studies. However, embryo-fetal lethality, neonatal mortality and reduced postnatal growth were produced in rats at 0.9 mg/kg/day, and embryo-fetal lethality was produced in rabbits at 7.5 mg/kg/day. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

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

Pediatric Use
Safety and efficacy in pediatric patients below the age of 18 years have not been established.

Geriatric Use
There is no evidence that the efficacy or safety profiles for BromSite® differ in patients 65 years of age and older compared to younger adult patients.

NONCLINICAL TOXICOLOGY
Carcinogenesis, Mutagenesis and Impairment of Fertility
Long-term carcinogenicity studies in rats and mice given oral doses of bromfenac up to 0.6 mg/kg/day (129 times a unilateral daily dose assuming 100% absorbed, on a mg/m^2 basis) and 5 mg/kg/day (540 times a unilateral daily dose on a mg/m^2 basis), respectively revealed no significant increases in tumor incidence.

Bromfenac did not show mutagenic potential in various mutagenicity studies, including the bacterial reverse mutation, chromosomal aberration, and micronucleus tests.

Bromfenac did not impair fertility when administered orally to male and female rats at doses up to 0.9 mg/kg/day and 0.3 mg/kg/day, respectively (195 and 65 times a unilateral daily dose, respectively, on a mg/m^2 basis).

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

SUN-OPH-BRO-017-1 03/2017

SUN OPHTHALMICS

rejection occurs at the peripheral edge of the graft and is associated with localized engorged vessels and an elevated epithelial line, or ridge, which stains with fluorescein.[8,9] Subepithelial infiltrates, which are whitish and appear similar to those found in viral conjunctivitis, indicate another form of rejection known as subepithelial or chronic stromal rejection.[9] This presentation may be confused with viral conjunctivitis and should be considered rejection by the practitioner until proven otherwise to prevent misdiagnosis and further graft morbidity.

The most common form of rejection in up to 50% of cases is endothelial rejection, which is associated with limbal engorgement, corneal graft edema, anterior chamber reaction, keratic precipitates, and a Khodadoust line.[10] A Khodadoust line consists of segmented corneal edema and endothelial white blood cells adjacent to an area of clear cornea on the graft, which forms a distinct line.[8,9]

Understanding the severity of the rejection episode is critical to the survival of the graft. Research shows that following a rejection episode, graft failure is likely to occur in up to one third of these cases within six months.[10] One way of categorizing endothelial rejection by clinical findings is into three categories: possible, probable and definite (*Table 1*).[9] A vascularized cornea, especially deeper stromal vessels, is known as a high-risk cornea in transplant patients.[9] This is the single greatest risk factor for long-term failure. If a rejection episode is identified, then a phone consultation with the surgeon is always warranted, and these findings will help initiate the appropriate course of treatment and follow up.

Options

Treatment of graft rejection depends on the level of comfort of the practitioner, but in all suspected cases, treatment should be administered in consultation with a corneal surgeon. Initial dosage with topical steroids in a known rejection is Q2H with difluprednate 0.05% or Q1H with prednisolone 1%.[7,9] This may be given in conjunction with a sub-Tenon's injection of triamcinolone acetonide and/or oral prednisone initially (40-80mg PO), or possibly an IV methylprednisolone dose of 500mg.[7,9] The rate of reversal in severe endothelial rejection is as high as 60% when appropriate therapy is initiated.[11] This initial therapy is essential in high-risk patients, and may vary depending on the clinical findings.

Realizing that inflammation is the root cause of rejection, by identifying factors that can contribute to inflammation and further trigger an immune rejection, the clinician can provide the highest level of care to a transplant patient. For example, loose sutures can stimulate inflammation and incite infection and should be identified and removed. Poorly fit post-surgical contact lenses can cause superficial epithelial damage or long-term endothelial damage and trigger inflammation.[12] Additionally, conditions such as previous herpetic infection, dry eye, allergies and lid disease/lagophthalmos are just a few ocular comorbidities that can trigger inflammation on the ocular surface.[12]

All patients who have undergone corneal transplant surgery, regardless of type, should be placed on a daily topical immunosuppressant for life, such as loteprednol or fluorometholone. In cases when the patient is a steroid responder, antiglaucoma medications can be considered or, in severe cases, a topical cyclosporine may be considered for long-term therapy.

Educating our transplant patients that the earliest detection of symptoms such as pain, photophobia, redness or decreased vision is reason for an urgent visit will also help prevent loss of graft due to poor follow up. ■

Dr. Kuc specializes in glaucoma, cataract and dry eye care at Virginia Eye Consultants in Smithfield, VA.

Table 1. Endothelial Rejection Severity

Possible: Graft edema only.
Probable: Edema, cells/flare, keratic precipitates on donor button.
Definite: Edema, cells/flare, keratic precipitates on donor button, and Khodadoust Line.

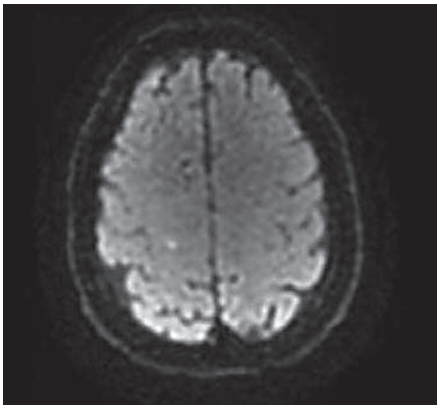
1. Frequently asked questions. Eye Bank Association of America. http://restoresight.org/wp-content/uploads/2015/07/Frequently-Asked-Questions.pdf
2. Maguire M, Stark W, Gottsch J, et al. Risk factors for corneal graft failure and rejection in the collaborative corneal transplantation studies. Ophthalmology. 1994;101(9):1536-47.
3. Anshu A, Price M, Price F. Risk of corneal transplant rejection significantly reduced with Descemet's membrane endothelial keratoplasty. Ophthalmology. 2012 Mar;119(3):536-40.
4. Sarnicola V, Toro P, Sarnicola C, et al. Long-term graft survival in deep anterior lamellar keratoplasty. Cornea. 2012;31:621–6.
5. Price M, Thompson R, Price F. Risk factors for various causes of failure in initial corneal grafts. Arch Ophthalmol. 2003;121(8):1087-92.
6. The Collaborative Corneal Transplantation Studies Research Group. Design and methods of the collaborative corneal transplantation studies. Cornea. 1993;12(2):93–103.
7. Qazi Y, Hamrah P. Corneal allograft rejection: immunopathogenesis to therapeutics. J Clin Cell Immunol. 2013;2013(Suppl 9):6.
8. Dua H, Azuara-Blanco A. Corneal allograft rejection: Risk factors, diagnosis, prevention, and treatment. Indian Journal of Ophthalmology. 1999;47(1):3-9.
9. Panda A, Vanathi M, Kumar A, Priya S. Corneal graft rejection. Survey of Ophthalmology. 2007;52(4):375-95.
10. Guilbert E, Bullet J, Sandali O, et al. Long-term rejection incidence and reversibility after penetrating and lamellar keratoplasty. Am J Ophthalmol. 2013;155(3):560-9.
11. Yamazoe K, Yamazoe K, Shimazaki-Den S, Shimazaki J. Prognostic factors for corneal graft recovery after severe corneal graft rejection following penetrating keratoplasty. BMC Ophthalmology. 2013;13(2):5.
12. Yorston D, Garg P. Corneal grafting: what eye care workers need to know. Community Eye Health. 2009;22(71):44–5.
13. Setälä K, Vasara K, Vesti E, Ruusuvaara P. Effects of long-term contact lens wear on the corneal endothelium. Acta Ophthalmol Scand. 1998:76:299–303.

CRAO: A New Way to Go

Research emphasizes emergent referral for retinal artery occlusion suspects.

By Michael Trottini, OD, and Michael DelGiodice, OD

A 65-year-old white male presented to the emergency room for an evaluation of acute vision loss in his right eye. He said that, while playing golf early in the morning, his vision started to "white out." Although his vision began to return that afternoon, his acuities were still decreased, prompting him to visit the emergency room. Initial testing revealed his blood pressure was elevated at 180/90. A computerized tomography scan of his brain was unremarkable, at which point we were consulted.



On MRI, a punctate acute stroke is noted along the medial aspect of the right central sulcus.

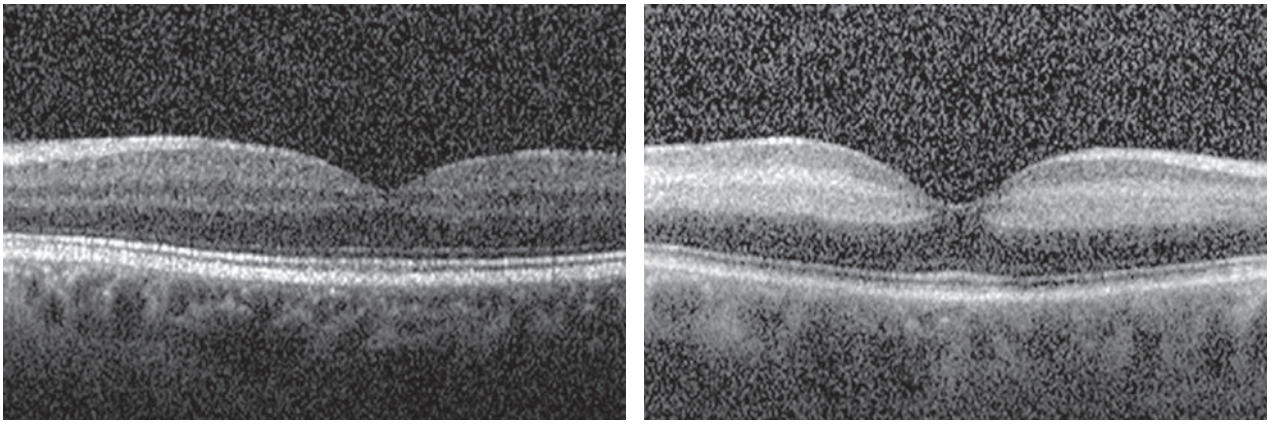
Emergent Workup

Upon examination, our patient denied any pain or neurologic symptoms associated with his vision loss. He denied scalp tenderness, headache or jaw claudication, symptoms of giant cell arteritis (GCA). At a recent visit, his internist told him he had borderline hypertension and cholesterol levels. At that time he was not started on any medication and was instructed to change his diet and engage in exercise.

The patient's visual acuities were recorded to be 20/100 OD and 20/20 OS. We noted a trace afferent pupillary defect in his right eye.

Anterior and posterior segment exams were both unremarkable, and we noted no vitreous hemorrhage, disc edema, vein occlusion, retinal detachment or maculopathy. The retina seemed well perfused at this point, with no emboli present; however, the patient's symptoms, recent vascular issues and absence of other clinical findings were highly suspicious for a possible diagnosis of reperfusing central retinal artery occlusion (CRAO).

We admitted the patient and ordered a complete blood count (CBC), complete metabolic panel (CMP), lipid profile, erythrocyte sedimentation rate (ESR) and c-reactive protein (CRP), as well as an MRI of the brain, MRA of the head and neck, and an echocardiogram. We also discussed the case with the attending physician and recommended the patient be treated for his elevated blood pressure.



The increased reflectivity and thickening of the right eye's nerve fiber layer (at right) is consistent with the acute phase of a retinal artery occlusion, contrasted with the appearance of the normal nerve fiber layer (at left) in the patient's left eye.

Diagnosis

Upon re-examination 24 hours later, his visual acuity had improved to 20/50 OD. MRI revealed a punctate acute stroke along the medial aspect of the right central sulcus, and MRA revealed 30% stenosis of the proximal right internal carotid artery secondary to an atherosclerotic plaque. The patient's total cholesterol and LDLs were both slightly elevated; CBC, CMP, ESR and CRP were normal and echocardiogram showed mild tricuspid valve regurgitation.

Our patient was diagnosed with a probable CRAO with an acute cerebrovascular accident (CVA) secondary to an atherosclerotic plaque of the right internal carotid artery. He was started on lisinopril, atorvastatin and clopidogrel and discharged two days later after cardiology and neurology determined he was stable.

Follow Up

On examination one week later, the patient's acuity was stable at 20/50 OD. Macular spectral-domain optical coherence tomography showed nerve fiber layer thickening consistent with the acute phase of a CRAO, and fluorescein angiography showed a delay in the filling time of the right eye's retinal arteries; the results of both tests were consistent with our diagnosis. We noted no additional clinical findings, and he was stable from an ocular perspective.

We instructed him to continue to follow up with his internal medicine, cardiology and neurology doctors for management of his underlying vascular issues and we will monitor him with dilated fundus examinations every three to six months.

Discussion

Although our patient was fortunate, the visual prognosis is generally quite poor for an eye that develops a retinal artery occlusion (RAO). Certain in-office procedures, such as digital massage and anterior chamber paracentesis, can help push through emboli and re-perfuse the retina, but the success of these techniques is quite limited. Therefore, the major goal in managing RAOs has always been to identify the underlying cause and prevent any future episodes, especially to the good eye.

RAOs are most commonly embolic in nature, but they may also result from vasculitic disorders or hypercoagulopathies.[1] Historically, the recommended workup included an examination with the patient's general practitioner, carotid artery ultrasound,

echocardiogram and lipid profile within one to two weeks in an outpatient setting.[1] Additionally, an ESR and CRP were recommended if patients presenting with CRAO were older or present with symptoms suggestive of GCA as well as evaluation of coagulopathies when suspicious.

However, a number of recent studies reveal a significant link between retinal artery occlusion and acute stroke. In one study, 24.2% of subjects who presented with retinal artery occlusion had acute brain ischemia concurrently, where most of the infarction patterns were small, multiple and scattered.[2] More alarming still, 37.5% of these subjects did not present with any neurologic symptoms or findings.[2] In another study, researchers looked at the increased incidence of acute stroke and found similar results, with the peak incidence of these events occurring one to seven days after the presentation of the RAO.[3]

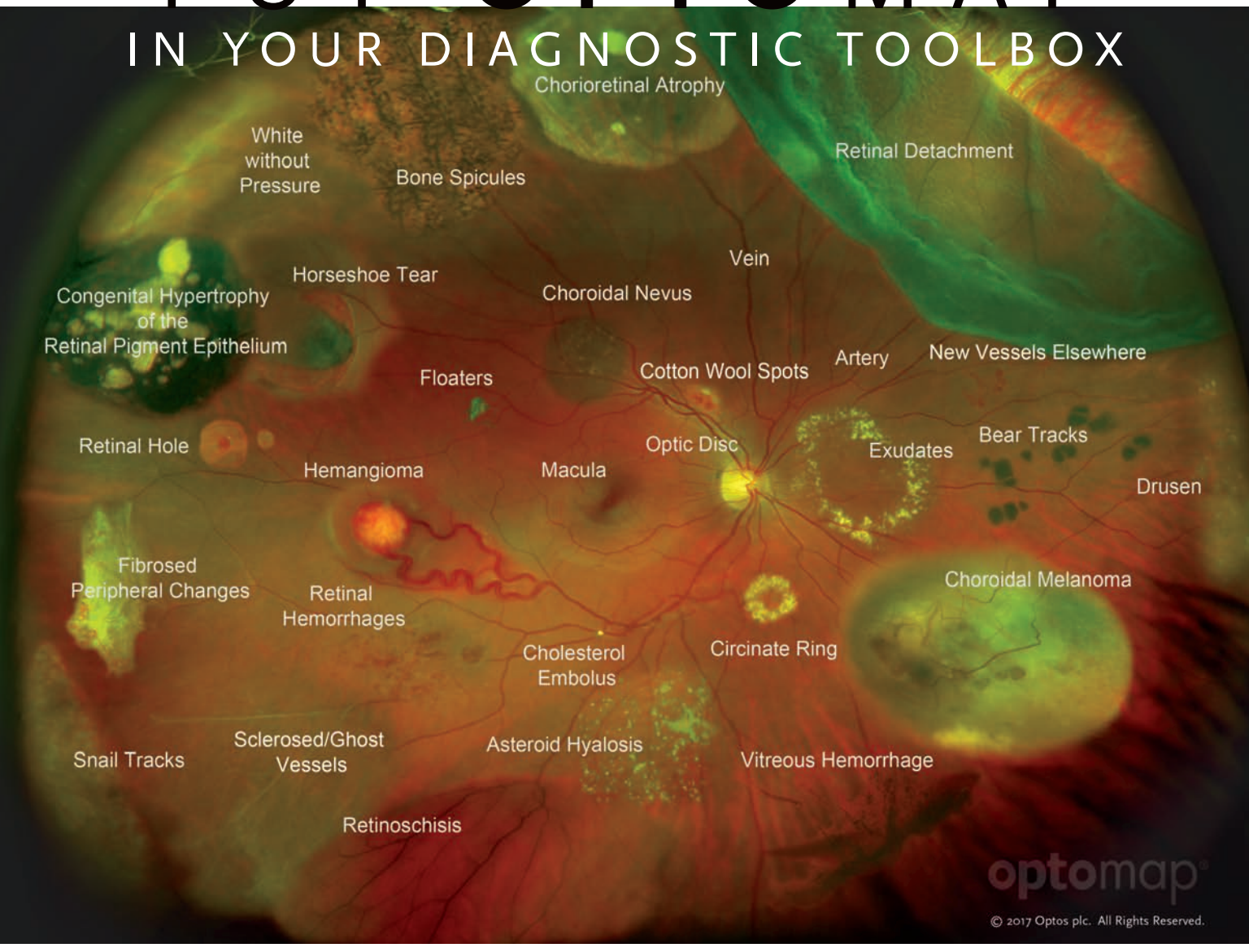
In light of these new findings, our management of retinal artery occlusion has changed significantly. Given the high risk and timing of acute CVA, instead of working up these individuals as outpatients, we are now sending them to the hospital for emergent admission and evaluation for stroke. Additionally, their workup is now much more extensive. These patients will require an MRI of the brain, MRA of the head and neck, echocardiogram, serologic studies to identify cardiovascular risk factors and neurology and cardiology consultations.

Workup for vasculitis and coagulopathies should also be performed when suspicious. Because the co-occurrence of RAO and CVA may not always be apparent, we strongly recommend eye care providers and hospital physicians communicate with one another for guidance regarding evaluation and management.

Although our patient went to the emergency room, often patients experiencing retinal artery occlusion will present to the optometrist's office first. It is our responsibility to not only manage the RAO and any ocular morbidities but to also refer these patients for emergent stroke evaluation. ■

1. Varma DD, Lee AW, Chen CS. Central retinal artery occlusion: general overview, diagnosis, and treatment causing sudden vision loss, this rare but troubling condition is linked to cardiovascular disease. Ret Physic. 2013;10(9):24-9.
2. Lee J, Kim SW, Lee SC, et al. Co-occurrence of acute retinal artery occlusion and acute ischemic stroke: diffusion-weighted magnetic resonance imaging study. Am J Ophthalmol. 2014;157(6):1231-8.
3. Park SJ, Choi NK, Yang BR, et al. Risk and risk periods for stroke and acute myocardial infarction in patients with central retinal artery occlusion. Ophthalmology. 2015;122(11):2336-43.

PUT OPTOMAP
IN YOUR DIAGNOSTIC TOOLBOX



Saving Site. Saving Lives.

- **Only optomap®** provides up to a 200° view of the retina in a single capture in less than half a second
- **The only** ultra-widefield, ultra-high definition 3-in-1 color depth and autofluorescence retinal imaging device
- Simultaneous evaluation of the peripheral and central retina
- Clinically supported by more than 400+ studies

Interested in adding ultra-widefield and ultra-widefield *af* capabilities to your practice?

Contact us today **800-854-3039**
or email **BDS@optos.com**

Nikon A Nikon Company

optos®
Building *The* Retina Company
optos.com

Advanced Refractive Solutions for Today's PRESBYOPIC PATIENT

An overview of the latest refractive surgical options available for presbyopes and strategies to comanage patients. ***By David Geffen, OD***

Presbyopia is on the rise as the population continues to age. An estimated 1.272 billion people worldwide were presbyopic in 2011, up from 1.04 billion in 2005.[1,2] By 2020, this number is projected to increase to almost 1.4 billion.[2] U.S. Census Bureau figures suggest that 112 million Americans had presbyopia in 2006, with increasing prevalence over the last decade.[3]

These rising numbers of presbyopes are impacting eye care practices around the country from a clinical and practice standpoint.

For one thing, presbyopic and older patients bring with them a variety of other eye issues, including cataracts, macular degeneration and diabetic retinopathy—translating into busier practices than ever. In addition, the vision care demands of an older population tend to be higher than those of younger patients, based on our experiences at the Gordon Schanzlin New Vision Institute in San Diego.

Amplifying this presbyopic patient growth is the fact that numbers of ophthalmologists entering the field will be relatively flat over the next three years.[4,5] The supply of active ophthalmologists is expected to increase by just 2% through 2020, although associated ophthalmologist manpower needs are expected to jump by 28% in the same period of time.[4,5] This provides an opportunity for optometrists to fill the gap of routine patient eye care.

LIFESTYLE NEEDS OF THE PRESBYOPIC PATIENT

Today's presbyopic patients have higher expectations than in the past about maintaining the freedom of movement they have been accustomed to in their daily activities. We are finding that Baby Boomers and Generation Xers are used to active lifestyles and insist on nothing short of exceptional vision to keep up their busy schedules.

Due to the prevalence of digital devices, many individuals—presbyopes included—are looking at digital screens for many hours per day. Among the younger population, a staggering 73% of Millennials (ages 18 to 35) are already reporting symptoms of digital eye strain.[6] Among Generation Xers (ages 36 to 51), 65% of adults in their 40s spend more than five hours a day on digital devices with 66% experiencing symptoms of digital eye strain; and among Baby Boomers (ages 52 to 70), 63.9% of adults in their 50s are reporting these symptoms as well.[6] This phenomenon may partially explain why we are seeing presbyopic symptoms at younger ages.

Today's presbyopes also want to maintain a youthful appearance. For example, some of our patients have undergone cosmetic procedures such as Botox and body sculpting. And many already have had refractive procedures with the goal of getting rid of their eyeglasses. However, frequently they are disappointed when we tell them that they may still

Release Date: June 2017

Expiration Date: June 15, 2018

Goal Statement: On completion of this educational activity, participants will be better informed about the latest refractive surgery options available in the United States and be able to educate their presbyopic patients on new refractive solutions.

Faculty/Editorial Board: David Geffen, OD

Credit Statement: This course is COPE approved for 2 hours of CE credit. COPE ID is **53790-RS**. Please check your state licensing board to see if this approval counts toward your CE requirement for relicensure.

Joint-Sponsorship Statement: This continuing education course is joint sponsored by the University of Alabama School of Optometry.

Disclosure Statement: Author **Dr. Geffen** is a consultant for Alcon, AMO, Annidis, Bausch + Lomb, eyeBrain Medical, Krypton Vision, ReVision Optics, TearLab, TLC Vision and Vmax Vision, and he is an investor in Bruder Healthcare. Editorial staff members **Jill Hoffman** and **Jack Persico** have no relationships to disclose.

Administered by
Review of Optometry ®

COPE® Approved

Supported by an unrestricted educational grant from
ReVision Optics

need to wear readers. Often, the first question I get is: "Can't I get more LASIK?"

Furthermore, many presbyopes are grappling with the psychological effects of getting older and struggling with their vision, possibly more so than in the past as a result of the ubiquitous presence of digital devices. One study found that presbyopia was associated with worse vision health-related quality of life than emmetropia in younger individuals.[7]

As such, it is clear that presbyopes face a host of issues when they visit the eye care professional today. As this growing number of patients with their unique vision and refractive needs come to see us, we need to be ready to meet the demand with new and creative solutions.

HALLMARK SYMPTOMS & SIGNS OF PRESBYOPIA

Presbyopia—the inability to focus at near distance due to a refractive problem—occurs naturally in people as they age. As the lens hardens over time, the eye is not able to focus light directly onto the retina. Aging also affects muscle fibers around the lens, making it harder for the eye to focus on close objects, so the lens forces light to concentrate behind the retina, resulting in poor vision for near objects.

As with many eye conditions, the key to uncovering patient problems begins with the optometrist asking the right questions. Early presbyopia symptoms characteristically will include the following presentations, so plan to ask patients about these indications:[8]

- Blurred vision and the inability to see fine details at near distances due to decreased amplitude of accommodation.
- Ocular discomfort or eyestrain after prolonged near work due to the tromboning effect of images moving in and out of focus.
- Minor headaches, squinting, fatigue or drowsiness from near work related to contraction of the orbicularis muscle or portions of the occipitofrontalis muscle. As a result of the physical effort needed for accommodation to maintain clear near vision, patients can also become frustrated and tense.
- Diplopia associated with increased exophoria and decreased positive fusional vergence amplitude.
- Need for brighter reading light to force pupillary constriction for greater depth of focus.

It is important to ask patients about their symptoms and how these physical manifestations are affecting their lives. Many of our patients subconsciously stop doing many near work activities to avoid experiencing symptoms. Prescribing visual solutions to aid near vision (e.g., reading glasses, multifocal contact lenses, multifocal intraocular lenses [IOLs] and corneal inlays) shortly after diagnosis can help patients maintain their daily lifestyle, perform at higher levels and be more productive.

LIMITATIONS OF EXISTING DISTANCE CORRECTION

With that said, presbyopic distance correction—including bifocal, trifocal and progressive spectacles; and multifocal and monovision contact lenses—is limited by various factors.

To start with, though customized progressive spectacles or presbyopic contact lenses can provide satisfactory near and distance vision to presbyopes without the potential risks of surgery, they cannot restore the true accommodation process of a younger individual.[9]

And while monovision correction of presbyopia is associated with vision health-related improvements, life quality measures are still rated worse than those of younger subjects with emmetropia in several areas, according to the findings of a 2003 study.[7]

From a patient satisfaction perspective, some patients tell us they are unhappy with their existing presbyopic correction because they think their eyeglasses make them look older. Others say they feel restricted by the use of eyeglasses or contact lenses, and seek the freedom they had as emmetropes or with previous refractive surgery. Yet other patients dislike the compromise in their distance acuity with monovision correction.

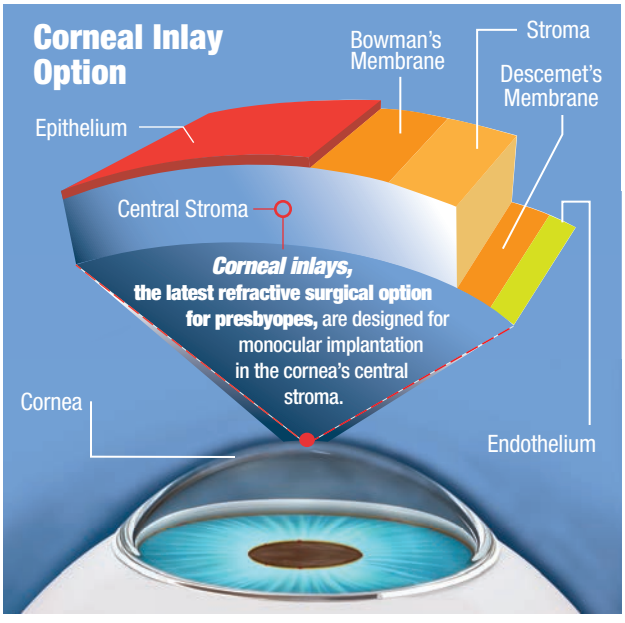
As a result of many of these complaints, we frequently get inquiries from presbyopic patients about possible surgical techniques to improve their vision.

TRADITIONAL SURGICAL OPTIONS FOR PRESBYOPES: WHERE MONOVISION FALLS SHORT

A number of factors have prevented widespread acceptance of traditional surgical correction for presbyopia.[9] These include: the fact that the procedures are more invasive; do not consistently produce high-enough quality vision; have the potential for resulting optical and visual distortions, regression of effects and complications such as corneal ectasia and haze, and anisometropia after monovision correction; and run the risk of impairment to distance vision.[9]

That being said, conventional surgical procedures to help presbyopes regain near vision include:

- ***LASIK (laser in-situ keratomileusis) and PRK (photorefractive keratectomy).*** These refractive surgeries to correct myopia, hyperopia and astigmatism have been in existence for nearly 20 years. However, for near correction, monovision (i.e., correcting one eye for emmetropia and the other eye for myopia) has been the primary solution available for these procedures. With monovision, we find that too great of a difference between the eyes can result in eyestrain and visual compromise in our patients.



- ***PresbyLASIK With Modified Monovision.*** This aspheric modification of the cornea under a flap (i.e., PresbyLASIK in one eye) uses an excimer laser to create a multifocal cornea by inducing higher-order aberrations that increase depth of focus.[10] The procedure may require the patient to continue wearing reading glasses, though to a lesser extent. We have found the most common complaint associated with modified monovision is slight distance blur. This is because the PresbyLASIK eye is corrected to be slightly myopic so patients have better near vision. At our practice, we have not found PresbyLASIK or PRK to be reliable solutions for our patients and rarely recommend them today.
- ***Multifocal LASIK.*** This procedure corrects presbyopia by using an excimer laser to reshape the cornea into different zones for near, intermediate and distance vision. An individual's brain selects the given zone that will yield the sharpest vision, depending on the distance of the perceived object. In each zone, light is refracted differently, similar to how multifocal contact lenses correct presbyopia, to restore good vision at all distances. Multifocal LASIK can be performed bilaterally or for modified monovision correction.

EVOLVING SURGICAL PROCEDURES FOR PRESBYOPES: IOLS GAIN TRACTION

Multifocal or accommodating IOL implantation can be a positive alternative for older presbyopes and higher hyperopes. However, many practitioners consider IOLs too invasive for individuals in their mid-40s to early 50s with a healthy crystalline lens, so, generally, we only recommend them for patients over 55 years of age—leaving a large group of presbyopes ineligible.[10] In addition, many patients perceive refractive lens exchange to be more invasive than other refractive surgeries, so the audience for these procedures is quite specific.

In spite of these stipulations, IOL adoption has been rapid in the past five years. Available IOLs include:

- ***Refractive IOLs.*** These IOLs create several focal points with concentric zones of varying optical power.[11] Since the aperture of each zone differs, image quality depends on pupil size, and reactivity to light and accommodation.[11]
- ***Accommodating IOLs.*** These IOLs fall into two categories: single-optic and dual-optic. Single-optic IOLs alter image focal points through anterior movement of the IOL and changes in the lens architecture.[11] To enhance the range of accommodation, dual-optic systems use two lenses: an anterior high plus lens coupled to a posterior minus lens; as the distance between the two lenses changes, optical power is altered.[11]
- ***Multifocal & Toric Multifocal IOLs.*** These IOLs are growing in popularity, and new, lower-add multifocal lenses help reduce the size of potential haloes. The IOLs are often better tolerated than older generations with +4D add powers, and we are having good success with these in our practice.

THE LATEST SURGICAL OPTIONS FOR PRESBYOPES: INLAY ADVANCEMENTS

The latest surgical option—corneal inlay implantation—has been available since 2015 in the United States.[12] Corneal inlays have several advantages over other refractive procedures. For one thing, the inlays are an additive technology that can be removed in the event of patient dissatisfaction, a complication or onset of other conditions. In addition, the procedures do not remove any tissue, so patients potentially can be candidates for future surgical solutions. And compared to lens surgery, the insertion procedure is less invasive. Plus, depending on the inlay, near correction often remains effective as presbyopia advances.

The three styles of corneal inlays, all designed for monocular implantation in the non-dominant eye, are: corneal reshaping inlays, refractive inlays and small-aperture inlays.

Currently, two corneal inlays are FDA approved, a third is in Phase III trials and a fourth is in development:

- ***KAMRA Near Vision Inlay* (AcuFocus).** Approved by the FDA in April 2015, this thin (5µm), small-aperture inlay is placed in a pocket in the corneal stroma to increase the patient's depth of field and improve near vision, but only minimally impact distance vision.[13] The opaque ring is 3.8mm wide with a 1.6mm aperture, and the inlay, which houses 8,400 laser holes to facilitate oxygen and nutrient transfer through the cornea, is placed in the non-dominant eye. Similar to a pinhole, near light rays coming through the aperture are focused clearly on the retina. Distance vision is often slightly decreased, typically by two to three lines. If the patient is unhappy with the procedure, the surgeon can remove the inlay, and vision should return to close to the preoperative state.

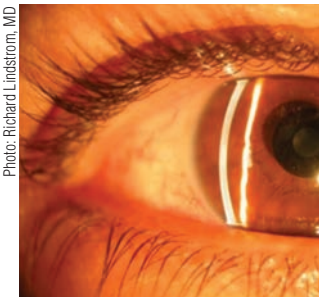


Photo: Richard Lindstrom, MD

FIGURE 2. The KAMRA Near Vision Inlay

The KAMRA inlay typically is implanted in the non-dominant eye during an outpatient procedure. It is placed within the first few layers of the cornea and centered over the pupil.

The KAMRA inlay is indicated for intrastromal corneal implantation to improve near vision by extending the depth of focus in the non-dominant eye of presbyopic patients between the ages of 45 and 60 years of age who have a cycloplegic refractive spherical equivalent of +0.50D to -0.75D with ≤0.75D of refractive cylinder. These patients do not need eyeglasses or contact lenses for clear distance vision, and require near correction of +1.00D to +2.50D of reading add. The ideal patient for this inlay has slight myopia but good distance vision.

The recommended postoperative care regimen includes:

- A broad-spectrum, topical ophthalmic antibiotic QID for a minimum of one week.
- Steroid ophthalmic suspension QID for the first postoperative week, TID for the second week, BID for the third week and QD for the fourth week.
- Preservative-free artificial tears QID for up to one month and continued as needed.
- Punctal plugs may be inserted at any time as needed.

Clinical study results demonstrated an improvement in uncorrected near vision (at 40cm) with the KAMRA inlay.[14] The uncorrected intermediate vision (80cm) was slightly improved, while the uncorrected distance vision (6m) in the implanted eyes was slightly decreased.[14] The mean changes at 12 months from baseline were: three lines in uncorrected near vision, one line in intermediate vision and half a line in distance vision.[14]

The effectiveness of the inlay was primarily assessed by monocular uncorrected near vision, and the endpoint target was 75% of subjects with 20/40 or better uncorrected vision in the inlay eye at 12 months post surgery.[14]

Preoperatively, none (0/508) of the subjects could see 20/40 or better at near without correction in the eye planned for inlay implantation. At 12 months post surgery, 83.5% (399/478) of subjects had 20/40 or better uncorrected near vision.[14] This increased to 87.2% (380/436) at 24 months, 87.1% (363/417) at 36 months and 87.1% (175/201) at 60 months.[14] The study successfully met the primary effectiveness criterion of 75%, with the lower 95 CI bound at 79.8% at 12 months post surgery.[14]

- ***Raindrop Near Vision Inlay* (ReVision Optics).** Approved by the FDA in June 2016, this corneal reshaping inlay is made of a hydrogel material and inserted under a flap, similar to LASIK. About 2mm in diameter, 30µm thick and made of approximately 80% water, the inlay is the same refractive index as the cornea. When placed in the cornea, it changes the shape of the central cornea, simulating a multifocal contact lens.

The Raindrop inlay is indicated for use in patients 41 to 65 years old, who have not had cataract surgery, are unable to focus clearly on near objects or small print, and need reading glasses with +1.50D to +2.50D of power. These patients do not need eyeglasses or contact lenses for clear distance vision.

Ideal candidates are those with slight hyperopia (+0.50 to 1.00D) with good distance acuity. Patients cannot have experienced changes in their distance vision within the last year, and they must have healthy eyes. The range of prescription is +1.00D to 0.50D with less than 0.75D of cylinder.

If the patient responds well to a trial contact lens and meets preoperative requirements, the surgeon can go forward with surgery. The inlay is implanted in the non-dominant eye and should only minimally affect distance acuity. If the patient is unhappy or other complications occur, the implant can be removed, and the cornea should return to its preoperative condition.

Photo: John Hovanesian, MD

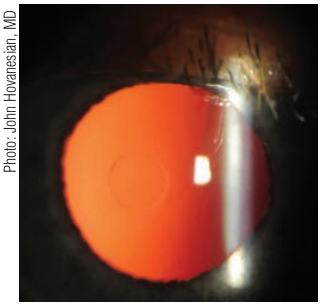


FIGURE 3. The Raindrop Near Vision Inlay
The Raindrop Near Vision Inlay, indicated for placement in the non-dominant eye to improve near vision of patients, is less than one-tenth of an inch in size and 2mm in diameter.

On average, we have found that patients gain five or more lines of near acuity and about 2.5 lines of intermediate by one week. In studies, at the 24-month visit, 87.5% of patients were 20/25 or better for near, and 76.2% were 20/25 for intermediate.[16]

- ***Flexivue Microlens* (Presbia).** Currently in Phase III FDA trials, this hydrophilic acrylic, variable-power inlay has a 3mm diameter, 0.015mm/15µm edge thickness and a plano central zone with increasing rings of higher power. Functioning similar to a multifocal contact lens, the inlay comes in a range of powers. It is inserted under a flap or in a pocket in the non-dominant eye, and the surgeon can remove the lens and replace it with a higher power inlay as the patient becomes more presbyopic. The inlay is offered in powers ranging from +1.5D to +3.5D, in 0.25D increments.

Photo: Richard Lindstrom, MD

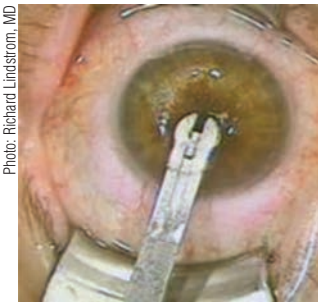


FIGURE 4. The Flexivue Microlens
The Flexivue Microlens is placed in a pocket created in the stroma with the aid of a proprietary insertion tool. The pocket self-seals and holds the lens in place at the center of the patient's visual axis.

Study results found that 12 months after implantation, uncorrected near visual acuity reportedly was 20/32 or better in 75% of operated eyes, whereas the mean uncorrected distance visual acuity of operated eyes statistically signifcantly decreased from 0.06 ± 0.09 logMAR (20/20).[17] Overall, higher-order aberrations increased, and contrast sensitivity decreased in the operated eye. No tissue alterations were found on corneal confocal microscopy, and no intra- or postoperative complications occurred.[17] Researchers concluded that the inlay appeared to be an effective way to address the corneal compensation of presbyopia in emme-

Postoperative Care for Raindrop Near Vision Inlay[15]

- One week of antibiotics QID.
- One month of a strong steroid (e.g., difluprednate) with a taper: QID for one week, TID at week two, BID at week three and QD at week four.
- Switch to a mild steroid (e.g., loteprednol) BID for the second month and QD for the third month.
- Preservative-free artificial tears daily for three months.
- The patient will need to wear an eye shield for up to four weeks to avoid rubbing the eyes during sleep.
- Patients should be instructed to avoid rubbing their eyes, wearing makeup, playing contact sports, exercising, swimming, gardening, smoking and being in dusty environments for at least the first week after surgery.
- Instruct the patient to call your office if any eye discharge or redness occurs, if there is a decrease in vision, or with the onset of flashing lights or floating spots.
- Plan to see the patient postoperatively at one day, one week, one month and then every six months, after which the patient should be seen for an annual eye exam or sooner if they experience any problems in the inlay-implanted eye.

Preoperative Procedures for Raindrop Near Vision Inlay

To help determine whether a patient is a good candidate for the Raindrop Near Vision Inlay, perform a complete eye exam and assess the patient's general health. Make sure to ask about all medical and eye conditions, and current medications, including over-the-counter products such as vitamins and supplements. Once candidacy is determined and the patient has decided to move forward with the Raindrop Near Vision Inlay:

- Have the patient try a contact lens similar to the inlay to be implanted or a high add multifocal soft contact lens for five days to see if the patient can adjust to the difference in vision between the two eyes once the inlay is implanted.
- Prescribe a steroid eye drop that the patient will begin using two days prior to surgery.
- Ask the patient to make arrangements to be driven home after surgery.
- Plan a postoperative and follow-up schedule.

tropic presbyopes between the ages of 45 and 60.[17]

• ***Icolens* (Neoptics).** Still in the early stages of development, this hydrophilic copolymer inlay has a 3mm diameter and an edge thickness of less than 15µm (depending on refraction). For presbyopia, the inlay offers powers ranging from +1.5D to +3D (in 0.5D steps). With no power in the center and positive refractive power in the periphery, this inlay's powers can be exchanged as presbyopia progresses. More information may come on this inlay later.

CORNEAL INLAY RISKS & WARNINGS

While research is revealing many positive vision outcomes associated with corneal inlays, it is important for eye care professionals to be aware of the possible risks and warnings associated with these new procedures. Some include the following precautions, including:[15]

- Possibility for new or worsening problems with glare, haloes, blurred or double vision, fluctuation of vision, dryness, foreign body sensation and pain.
- Possible decreased contrast sensitivity.
- Risk of infection, inflammation or both to the front part of the eye.
- Risk of developing a new dry eye condition or worsening of an existing condition.
- Risk of corneal complications.
- Possibility of cataract symptoms worsening or occurring sooner.
- Chance of decreased distance vision in the implanted eye.
- Risk of eye pressure elevation as a result of steroid eye drop usage to suppress inflammation from the procedure.
- Possibility for the need for more surgery to remove the inlay permanently or to exchange the inlay for a new one to treat a complication.
- Possible loss in best-corrected distance vision with eyeglasses or contact lens; in some cases, removal of the inlay will not restore pre-surgery vision.

Follow-Up Care for Corneal Inlays[15,18]

Follow-up care should be similar for all corneal inlays.

- Have the patient back postoperatively at: one day, one week, one month, six months and one year.
- Outcomes will vary by patient, but the day one visit still may be blurry due to edema.
- At one to three months, expect vision to stabilize.
- By six months, refractions and corneal topographic maps should be completely stable.
- Watch for any haze development, and treat aggressively.
- Monitor for dry eye symptoms because inlays perform best with a healthy tear film.
- At 12 to 24 months, assess biological compatibility, as some patients will develop an inflammatory reaction and may require steroids; in some patients, the inflammation may persist and require additional medications to calm the eye.

GROWING INTEREST IN ADVANCED REFRACTION TECHNOLOGIES

According to the 2015 American Society of Cataract and Refractive Surgery Clinical Survey, 70% of member respondents reported that new surgical techniques and technologies such as premium IOLs are topics their patients are most interested in learning about.[22] Members also reported

Latest Research On Cornea Inlay Technology

Research associated with corneal inlay solutions is revealing many positive safety and efficacy outcomes for these vision solutions.[19-21]

In one prospective, nonrandomized, multicenter FDA Investigational Device Exemption clinical trial that studied the one-year safety and efficacy outcomes of the Raindrop Near Vision Inlay, researchers reported that the inlay provided "significant improvement in near and intermediate visual performance," with no significant change in binocular distance vision or contrast sensitivity.[19]

As part of the trial, the non-dominant eyes (n=373) of emmetropic presbyopic subjects were implanted with the inlay at 11 sites; 340 eyes underwent one-year follow-up.[19] During these visits, on average, uncorrected near visual acuity (UNVA) improved by 5.1 lines, uncorrected intermediate visual acuity (UIVA) improved by 2.5 lines and uncorrected distance visual acuity (UDVA) decreased by 1.2 lines. From three months through one year, 93% of subjects achieved UNVA of 20/25 or better, 97% achieved UIVA of 20/32 or better and 95% achieved UDVA of 20/40 or better.[19]

Binocularly, the mean UDVA exceeded 20/20 from three months through one year.[19] Contrast sensitivity loss occurred only at the highest spatial frequencies, with no loss binocularly.[19] Absent or mild scores were reported in 96% of subjects for visual symptoms (i.e., glare, haloes, double vision and fluctuations in vision); in 99% for ocular symptoms (i.e., pain, light sensitivity and discomfort); and in 95% for dryness.[19] Adverse events were treatable and resolved. Eighteen inlays were replaced, usually soon after implantation because of decentration, but UNVA was barely affected in the group thereafter. In the 11 cases requiring inlay explantations, 100% achieved a corrected distance visual acuity of 20/25 or better by three months after explant.[19]

Another study found that the KAMRA inlay was "a viable treatment option" resulting in improved UNVA.[20] Researchers in the retrospective chart analysis evaluating six-month postoperative efficacy and safety outcomes in emmetropic presbyopic patients concluded that safety rates were high, while explantation and recentering rates were low.[20] They added that increased pocket depth could be associated with better postoperative outcomes.[20]

In addition, Flexivue Microlens corneal inlay visual outcomes for both near and distance vision were found to be satisfactory in another study, and the inlay did not appear to have significant effects on biometry or IOL power calculations.[21]

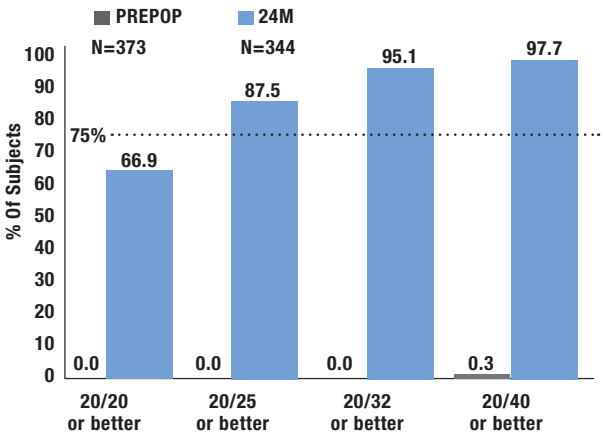


FIGURE 5. Monocular UCNVA At Preop And 24 Months Postop Visit*

The effectiveness of the Raindrop inlay for improving near visual acuity was assessed in emmetropic presbyopic subjects. The primary effectiveness endpoint was defined as improvement in uncorrected near visual acuity (40cm/16in) at 24 months postoperatively, i.e., 75% of eyes should achieve UCNVA of 20/40 or better. The primary effectiveness endpoint was met: More than 75% of subjects achieved 20/25 or better at the 24-month visit for near distance.

*Raindrop® Near Vision Inlay Professional Information Brochure. Available at: http://www.revisionoptics.com/wp-content/uploads/2016/09/710-0015-Rev-6-Artwork-FDA-Professional-Information-Brochure-US.pdf (last accessed May 26, 2017).

that rates of toric and presbyopia-correcting IOL adoption increased at a higher rate than cataract surgery volume during 2014.[22]

This growing interest in refractive technologies aligns with the fact that today's presbyopes have higher expectations than those in the past. Today, for example, we are finding that replacing a patient's cataract with an IOL and then giving the patient eyeglasses for full-time use is no longer acceptable for the average patient. Most presbyopes who come to us expect not to need distance correction after surgery, and many hope not to wear eyeglasses at all.

As well, more patients are doing their own research and are aware of advanced surgical procedures available to them, and they are asking about these options when they come in for visits. As a result, eye care professionals and the profession of optometry as a whole will serve an important role in educating our patients about these new procedures.

We need to be part of the initial conversation to inform our patients about evolving technologies and remain involved in ongoing care plans for patients who decide to go forward with refractive surgery. We find that our patients are interested in learning about advanced surgical options, but very often, they tell us that other eye care professionals never discussed these interventions that could potentially improve their vision outcomes.

APPROACHING THE PRESBYOPIC PATIENT ABOUT REFRACTIVE SURGERY OPTIONS

Eye care professionals owe it to their patients to proactively talk about all available solutions for vision correction. Nothing is worse than running into a patient at the store and finding out that the individual had a surgical procedure that we didn't know about. Often, when we ask these patients why they didn't consult with us first, they say we never brought up the topic of surgery so they didn't think we were knowledgeable in this area.

To avoid this unfortunate scenario, during your summary remarks with presbyopic patients, make sure that you mention new refractive surgical options such as advanced corneal inlay procedures, and why the patient may or may not be a candidate.

To intelligently communicate this information to patients, it is essential that you continue to learn about these advancing technologies. It is also imperative that you maintain good relationships with referring cataract surgeons and stay up-to-date on what procedures surgeons are recommending for your patients. You don't want your patients to think you aren't educated on this information and so seek it out on their own. Worse yet, you don't want to miss the opportunity to comanage your patients, and provide them with preoperative and postoperative care.

Tips for Partnering With the Right Cataract & Refractive Surgeon

Selecting the right corneal surgery partner is not always an easy decision, and your choice should be based on a variety of factors. Don't make the mistake of collaborating with a surgeon just because their comanagement fee is higher.

The best way to get to know a prospective partner is to visit the surgical center. Here are some tips to consider during your visit:

- First, see how surgical center staff members and surgeons treat their patients. This treatment will be a reflection on your office.
- Learn how the surgeon chooses the procedures that they perform on presbyopic patients so you can better guide patients.
- Watch a surgery being performed in order to be able to describe it to interested candidates.
- Make sure the surgeon can perform multiple procedures to ensure you can offer a variety of solutions to your patient base.

TALKING POINTS FOR YOUR PRESBYOPIC PATIENTS

When discussing the pros and cons of different procedures with presbyopic patients who may be candidates for refractive surgical options, it is important to maintain a balanced approach. However, if there are specific attributes of one intervention you feel strongly about, don't hesitate to tell the patient why.

We must provide patients with the fundamentals of each technology, so they can make an educated decision. At the same time, we need to ensure that we are sending our patients to a reputable surgery center that we trust will keep the patients' best interests in mind.

FUTURE OF REFRACTIVE SURGERY FOR PRESBYOPIC PATIENTS

Advanced refractive surgical solutions for presbyopes are in their infancy at this time, but they increasingly will play a larger role in all of our eye care practices. It is mission critical that we stay informed about these developments and continue to expand our knowledge about available refractive options for presbyopic patients.

Armed with this intelligence, we will be able to convey accurate information to prospective candidates and coordinate care with the cataract surgeon, helping to ensure that we follow these patients before and after surgery, and well into the future. ■

Assessing Patient Candidates for Refractive Surgery Options

Here are important considerations when evaluating a presbyopic patient for refractive surgery:[15,23]

- ***Assess the health of the ocular surface.*** This first step in determining candidacy for refractive surgery is crucial for corneal inlays since all inlays depend on great central optics of the cornea. Make sure the corneal surface is smooth and moist, and confirm that the patient has no active eye infection or inflammation.
- ***Examine the tear film.*** Look for any signs of dry eye and any meibomian gland dysfunction (MGD). Treating dry eye and MGD are a requirement before all corneal and lens surgeries.
- ***Pentacam and corneal topography.*** This sensitive, yet underutilized, instrument in practice today helps identify and characterize corneal disorders or irregularities. The Pentacam (Oculus) employs a Scheimpflug imaging system, an alternative to placido-based systems, and is essential before any refractive procedure. Don't forget to determine whether a patient has enough corneal thickness to safely undergo surgery.
- ***Thorough retinal exam.*** This includes optical coherence tomography to evaluate any retinal pathology or change before surgery.
- ***Gauge intraocular pressure.*** Verify that the patient does not have glaucoma or an uncontrolled buildup of pressure in the eye.
- ***Assess recent infections.*** Inquire as to whether the patient has had a recent herpes eye infection or complications from a previous infection.
- ***Determine diabetic status.*** Establish that the patient does not have diabetes.
- ***Evaluate mental state of the patient.*** This should not be overlooked. Make sure the patient has realistic expectations for success.

1. Frick KD, Joy SM, Wilson DA, et al. The Global Burden of Potential Productivity Loss from Uncorrected Presbyopia. Ophthalmology. 2015 Aug;122(8):1706-10.
2. Holden BA, Fricke TR, Ho SM, et al. Global vision impairment due to uncorrected presbyopia. Arch Ophthalmol. 2008 Dec;126(12):1731-9.
3. American Optometric Association. Care of the Patient with Presbyopia. Available at: http://www.aoa.org/documents/optometrists/CPG-17.pdf (last accessed January 31, 2017).
4. U.S. Department of Health and Human Services Health Resources and Services Administration Bureau of Health Professions October 2006. Physician Supply and Demand: Projections to 2020. Available at: https://bhw.hrsa.gov/sites/default/files/bhw/nchwa/projections/physician2020projections.pdf (last accessed March 7, 2017).
5. U.S. Department of Health and Human Services Health Resources and Services Administration Bureau of Health Professions December 2008. The Physician Workforce: Projections and Research into Current Issues Affecting Supply and Demand. Available at: https://bhw.hrsa.gov/sites/default/files/bhw/nchwa/projections/physiciansupplyissues.pdf (last accessed March 7, 2017).
6. The Vision Council: 2016 Digital Eye Strain Report. Eyes Overexposed: The Digital Device Dilemma. Available at: http://www.thevisioncouncil.org/sites/default/files/2416_VC_2016EyeStrain_Report_WEB.pdf (last accessed November 30, 2016).
7. McDonnell PJ, Lee P, Spritzer K, et al. Associations of presbyopia with vision-targeted health-related quality of life. Arch Ophthalmol. 2003 Nov;121(11):1577-81.
8. American Optometric Association. Care of the Patient with Presbyopia. Available at: http://www.aoa.org/documents/optometrists/CPG-17.pdf (last accessed January 31, 2017).
9. Papadopoulos PA, Papadopoulos AP. Current management of presbyopia. Middle East Afr J Ophthalmol. 2014 Jan-Mar;21(1):10-7.
10. Black S, Karpecki P, Brooker E, et al. New surgical options for presbyopia. Rev of Optom. 2015 June;152(6)35-42.
11. Crabb A, Krueger, R. The final cut: Surgical correction of presbyopia. Review of Cornea and Contact Lenses. 2014 Nov;151(8):20-6.
12. FDA approves first-of-its-kind corneal implant to improve near vision in certain patients. Available at: https://www.fda.gov/NewsEvents/Newsroom/PressAnnouncements/ucm443471.htm (last accessed April 12, 2017).
13. Cole J. A closer look at presbyopia correction. Rev of Optom. 2016 Feb;153(2):54-61.
14. Kamra® inlay professional use information. Available at: https://www.accessdata.fda.gov/cdrh_docs/pdf12/p120023d.pdf (last accessed March 31, 2017).
15. Raindrop Near Vision Inlay Patient Information Brochure. Available at: http://www.revisionoptics.com/wp-content/uploads/2016/08/Patient-Information-Brochure-710-0014-Rev-3-Artwork-FDA-Patient-Information-Brochure-US_To-Print.pdf (last accessed April 4, 2017).
16. Raindrop near vision inlay patient information brochure. Available at: https://www.accessdata.fda.gov/cdrh_docs/pdf15/P150034d.pdf (last accessed March 31, 2017).
17. Limnopoulou AN, Bouzoukis DI, Kymionis GD, et al. Visual outcomes and safety of a refractive corneal inlay for presbyopia using femtosecond laser. J Refract Surg. 2013;29:12-8.
18. Dalton M. Correcting presbyopia: Monovision or corneal inlays? Available at: https://www.eyeworld.org/article-correcting-presbyopia--monovision-or-corneal-inlays- (last accessed April 5, 2017).
19. Whitman J, Dougherty PJ, Parkhurst GD, et al. Treatment of presbyopia in emmetropes using a shape-changing corneal inlay: One-year clinical outcomes. Ophthalmology. 2016 Mar;123(3):466-75.
20. Dexl AK, Jell G, Strohmaier C, et al. Long-term outcomes after monocular corneal inlay implantation for the surgical compensation of presbyopia. J Cataract Refract Surg. 2015 Mar;41(3):566-75.
21. Stojanovic NR, Feingold V, Pallikaris IG, et al. Combined cataract and refractive corneal inlay implantation surgery: Comparison of three techniques. Refract Surg. 2016 May 1;32(5):318-25.
22. ASCRS Clinical Survey 2015. Available at: https://www.eyeworld.org/supplements/ASCRS-Clinical-Survey-2015.pdf (last accessed February 7, 2017).
23. Kojima R, and S. Eiden SB. What corneal shape reveals about corneal health. RCCL. 2016 May;30-35.

CE TEST

To obtain two hours of continuing education credit, complete the exam by recording the best answer to each self-assessment question online at: https://www.reviewofoptometry.com/ce/advanced-refractive-solutions-for-todays-presbyopic-patient Or, mail the Examination Answer Sheet on the next page to: Jobson Medical Information, Dept.: Optometric CE, 440 9th Avenue, 14th Floor, New York, NY 10001. A minimum score of 70% is required to obtain a certification of completion. There is no fee for this course.

1. By 2020, the number of cases of presbyopia worldwide number is projected to increase to how many?
a. 1.4 million
b. 1.4 billion
c. 10.4 million
d. 10.4 billion

2. The supply of total active ophthalmologists is expected to increase by what percentage between 2000 and 2020?
a. 2%
b. 10%
c. 25%
d. 35%

3. A 2016 report found that, among Generation Xers, 65% of adults in their 40s spend more than _____ hours a day on digital devices.
a. 1
b. 3
c. 5
d. 7

4. What percentage of adults in their 50s report symptoms of digital eye strain?
a. 43.9%
b. 53.9%
c. 63.9%
d. 73.9%

5. The following statement(s) is/are true about presbyopia:
a. Presbyopia is the inability to focus up close due to a refractive problem.
b. Presbyopia is the inability to focus at distance objects due to a refractive problem.
c. Presbyopia occurs naturally in people as they age.
d. Both a and c

6. What is not a symptom of presbyopia?
a. Red eye
b. Blurred vision
c. Ocular discomfort, eyestrain and headaches
d. Diplopia

7. What are reportedly limitations of traditional surgical correction for presbyopia?
a. Invasive nature of the techniques
b. Potential for optical and visual distortions
c. Potential for corneal ectasia and haze
d. All of the above

8. Monovision correction of presbyopia reportedly was rated _____ than/as that of younger subjects with emmetropia on several life-quality measures, according to a 2003 study.
a. Better
b. Worse
c. The same
d. None of the above

9. Which of these is *not* a conventional surgical procedure to help presbyopes regain near vision?
a. LASIK (laser in-situ keratomileusis) and PRK (photorefractive keratectomy)
b. PresbyLASIK With Modified Monovision
c. Trabeculectomy
d. Multifocal LASIK

10. Multifocal or accommodating IOL implantation is recommended for what age range of patients?
a. Over 40 years of age
b. Over 45 years of age
c. Over 50 years of age
d. Over 55 years of age

11. IOL implantation advancements include:
a. Refractive IOLs
b. Single-optic and dual-optic accommodating IOLs
c. Multifocal and toric multifocal IOLs
d. All of the above

12. What is *not* one of the three different styles of corneal inlays?
a. Small-aperture inlays
b. Large-aperture inlays
b. Corneal reshaping inlays
c. Refractive inlays

13. Which corneal inlays were FDA approved as of March 2017?
a. KAMRA Near Vision and Flexivue Microlens inlays
b. Raindrop Near Vision and Flexivue Microlens inlays
c. KAMRA Near Vision and Raindrop Near Vision inlays
d. Flexivue Microlens and Icolens inlays

14. Which corneal inlay was the first

CE TEST

approved by the FDA in April 2015?
a. KAMRA Near Vision
b. Raindrop Near Vision
c. Flexivue Microlens
d. Icolens

15. Which inlay is 30μm thick?
a. KAMRA Near Vision
b. Raindrop Near Vision
c. Flexivue Microlens
d. Icolens

16. What is *not* a recommendation for post-operative care of the Raindrop Near Vision inlay?
a. One week of antibiotics QID
b. One month of a strong steroid
c. Preservative-free artificial tears daily for three months
d. The patient should flush the inlay eye with water immediately after surgery

17. What are some possible risks and warnings associated with corneal inlay procedures?
a. New or worsening problems with glare, halos, and blurred or double vision,
b. Decreased contrast sensitivity
c. Risk of infection and inflammation to the front part of the eye
d. All of the above

18. What did respondents of the 2015 American Society of Cataract and Refractive Surgery Clinical Survey report about rates of toric and presbyopia-correcting IOL adoption during 2014?
a. They stayed the same
b. They decreased at a higher rate than cataract surgery volume
c. They increased at a higher rate than cataract surgery volume
d. None of the above

19. Considerations when evaluating a patient for refractive surgery include:
a. Assessment of the health of the ocular surface
b. Corneal topography
c. Evaluate intraocular pressure
d. All of the above

20. It is imperative that eye care professionals maintain good relationships with which referring doctors for refractive surgical patients?
a. Glaucoma specialists
b. Cataract and refractive surgeons
c. Retinal specialists
d. Cosmetic eye surgeons

Examination Answer Sheet

Valid for credit through June 15, 2018

This exam can be taken online at: https://www.reviewofoptometry.com/ce/advanced-refractive-solutions-for-todays-presbyopic-patient 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.

Advanced Refractive Solutions for Today's Presbyopic Patient

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

Mail to: Jobson Medical Information, Dept.: Optometric CE, 440 9th Avenue, 14th Floor, New York, NY 10001.

COPE approved for 2 hours of CE credit. COPE ID is 53790-RS

This course is supported by an unrestricted educational grant from ReVision Optics.

There is an eight- to 10-week processing time for this exam.

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:
A=Poor, B=Fair, C=Neutral, D=Good, E=Excellent

21. Provided me with a better understanding of the needs of presbyopic patients today. Ⓐ Ⓑ Ⓒ Ⓓ Ⓔ
22. Offered me a thorough overview of surgical correction options for presbyopia today. Ⓐ Ⓑ Ⓒ Ⓓ Ⓔ
23. Advanced my knowledge of corneal inlays available today. Ⓐ Ⓑ Ⓒ Ⓓ Ⓔ
24. Increased my awareness of preoperative and postoperative recommendations for presbyopic patients undergoing corneal inlay procedures today. Ⓐ Ⓑ Ⓒ Ⓓ Ⓔ
25. Presented me with possible risks and warnings associated with corneal inlay procedures today. Ⓐ Ⓑ Ⓒ Ⓓ Ⓔ
26. Gave me strategies for working with referring cataract and refractive surgeons who perform refractive surgeries for presbyopic patients. Ⓐ Ⓑ Ⓒ Ⓓ Ⓔ

Rate the quality of the material provided:
A=Strongly disagree, B=Somewhat disagree, C=Neutral, D=Somewhat agree, E=Strongly agree

27. The content was evidence-based. Ⓐ Ⓑ Ⓒ Ⓓ Ⓔ
28. The content was balanced and free of bias. Ⓐ Ⓑ Ⓒ Ⓓ Ⓔ
29. The presentation was clear and effective. Ⓐ Ⓑ Ⓒ Ⓓ Ⓔ
30. Additional comments on this course:

Please retain a copy for your records. Please print clearly.
You must choose and complete one of the following three identifier types:

① SS # ___ – ___ – ___

② Last 4 digits of your SS # and date of birth ___ – ___

③ State Code and License #: (Example: NY12345678) ___

First Name

Last Name

E-Mail

The following is your: ☐ Home Address ☐ Business Address

Business Name

Address

City

State

ZIP

Telephone # ___ – ___ – ___

Fax # ___ – ___ – ___

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 # 114916 RO-UAB-0617

Can an Eye Drop Eliminate PRESBYOPIA?

New therapies under investigation have the potential to radically alter your approach to this age-old problem. **By Jane Cole, Contributing Editor**

Three years ago, Douglas Devries, OD, of Sparks, Nev., was a guinea pig for a presbyopia-correcting eye drop that was in early development. At the time, Dr. Devries, who was on the drug's advisory panel, had very low nearsightedness in one eye and no distance correction in the other eye. After he put the drops in, not only did his near vision improve, but his distance vision sharpened as well, he says. The clarity was so "phenomenal," he drove around his Reno neighborhood to look at Christmas lights because his vision had never been better, Dr. Devries recalls.

"It was rather amazing. The effect lasted for over 12 hours. And it gave me probably the clearest distance vision I've ever had," says Dr. Devries. The drop worked by creating a miotic pupil (pinhole) without the typical side effects of ciliary spasm or brow ache, and the miotic pupil eliminated most of the higher order aberrations resulting in very clear vision, he adds.

Today, patients with presbyopia may be one step closer to correcting their vision by eye drop, as several different research teams are investigating this alternative, noninvasive treatment that some optometrists believe could be a game changer.

Photo: Felipe Vejarano, MD

Colombian researcher Felipe Vejarano, MD, a lead investigator for FOV Tears, instills the presbyopia-correcting drop into a patient's eye. Initial study results suggest she could be completely independent of her near vision correction for normal activities after using the drops for three months.

"I see successful presbyopia correction as the 'Holy Grail' of vision correction surgery and something as simple as an eye drop as the icing on the cake," says J. Christopher Freeman, OD, of Oklahoma City, Okla. "The need for presbyopic correction is a major point of visual dissatisfaction for patients, and to have nonsurgical options to eliminate the need for spectacle or contact lens correction would be a welcome addition to our presbyopia correction armamentarium."

Here's a snapshot of the new generation of presbyopia-correcting drops under development.

Reversing the Aging Eye

One promising new agent is EVO6 (Novartis), which is designed to restore crystalline lens flexibility and, hopefully, reverse the effects of aging in the eye.[1]

EV06 is a prodrug comprised of lipoic acid choline ester 1.5%, which penetrates the cornea and then breaks down into two naturally occurring substances: lipoic acid and choline, says Thomas Quinn, OD, of Athens, Ohio. The

lipoic acid then converts to the active agent dihydolipoic acid, which breaks down disulphide bonds in the lens, improving flexibility, he says.

EV06 may potentially halt or reverse lens hardening and, in turn, would allow the lens to maintain or regain its ability to accommodate.[1]

In a Phase I-II masked, placebo-controlled proof-of-concept study, 50 patients were treated daily for 90 days with topical EV06 and 25 patients with placebo. EV06 showed a statistically significant difference from placebo in distant-corrected near vision at all time points measured (from day eight); at day 90, 82% of participants treated with EV06 had 20/40 near vision vs. 48% in the placebo group.[2]

The results are promising, as EV06 was well tolerated with comfort scores equivalent to the control drop, with no loss in best-corrected distance vision, Dr. Quinn says.

"Many of the other tools we currently employ to aid the presbyopic patient require the visual system to adapt to an imbalance (monovision) or simultaneously share multiple points of focus," Dr. Quinn says. "Some brains deal with these optical adjustments just fine, while some struggle. With EV06, no such adjustment is required. Its optical effect is more like the natural focusing process we all are accustomed to early in life."

While early results are positive, there are still many unknowns, as the study was small in scope, "so we must be cautious until we see results following use by a greater number of subjects," says Dr. Quinn. "The big unanswered question is, 'How long does the effect

Finding a Niche in a Crowded Field

Presbyopes have myriad options, from tried-and-true readers, progressives and multifocal contact lenses to more invasive techniques such as IOLs and corneal inlays. If drops do make it to market, they could make significant waves in the presbyopia correction world.

Working in Tandem

Studies have yet to clarify how drops could perform under extreme circumstances such as in low light levels or small print, says Dr. Brujic. "I think in these cases, patients would likely still need progressives or reading glasses to help them see up close."

Dr. Freeman also believes patients may still require some assistance above and beyond the drops, such as a low-power pair of reading glasses or a very weak monovision or multifocal contact lens for those with early presbyopia. "This could be quite an advantage for a patient to not progress past early presbyopia until around the time of cataract surgery," he says. It's too early to say how drops may affect various cataract forms. "Nuclear sclerosis cataract progression, for example, might be delayed. This might allow for a longer time-value of laser vision correction, such as LASIK," bolstering that option, Dr. Freeman notes.

Let's Compare

Functionally, the big advantage of a presbyopia-correcting drop is that a patient is not dependent upon a device, Dr. Freeman says. Compared with contact lenses, there would be no sacrifice of visual quality to get light to focus at more than one focal point, not to mention overcoming overwear, irritation and other CL-associated problems, he adds.

Drops would also eliminate many problems associated with spectacle lens wear. There wouldn't be the limitation of only being able to see at near when looking through the lens, or frustrations of wearing them in situations where spectacles are inconvenient, such as outdoors, in inclement weather or for exercise, Dr. Freeman says.

In addition, drops avoid the potential risks of a surgical procedure such as an IOL or a corneal inlay, Dr. Freeman says. "Different surgical options have different limitations, too, such as only one eye providing the near and intermediate vision with corneal inlays. And presbyopia-correcting IOLs may have varying levels of visual quality changes depending on lens type and which one is chosen," Dr. Freeman says.

Patients could use drops when they wanted without making a permanent, invasive choice. "I think that's where the real appeal is. They try it, and if it works, great and then they continue. With a corneal inlay, that's a lot bigger commitment," Dr. Devries says.

A Good Partner for Contact Lenses

Dr. Brujic feels drops may actually help CL wearers stay in their contact lenses and even "help introduce more patients to contact lenses." Often, multifocal contact lens patients drop out because of perceived discomfort and visual limitations, he says. But patients could wear a single-vision lens along with the drops instead of multifocals, he adds. "When we look at presbyopes and their multifocal options, low-add patients are generally more successful than high-add patients because there's less discrepancy between the distance and near Rx," Dr. Brujic says. "So if we can take advanced presbyopic patients and improve their vision to a level where they really just need a low add, then we can increase their chances of becoming more successful in the contact lens. There are a lot of offshoot benefits to the drops once we get them in our hands and see what predicted benefits actually come to fruition."

Still No "Magic Bullet"

Despite the excitement over possible topical therapies, this approach would come with a familiar Achilles' heel: the potential for noncompliance. Lens-based methods of correcting presbyopia—imperfect though each may be—still beat the bottle on that score.

last?' We won't know that until studies currently underway are completed."

Due to the simplicity of the optics it regenerates, EV06 may provide some exceptional visual benefits, according to Dr. Quinn. However, in the study, nearly 20% of subjects could not see at the 20/40 level after three months. "Would that improve after using the drop a few more weeks or months? We don't know. If not, employment of one or more of the other options we now have at our disposal may be necessary," Dr. Quinn adds.

Although some optometrists are optimistically cautious, others think EV06 could create an explosive new category for presbyopes.

"Really, this could potentially be a game changer," says Mile Brujic, OD, of Bowling Green, Ohio. "This could open up a whole new world. It could ultimately change the way we think about treating presbyopia. Right now, we only know three-month results. In two or three years, we may find that the drop has the ability to alter the way the lens naturally ages."

As patients age, there are certain expectations, including the need for cataract surgery, and lens hardening and presbyopia are only precursors to this, Dr. Brujic says. "So, by actually adding some functionality back to the lens, we are reversing some of the natural aging process. The eye care field needs to be prepped for this because it's going to change the way we think about everything, and I think it's going to change things in a really good way."

The company expects pivotal phase III studies to begin in 2018 to evaluate long-term safety and duration of treatment, according to Dr. Freeman.

The Great Unknown

The promise of presbyopia correction by drop sounds good to many on paper, yet there are still unanswered questions and a few perceived inconveniences.

"The big question will be whether or not continued use will be required and thus continued expense over an extended period of time, or will a specific time of treatment be sufficient for correction, or even repeated courses of treatment, each for a certain amount of time," Dr. Freeman says.

Further research and cost analysis must better compare drops with the time-value of a surgical option, taking into consideration the risks and visual outcome of surgery, for quality comparison, he adds.

Another unanswered question is which patients would make ideal candidates, Dr. Freeman says. "We'll need to know more about comfort and tolerability as far as any adverse effects on the ocular surface. And in regard to the small-aperture optics of the bimonidine-carbachol drop, ocular scatter, especially as it related to any lenticular opacities, could be a limiting factor. The big advantage is that if it doesn't work, the patient simply stops using the eye drop, as long as no long-term detrimental effects are discovered."

And while drops offer a noninvasive solution to presbyopia correction, some patients may prefer the usual standbys of readers or progressives to avoid putting a drop in their eye.

A "Lifestyle Drop"

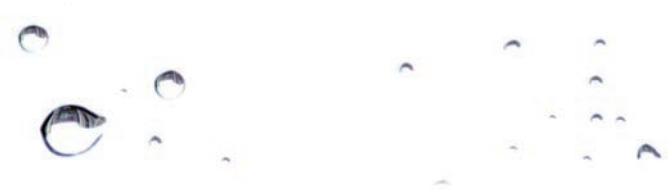
Presbyopia Therapies is moving forward with development of its Liquid Vision drops, a temporary presbyopia-correcting therapeutic designed to last five hours or longer. Liquid Vision recently completed its Phase IIa trial that studied dosing, safety and efficacy, and a Phase IIb trial is expected to have preliminary results by the end of the year.

Liquid Vision eye drops combine aceclidine (a miotic) with tropicamide (a cycloplegic) to create a "super pinhole" effect and moderate accommodation, according to the company. Aceclidine is used for creating the pinhole effect (between 1.9mm and 1.5mm in pupil diameter). However, by itself this creates strong accommodation, including ciliary spasm and distance vision blur. Tropicamide moderates accommodation, and the ratio and interaction between these two drugs is required to improve both near and distance vision at the same time, Jim McCollum, cofounder of Presbyopia Therapies, says.

The drop is relatively fast acting, with a 30-minute onset after application, according to Mr. McCollum. The drop is a binocular treatment and is designed to improve near and distance vision, Mr. McCollum says. In the early days of development, the company initially concentrated on miotics that constricted the pupil but also created the unwanted ciliary side effects. To prevent this, Presbyopia Therapies added tropicamide to help moderate accommodation and reduce the associated distance vision blur.

The drops are intended for daily use and provide a "lifestyle enhancement" as a complement to current treatment options, not a permanent replacement of spectacles or contact lenses, according to Mr. McCollum. "The idea is, the patient uses the drops in the morning, drives to work, waits about 30 minutes for the drop to take effect, and then it will last half a day or longer. So if you're working on the computer, you don't need correction," says Mr. McCollum. "If you need a second drop, you use a second drop. It's a temporary solution by design."

As Liquid Vision continues to move through FDA trials, the com-



pany hopes the drops will be on the market by 2021, according to Mr. McCollum.

Correction Buildup

FOV Tears, a binocular presbyopia-correcting drop, is now available in Colombia and is working its way through the approval pipelines in Argentina, Peru and Spain.

FOV Tears is a combination of a parasympathetic, alpha agonists 1 and 2, an anti-cholinesterase and an NSAID, according to Colombian researcher Felipe Vejarano, MD, a lead investigator for the drug.

The drop affects the ciliary muscle, which causes a physiological accommodation and a dynamic pseudo-accommodation. "This means a mild and dynamic miosis, which changes with light intensity."

A recent follow-up to a pilot study tracked patients who used the drop for three months.[3] Although small—only 14 emmetropic presbyopic subjects with an average age of 55—the study provided some promising results. The study found that while the duration of the drop's effect lasted four to five hours initially, duration increased to eight hours if the patient used the drop over time, says Dr. Vejarano. The onset of the drop's effects also accelerated with continual use, he says. While 25% of study participants said the drop took five to 10 minutes to take effect in week one, roughly 70% said it took only five to 10 minutes by the third month.[3] By the third month of use, 100% of participants said they were totally independent of their near vision correction for normal activities.[3]

Initial research found the drops improved near vision by two to three lines.[3] After three months, they found near vision improved by an additional one to two lines. Results showed distance vision increased by one line, and study participants had intermediate vision of 20/25 or better, according to Dr. Vejarano.

The initial research showed a few additional benefits, such as a decrease of 1mm Hg in patients' intraocular pressure with continued use.[3] The patient satisfaction survey also revealed patients felt the drops improved the uncomfortable sensation associated with dilation.[3]

Combination Correction

Researchers are also investigating a carbachol and brimonidine drop for presbyopia correction. In a recent pilot study, 10 naturally emmetropic and presbyopic subjects between the ages of 42 and 58 with uncorrected distance visual acuity of at least 20/20 in

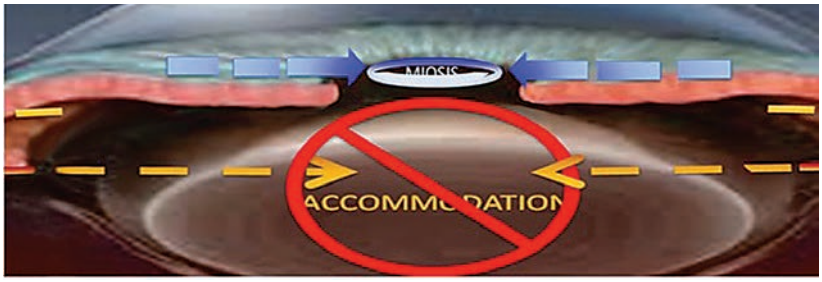
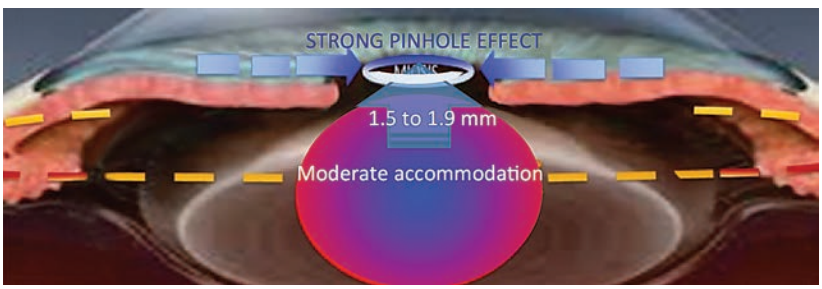


Image: Presbyopia Therapies

This image depicts the effect of a traditional miotic drug, pilocarpine, with pupil size at roughly 2.3mm. The drop provides some effect, but not a pinhole effect. Accommodation is achieved, but distance vision blur is present.



This image shows the results of a Liquid Vision drop. Aceclidine provides a stronger pinhole effect with the pupil less than 2mm. Tropicamide modulates the accommodation. Proponents say the combination/ratio of both drugs allows for improved near and distance vision at once, without typical side effects.

both eyes received 3% carbachol and 0.2% brimonidine in both combined and separate forms, 3% carbachol alone and 0.2% brimonidine (control) alone in their non-dominant eye.[4] Researchers found statistically significant improvement in mean near visual acuity in all subjects who received combined 3% carbachol and 0.2% brimonidine in the same formula compared with those who received separate forms, carbachol alone or brimonidine alone ($p < 0.0001$).[4]

The monocular pharmacologic treatment of presbyopia with one drop a day of carbachol/brimonidine in the non-dominant eye permits acceptable reading vision for many presbyopes, even older subjects, the researchers concluded.[4]

This drop constricts the pupil and increases depth of focus with the pinhole effect, similar to the Kamra corneal inlay (AcuFocus), according to Dr. Freeman. The disadvantage, he says, "is that it is only in one eye and the potential for reduced quality of vision or dimness sometimes is experienced with small-aperture optics." However, many patients are happy with their improved near and intermediate vision following Kamra implantation, with the dominant eye usually masking perceived dimming monocularly in the non-dominant eye, he adds.

"The difference [with Kamra] is, the patient's not paying for and using eye drops every day to enjoy that vision," adds Dr. Freeman. "Improvement could be significant enough that a patient may elect this treatment over wearing spectacles or contact lenses if they are highly motivated not to wear contacts or glasses."

Despite the small number and the heterogeneity of the patients involved in this pilot study, its findings suggest this drop is also promising.[4] Additional studies are planned with hyperopic and myopic presbyopes and in pseudophakic and post-LASIK presbyopes.[4]

Opportunity for Everyone

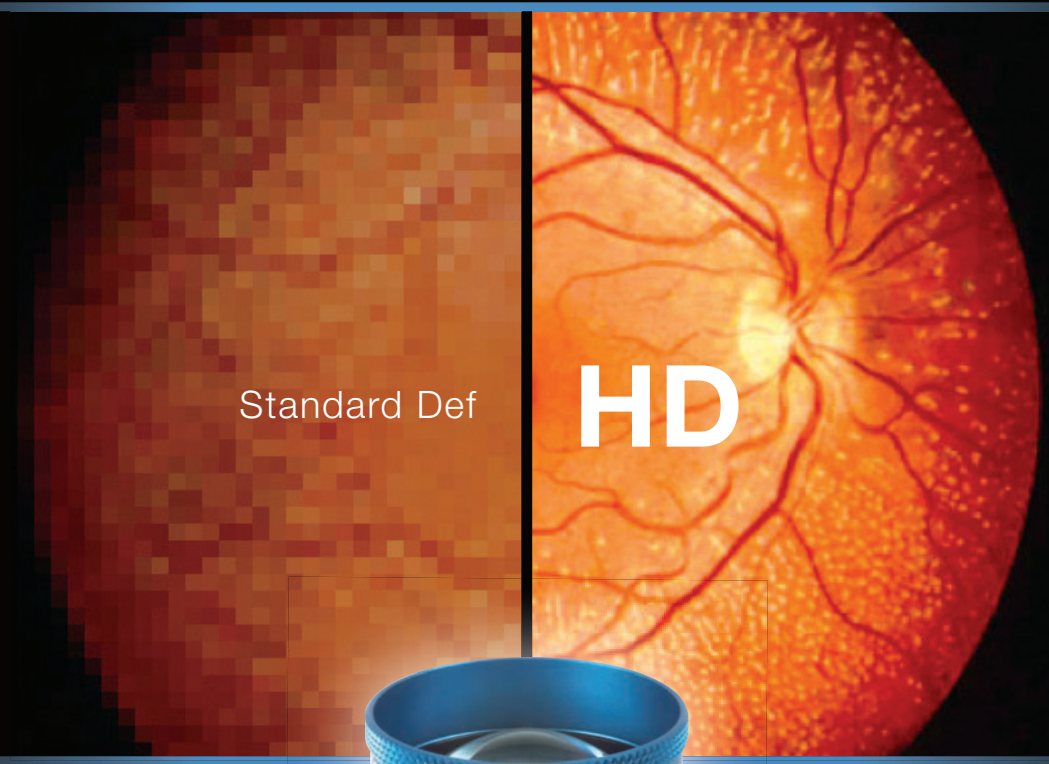
"I look at this as a remarkable opportunity," Dr. Brujic says. "My philosophy is very simple when it comes to an eye care practice. Do everything that is in the best interest of the patient. "With a drop that actually helps presbyopic symptoms, we can reduce dependency on presbyopic correction such as reading glasses and progressives."

Presbyopia-correcting drops could also open up a door to new patients who thought they only needed over-the-counter reading glasses, Dr. Brujic says. "Now we'll have the opportunity to help these patients because they're going to be coming in asking if they are candidates for the drop."

Patient interest in this advancement is "intense," Dr. Devries says. "I still have patients who say, 'You told me about a drop a few years ago that would let me see up close. Is it on the market yet?' So they continue to ask. I think the relevance of a presbyopia drop to patients is going to be extreme." ■

1. Encore Vision Announces Successful Phase I-II Study of Topical EV06 for the Treatment of Presbyopia. PR Newswire. May 5, 2016. www.prnewswire.com/news-releases/encore-vision-announces-successful-phase-i-ii-study-of-topical-ev06-for-the-treatment-of-presbyopia-300263690.html. Accessed May 1, 2017.
2. Novartis. Novartis bolsters ophthalmology pipeline through acquisition of Encore Vision, Inc. December 20, 2016. www.novartis.com/news/media-releases/novartis-bolsters-ophthalmology-pipeline-through-acquisition-encore-vision-inc. Accessed May 1, 2017.
3. Renna A, Vejarano LF, De La Cruz E, et al. Pharmacological treatment of presbyopia by novel binocularly instilled eye drops: A pilot study. Ophthalmol Ther. 2016 Jun;5(1):63-73.
4. Abdelkader A, Kaufman HE. Clinical outcomes of combined versus separate carbachol and brimonidine drops in correcting presbyopia. Eye Vis (Lond). 2016 Dec 5;3:31. [Epub].

[See the **Def**-erence]



VOLK® DigitalLenses

Volk's Best Exam Lenses

Highest Resolution, Magnification & Widest Field

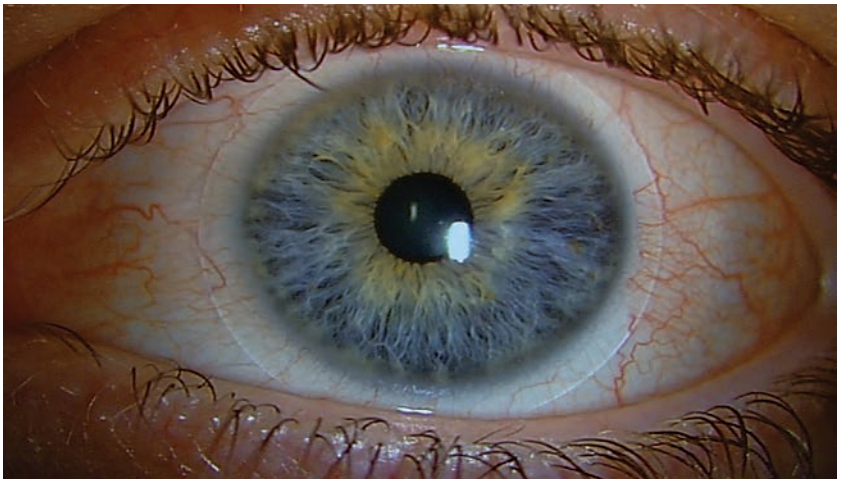
Free Engraving & Free Multi-Lens Case

Find out more at **volk.com**

Lifelong Contact Lens Success: Keep Allergy and Dry Eye at Bay

Armed with the right information, you can help patients of any age remain happy in their lenses. **By Heidi Wagner, OD, MPH**

Dry eye and allergy are common presentations in optometric practice, and they can wreak havoc on a patient's ocular comfort, especially for contact lens wearers. But with prompt diagnosis and management, astute clinicians can keep patients comfortable in their contact lenses, even in the face of allergy, dry eye or both.



A hypersensitivity or toxic reaction to the preservative in the contact lens solution is typically characterized by conjunctival injection and superficial punctate keratitis.

Know the Enemy

The first step to diagnosis is knowing what to look for. While allergy and dry eye often present with similar symptoms, their differing pathophysiology and prevalence among certain patient populations can help with the differential diagnosis.

Allergy. Recent reports suggest the prevalence of allergic conjunctivitis (AC) may be as high as 40%.[1] As with asthma, the increase in AC prevalence has been observed globally in adults as well as children.[2] Although seasonal AC is the most common presentation, chronic forms such as vernal and atopic keratoconjunctivitis contribute to the overall disease spectrum as well. While vernal keratoconjunctivitis has a predilection for males younger than 18 years, seasonal and perennial AC is often present in all genders from childhood through middle age.[3,4] Although symptoms associated with atopy tend to decrease with age—in opposition to dry eye—AC may persist well beyond middle age.[1]

Itching is generally perceived as the harbinger of allergies.[5] Milder presentations may exhibit relatively modest clinical signs. With more severe disease, a clinically consistent picture would include chemosis, conjunctival injection, eyelid edema or a combination of all three.[3] First-line treatment typically includes antihistamines (often in association with a mast cell inhibitor).[6] Over-the-counter (OTC) topical agents such as ketotifen fumarate administered twice daily are accessible to the patient at a relatively modest cost. Prescription medications potentially add convenience (once-daily dosing) and higher efficacy.[7] A "softer" corticosteroid with a lower propensity for side effects (ocular hypertension, cataract) may be added to the regimen on a short-term basis, particularly when the clinical signs are

Save *time, money, and space.*
Enhance *the patient's experience.*

TelScreen

EyeRes™
Digital Imaging Systems

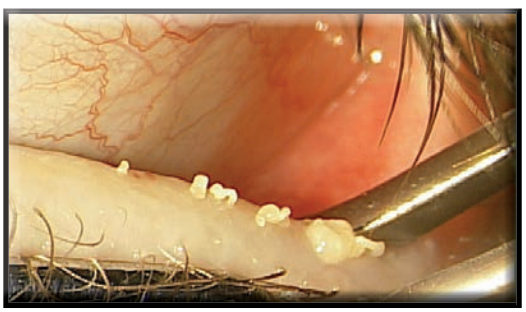
A versatile system:

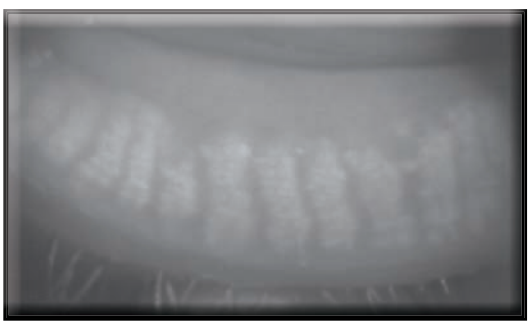
Anterior-segment imaging, Retinal imaging, and Infrared imaging from a slit lamp.

Focused on Slit Lamp Digital Imaging since 1993

Info@TelScreen.com

TelScreen's IR camera
Infrared Technology





A New Approach to Infrared Imaging

Other systems flood the eye with Infrared illumination. Just as our visible light systems don't need flash or background illumination, our Infrared imaging system does not require additional Infrared illumination.

Reap the many benefits of owning an **EyeRes**™ imaging system with TelScreen's IR camera.

Call* 800.769.4933 *today.

www.TelScreen.com

more severe.[6] Cold compresses and avoiding the allergen, when feasible, may be beneficial as well.[6,7]

Dry eye. In 2007, the International Dry Eye Workshop (DEWS) examined available data and reported potential risk factors for dry eye disease (DED).[8] DEWS retained the 1995 National Eye Institute/Industry Workshop classification of aqueous tear-deficient dry eye and evaporative dry eye.[9] Notably, the researchers found DED in 5% to 30% of the population aged 50 years or older with lower rates presumably reflecting patients with more severe disease and higher rates including patients with milder forms.[8] DEWS also found DED increases with age and is more prevalent in women.[8] In addition to age and gender, connective tissue disease, vitamin A deficiency, LASIK surgery, antihistamine use, radiation therapy, stem cell transplantation, androgen deficiency and a diet low in omega-3 essential fatty acids are consistently associated with the condition.[8] Less substantiated risk factors include other medications (tricyclic antidepressants, diuretics, beta-blockers), diabetes, systemic chemotherapy and penetrating keratoplasty. The association of DED with oral contraceptives, pregnancy and menopause, while cited, is less clear.[8]

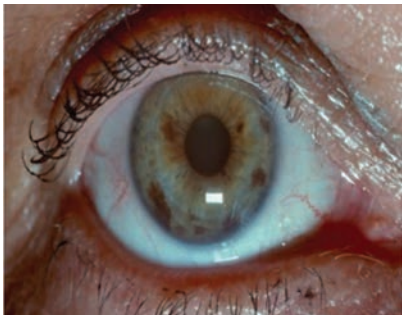
If itching is the harbinger of ocular allergies, burning is the hallmark of dry eye. Current practices emphasize management of evaporative, rather than aqueous tear-deficient, dry eye. Treatment strategies focus on the lipid layer of the tear film, which in turn protects the cornea from the hyperosmolarity associated with ocular inflammation. Initial intervention typically includes heat to enhance meibomian gland function, lipid-based artificial tears, nutritional counseling (omega-3 essential fatty acids) and lid hygiene.

Further management may include a short course of corticosteroids to more aggressively manage the inflammation and, as appropriate, jump start other ophthalmic agents such as cyclosporine A 0.5% and lifitegrast 5%. Oral antibiotics can provide additional anti-inflammatory effects. Heat masks (Bruder or ThermalOn) and lid hygiene products (OcuSoft and Avenova, NovaBay Pharmaceuticals) are useful in milder presentations of DED, while higher technology strategies, when accessible, may be necessary with more advanced disease. DED management is generally complementary to that of AC; however, clinicians should carefully balance the mechanical component of lid hygiene and the drying effects of oral antihistamines, as they can exacerbate symptoms of coexisting allergy/DED.

Contact lens discomfort (CLD). This remains the primary reason for discontinuation of lens wear, contributing to a dropout rate greater than 20%.[10-12] As with DED, clinical signs and patient symptoms of CLD often do not correlate. Patients with dry eye frequently have CLD, and lens wearers with CLD often present without clinical signs of DED.[13] In contrast to dry eye, the relationship between age and CLD remains an enigma. Some investigators suggest CLD is inversely associated with age, others report that younger wearers may have more intense dryness symptoms and some even suggest no association between CLD and age exists.[14-17] Regardless, factors that promote lens comfort are integral to lifetime contact lens success.

Managing Coexisting Disease

Caring for patients with allergy and dry eye becomes more complicated when the two conditions coexist. DEWS defined DED as "a multifactorial disease of the tears and ocular surface that results in symptoms of discomfort, visual disturbance and tear film instability with potential damage to the ocular surface. It is accompanied by increased osmolarity of the tear film and inflammation of the ocular surface."[8] Inflammation is also inherent in the expression of allergy, which is defined by its hypersensitive state following exposure to an antigen.[18] Other reports have also highlighted the connection between AC and dry eye.[19] For example, significant symptoms of itching in conjunction with dry eye suggest coexisting atopic disease.[19] Moreover, patients with AC who exhibit disruptions in tear film integrity, symptoms of burning or both may have coexisting DED.[8]



Adults may benefit from preemptive management of coexisting dry eye and allergy.

While acknowledging that AC and dry eye can coexist, one condition often overshadows the other when it comes to patient symptoms. The clinician can use the dominant symptoms to customize a treatment plan to the needs of each patient.

Lifelong Success

Clinicians may consider modifying a patient's contact lens wear regimen depending on the symptoms of AC and DED. The severity of the disease will help determine whether to initiate treatment prior to lens wear in neophyte wearers or whether lens wear should be discontinued in

Coding Connection

By John Rumpakis, OD, MBA, Clinical Coding Editor

Coding For Concomitant Conditions

When dry eye, allergy and contact lenses collide, be sure to keep your record straight.

Often, when focusing on medical record compliance and coding of a particular procedure, it is easy to get lost amid the rules, regulations and guidelines and forget about clinical care—more specifically, the patient. This may be particularly true for patients with more than one complication. Of course, these are precisely the patients who need your undivided attention the most.

Concurrent anterior segment conditions, for example, are quite common, particularly with dry eye and ocular allergy. One survey found 40% of the participants reported symptoms of ocular allergy at least once during the previous year, and researchers have noted dry eye may be present in as much as 30% of the US population.[1,2] Combine that with contact lens wear and you have the potential for clinical confusion, a sloppy medical record, over or under coding and lost revenue.

So let's set the record straight on how these concomitant conditions should be handled in the medical record, particularly in the era of health care reform where efficiency and effectiveness are paramount.

Forever Patients

Dry eye, allergy and contact lens wear are often looked at as annuity disease states—they require constant, continual management. When you have a patient diagnosed with dry eye, allergy or both, or they are contact lens wearers, it's important to record all of these conditions as reasons for the visit or chief complaint each time you see them for a scheduled follow-up. Your record should read something like: "contact lens patient returning per doctor-directed order for further diagnostic evaluation and follow-up for dry eye (or ocular surface disease) and allergic conjunctivitis. Additional symptoms noted since last visit are…".

The greatest specificity you can provide in the medical record leads to clinically appropriate case history, level of physical exam, medical decision-making and, ultimately, a more accurate code for the encounter.

It's not unusual to deal with a patient whose allergic response is elevated due to a compromised ocular surface, and whose contact lens wearing time is reduced or quality of vision is affected—all on the same visit. Does that mean you can code higher-level visits? The answer is yes and no.

Coding Just Got Complicated

Multiple diagnoses do play a role in elevating the level of an office visit by affecting the case history, the medical decision-making and, to a lesser degree, the physical exam, but only if you are recording items properly in the record (don't forget the concept of medical necessity).

Do not do anything to unnecessarily embellish the medical record simply to elevate the visit to another level. The same concept applies to additional point-of-care testing, such as MMP-9, osmolarity, anterior segment photography (including meibography) and topography, to name a few. For example, many practitioners follow a clinical protocol for certain disease states. However, it's often unnecessary to do every test within the protocol on every patient. A protocol should be viewed as a toolbox from which a physician chooses the test or tests that provide the information necessary to manage the case, not just acting in a confirmatory fashion.

Additionally, you should never embellish the medical record to justify the frequency of office visits throughout the year to manage these conditions.

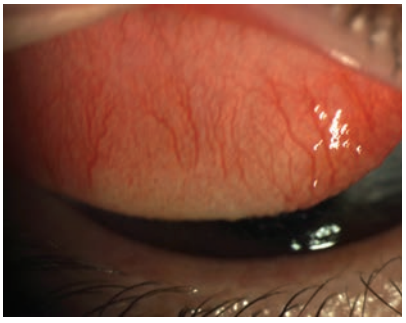
Practice Measures

The implementation of ICD-10 has provided all stakeholders with a very discrete metric by which practitioners are measured. While these three conditions are not specific measurement outcomes with respect to merit-based incentive payment systems, a practitioner can easily be measured by the number of office visits performed in a given period of time, providing an easy path to calculate the economics of disease states and the clinical efficiencies and effectiveness of a practitioner. Make sure you rely only on what you can support by the medical necessity noted in the record when you are calculating the economic upsides of these concomitant conditions.

Knowing how handle the medical record at the outset can keep your mind focused on what's important: the patient, who needs your expertise to help them maintain comfortable, productive lifestyles. ■

Send your own coding questions and comments to rocodingconnection@gmail.com.

1. Singh K, Axelrod S, Bielory L. The epidemiology of ocular and nasal allergy in the United States 1988-1994. J Allergy Clin Immunol. 2010;126(4):778-83.
2. The epidemiology of dry eye disease: report of the Epidemiology Subcommittee of the International Dry Eye Workshop. Ocul Surf. 2007;5(2):93-107.



Normally functioning meibomian glands are integral to a healthy tear film, while blockage can lead to evaporative dry eye.

established wearers. Modifying the replacement schedule or lens material may help combat allergy and dry eye symptoms. Daily replacement is often ideal, as it provides a clean lens surface and minimizes exposure to lens care products.

While patients often turn to OTC lubricants to relieve dry, itchy eyes, these agents can have both a positive and negative effect on contact lens wear. The vehicle can provide short-term relief of ocular symptoms, yet other ingredients such as preservatives may aggravate the ocular surface. Clinicians should recommend preservative-free or contact lens "approved" options to avoid toxicity often exacerbated by the chemicals binding to the lens material.

While consensus exists that lens material and comfort are intertwined, it is difficult to be prescriptive regarding lens materials. Despite advances in technology, our understanding of the impact of varying contact lens designs is limited.[20] Today's lens materials defy traditional FDA groupings, and older dogma relating to water content or even the inclusion of silicone is too simplistic to characterize recent technological advances. Consequently, practitioners must use their best professional judgment and consider the unique needs of each patient when prescribing contact lenses.

Beyond the lens material, preservative-free lens care regimens or multipurpose brands with known formulations are suitable choices, given the association of lens-solution interactions with adverse events.[21] Clinicians should caution patients on using store brands, which may not incorporate the newest products or may shift in formulation over time. Educate patients that the lens care system may influence comfort, particularly for sensitive wearers.

While some strategies are appropriate for all contact lens wearers, others are specific to certain ages and life phases:

Childhood. In the past, children younger than 10 were fit only when contact lenses were deemed medically necessary—as in the case of unilateral pediatric aphakia or other circumstances where amblyopia may result from high or anisometropic refractive error.[22] Recently, however, the interest in fitting children with contact lenses has shifted from correcting refractive error to slowing progression of myopia.[23] Given the prevalence of myopia and the potential benefits of intervening with contact lenses, this trend is likely to continue.[24,25]

Atopic conditions can be a concern in this group, given the propensity for allergies in pediatric patients.[18] Parents and other caregivers are integral to the successful management of both contact lenses and adjunctive therapy for allergies. Children generally will benefit from straightforward lens care regimens, avoidance of eye drops when possible and management of coexisting conditions.

Adolescence to young adulthood. Most providers are adept at managing these patients, as they are traditionally motivated to wear contact lenses and are often easy to please. This population is highly involved with activities away from home. While parents may oversee the care of a young adolescent between the ages of 10 and 14, independence increases with age and older adolescents ages 15 to 19 and young adults ages 20 to 24 learn to manage their own ocular health.[22]

Providers can work with teens (and parents) and young adults to schedule periodic follow-up for contact lens evaluations and anterior segment conditions. Patients who attend school out of town merit special consideration; they benefit from additional education in proactively scheduling health care visits, managing health insurance, ordering ophthalmic materials and accessing prescription medications. While dry eye is relatively rare in this group, atopy is common.[8,18] Furthermore, corneal inflammatory events are more prevalent in older teens and young adults than in younger or older contact lens wearers.[26,27] An ample supply of lenses (i.e., 'pantry effect'), back-up spectacles and instructions for accessing care when away from home go a long way to minimize the sequela of adverse events.[28] Older teens and young adult wearers may benefit from advice about risky behaviors that are associated with corneal infectious or inflammatory events and are more prevalent in this age group, such as water exposure, napping in contact lenses and overnight lens wear.[29]

Pre-presbyopic adults. These patients are also relatively easy to manage regarding vision expectations, access to care and patient education. As discomfort and dryness are the primary reasons for contact lens discontinuation, these patients often benefit from preemptive management of coexisting dry eye, allergy or both.[10,11] Moreover, the Vision Council reports that more than 60% of these patients

Biotruth #15

Hyaluronan (HA) can hold 1,000 times its weight in water[1]

Biotrue® uses HA to keep lenses moist for up to 20 hours[2]

Biotrue® keeps lenses comfortable all day long, with a unique bio-inspired formulation that **works like your patients' eyes**. The HA in Biotrue® attracts water to the lens surface and envelops the lens in a moisture-rich cushion. No wonder Biotrue® is the multi-purpose solution used by more patients.*

3 BIO-INSPIRED INNOVATIONS

- Matches the pH of healthy tears (7.5)[1]
- Has hyaluronan (HA), a lubricant found naturally in the eyes, helping to provide up to 20 hours of moisture[2]
- Keeps key beneficial tear proteins such as lysozyme active[1]

Let patients know their solution matters.

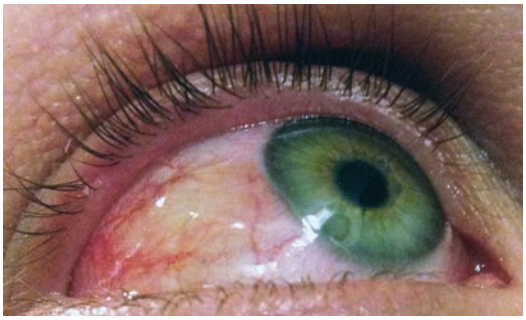
Recommend Biotrue® multi-purpose solution.

For more information, call 1-800-828-9030 or visit Bausch.com/ecp

REFERENCES: 1. Data on file. Bausch & Lomb Incorporated. Rochester, NY. **2.** In vitro studies evaluated the rate of release of sodium hyaluronate (HA), a conditioning agent in the BPZ02 multi-purpose solution, from both conventional and silicone hydrogel contact lenses over a twenty-hour time period. HA was adsorbed on all traditional and silicone hydrogel contact lenses tested upon soaking in the solution overnight. HA is then released from the lenses throughout at least a twenty hour time period when rinsed with Hank's balanced salt solution at a rate mimicking tear secretions. The in-vitro performance of BPZ02 multi-purpose solution suggests that it will provide lens conditioning throughout a twenty hour time period.

*Highest household penetration among multi-purpose solutions; IRI Panel 52 weeks ending 12/25/16.

BAUSCH+LOMB
See better. Live better.



Ocular allergy often includes chemosis, conjunctival injection and eyelid edema.

report digital eyestrain and they may be candidates for contact lenses designed for digital device users.[30]

Pregnancy. This has been associated with contact lens intolerance and changes in refractive error.[31,32] Contact lens wearing experiences during this time frame are unpredictable and vary with the patient and the pregnancy.[33] Proactive advice on contact lens management and anterior segment conditions can help patients plan accordingly. This might be an ideal time to update spectacles for part-time wear and maximize convenience with daily disposables.

Presbyopia and beyond. While refractive management may be more complex in patients with presbyopia, practitioners have a plethora of products to choose from, increasing the chance of meeting the patient's vision needs. This population may benefit from increased emphasis on dry eye management, potentially with less concern about atopic disease. Furthermore, additional care may be required with more mature patients who may present with other ocular and systemic conditions.

Post-surgery. Most patients choose refractive surgical options to decrease their dependence on vision correction. However, contact lenses may be necessary post-surgery to fine-tune vision (astigmatism, presbyopia) or manage unexpected outcomes (anisometropia, corneal ectasia). As LASIK is an established risk factor for dry eye, practitioners may devote additional attention to signs and symptoms of dry eye in this population.[8]

Although dry eye and allergy often present additional deterrents to contact lens wear, they shouldn't prevent most patients from successfully wearing contact lenses. The first step to ensuring success is properly educating patients on the particulars of lens wear when experiencing dry eye, allergy or both—including lens replacement and care products. Clinicians can further tailor dry eye disease and allergic conjunctivitis management to individual patients of all ages and life phases to promote healthy and comfortable lens wear. Don't let allergy or dry eye prevent lifelong contact lens success. ■

Dr. Wagner is a professor of clinical optometry and director of extern programs at Ohio State University. She is a diplomate in the Cornea, Contact Lens and Refractive Technologies Section of the American Academy of Optometry and a distinguished practitioner and fellow in the National Academies of Practice in Optometry.

1. Rosario N, Bielory L. Epidemiology of allergic conjunctivitis. Curr Opin Allergy Clin Immunol. 2011;11(5):471-6.
2. Pawankar R, Canonica GW, Holgate ST, et al. The World Allergy Orgnaization White Book on Allergy. Milwaukee, Wis.: The World Allergy Organization; 2013.
3. Leonardi A, Castegnaro A, Valerio AL, Lazzarini D. Epidemiology of allergic conjunctivitis: clinical appearance and treatment patterns in a population-based study. Curr Opin Allergy Clin Immunol. 2015;15(5):482-8.
4. Bielory L, Friedlaender MH. Allergic conjunctivitis. Immunol Allergy Clin North Am. 2008;28(1):43-58.
5. Ackerman S, Smith LM, Gomes PJ. Ocular itch associated with allergic conjunctivitis: latest evidence and clinical management. The Adv Chronic Dis. 2016;7(1):52-67.
6. Wong AH, Barg SS, Leung AK. Seasonal and perennial allergic conjunctivitis. Recent Pat Inflamm Allergy Drug Discov. 2014;8(2):139-53.
7. Borazan M, Karalezli A, Akova YA, et al. Efficacy of olopatadine HCl 0.1%, ketotifen fumarate 0.025%, epinastine HCl 0.05%, emedastine 0.05% and fluorometholone acetate 0.1% ophthalmic solutions for seasonal allergic conjunctivitis: a placebo-controlled environmental trial. Acta Ophthalmol. 2009;87(5):549-54.
8. Smith JA, Albeitz J, Begley C, et al. The epidemiology of dry eye disease: report of the epidemiology subcommittee of the international dry eye workshop. Ocul Surf. 2007;5(2):93-107.
9. Lemp MA. Report of the National Eye Institute/Industry workshop on clinical trials in dry eye. CLAO J. 1995;21(4):221-32.
10. Dumbleton K, Woods CA, Jones LW, Fonn D. The impact of contemporary contact lenses on contact lens discontinuation. 2013;39(1):92-8.
11. Richdale K, Sinnot LT, Skadahl E, Nichols JJ. Frequency of and factors associated with contact lens dissatisfaction and discontinuation. Cornea. 2007;26(2):168-74.
12. Rueff EM, Bailey MD. Presbyopic and non-presbyopic contact lens opinions and vision correction preferences. Cont Lens Anterior Eye. March 23, 2017. [Epub ahead of print].
13. Dumbleton K, Caffery B, Dogru M, et al. The TFOS International Workshop on Contact Lens Discomfort: report of the subcommittee on epidemiology. Invest Ophthalmol Vis Sci. 2013;18(11):TFOS20-36.
14. Chalmers R, Begley C. Dryness symptoms among an unselected clinical population with and without contact lens wear. Cont Lens Anterior Eye 2006;29(1):25-30.
15. du Toit R, Situ P, Simpson T, Fonn D. The effects of six months of contact lens wear on the tear film, ocular surfaces, and symptoms of presbyopes. Optom Vis Sci. 2001;78(6):455-62.
16. Nichols JJ, Sinnot LT. Tear film, contact lens, and patient-related factors associated with contact lens-related dry eye. Invest Ophthalmol Vis Sci. 2006;47(4):1319-28.
17. Rueff EM, Varghese RJ, Brack TM, et al. A survey of presbyopic contact lens wearers in a university setting. Optom Vis Sci. 2016;93(8):848-54.
18. Berger WE, Granet DB, Kabat AG. Diagnosis and management of allergic conjunctivitis in pediatric patients. Allergy Asthma Proc. 2017;38(1):16-27.
19. Hom MM, Nguyen AL, Bielory L. Allergic conjunctivitis and dry eye syndrome. Ann Allergy Asthma Immunol. 2012;108(3):163-6.
20. Stapleton F, Tan J. Impact of contact lens material, design, and fitting on discomfort. Eye Contact Lens. 2017;43(1):32-9.
21. Carnt NA, Evans VE, Naduvilath TJ, et al. Contact lens–related adverse events and the silicone hydrogel lenses and daily wear care system used. Arch Ophthalmol. 2009;127(12):1616-23.
22. Sawyer SM, Afifi RA, Bearinger LH, et al. Adolescence: a foundation for future health. Lancet. 2012;379(9826):1630-40.
23. Gifford P, Gifford KL. The future of myopia control contact lenses. Optom Vis Sci. 2016;93(4):336-43.
24. Vitale S, Sperduto RD, Ferris FL 3rd. Increased prevalence of myopia in the United States between 1971-1972 and 1999-2004. Arch Ophthalmol. 2009;127(12):1632-9.
25. Walline JJ, Lindsley K, Vedula SS, et al. Interventions to slow progression of myopia in children. Cochrane Database Syst Rev. 2011:CD004916.
26. Chalmers R, Wagner H, Mitchell GL, et al. Age and other risk factors for corneal infiltrative and inflammatory events in young soft contact lens wearers from the Contact Lens Assessment in Youth (CLAY) study. Invest Ophthalmol Vis Sci. 2011;52(9):6690-6.
27. Stapleton F, Keay L, Jalbert I, Cole N. The epidemiology of contact lens related infiltrates. Optom Vis Sci. 2007;84(4):257-72.
28. Schnider CM, Jedraszczak AM. The "pantry load" effect—can it help drive more compliant contact lens replacement? Optom Vis Sci. 2012;89:E-abstract 120652.
29. Wagner H, Richdale K, Mitchell GL, et al. Age, behavior, environment, and health factors in the soft contact lens risk survey. Optom Vis Sci. 2014;91(3):252-61.
30. The Vision Council. Hindsight is 20/20/20: protect your eyes from digital devices. Digtal eyestrain report. 2015. www.thevisioncouncil.org/sites/default/files/VC_DigitalEyeStrain_Report2015.pdf. Accessed May 4, 2017.
31. Park SB, Lindahl KJ, Temnycky GO, Aquavella JV. Effect of pregnancy on corneal curvature. CLAO J. 1992;18(4):256-9.
32. Imafidon CO, Imafidon JO. Contact lenses in pregnancy. Br J Obstet Gynaecol. 1992;99(11):865-8.
33. Sharma S, Wuntakal R, Anand A, et al. Pregnancy and the eye. Obstetrician and Gynaecologist. 2006:141-6.

SMART SYSTEM® 2 20/20 USA

NEW!

The New All-in-One System delivers Simplicity and Power. Its sleek, compact appearance will streamline the look of your innovative office. The **Smart System® 2** includes the same revolutionary features of the current system plus its overall lighter weight and faster processor gives you improved performance.

20 Different Vision Tests, including VA, contrast, stereopsis, binocular balance and color

Near, Intermediate and Distance Testing

DVD Functionality allows the user to play DVDs directly providing immediate fixation on retinoscopies

Patient Education includes 26 preloaded patient education images with the flexibility for additional images and videos

Internal Marketing Load PowerPoint slides, images or videos to promote your practice and educate your patients

LEA SYMBOLS® & LEA NUMBERS® now standard in Pediatrics, user selective

Give us a call 1/847-763-0500

Learn more about the Visual Acuity Testing System which includes the best local customer service and technical support in the industry!

M&S® TECHNOLOGIES

The First Choice in Vision Testing Systems

mstech-eyes.com

CE

M&S holds US Patents 7,354,155; 7,926,948; 8,425,040; 8,167,429; 8,419,184 & 8,550,631. Other Patents Pending.

Made in the USA

8th Annual Retina Report

Diabetes Care in the Age of Anti-VEGF

These versatile drugs are rewriting the rulebook. Here's what an OD should know.

By Richard Zimbalist, OD, and Amber Scharnweber, OD

We all encounter patients with diabetes in our practice, so it's incumbent upon us to keep up to date on the extent of its impact and the options available to help, especially in an environment where the research and clinical protocols are rapidly evolving. Retina specialists are busier than ever and expect the referring OD to play a vital role in early identification, patient education and ongoing—possibly lifelong—follow-up. So, let's make sure we're on the same page as our syringe-wielding colleagues to ensure patients get the best care possible.

Diabetic eye disease is the leading cause of blindness in American adults, affecting approximately 7.7 million individuals, and the number of people with visual impairment due to diabetic eye disease worldwide is rising—from 2000 to 2010, the number of diabetic retinopathy (DR) cases increased 89%.[1,2] Looking forward, the number of patients with DR is expected to double between the years 2010 and 2050.[1]

Diabetic macular edema (DME) is the leading cause of vision loss and affected approximately 750,000 people in the United States in 2010.[3] From 2005 to 2008, an estimated 4.4% of diabetes sufferers had vision-threatening diabetic retinopathy.[4] The burden of diabetes-related blindness is not only devastating for the person afflicted, it also costs the United States approximately $500 million annually.[5]

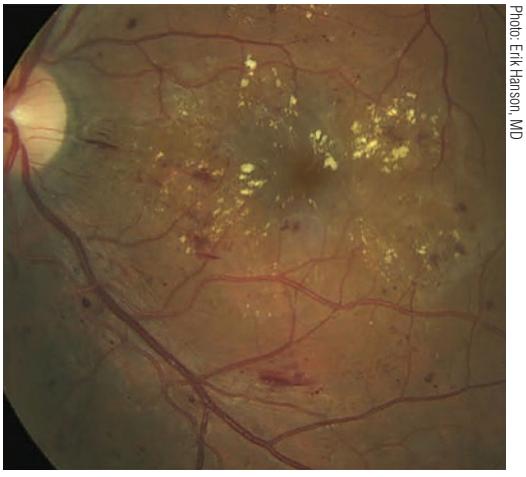


Photo: Erik Hanson, MD

Scattered microaneurysms, intraretinal hemorrhages and nerve fiber layer hemorrhages, along with prominent exudate with clinically significant macular edema. The retinal architecture is partially obscured, indicative of macular thickening.

Drug options

Vascular endothelial growth factor (VEGF) plays a critical role in the pathogenesis of DME and DR. Currently, three primary anti-VEGF molecules are widely used to treat ocular angiogenesis: Avastin (bevacizumab, Genentech), Lucentis (ranibizumab, Genentech) and Eylea (aflibercept, Regeneron). Each debuted in eye care as age-related macular degeneration (AMD) treatments and have since been studied in vein occlusion and DR.

Avastin. This was the first anti-VEGF antibody designed to block all isoforms of VEGF.[6] It was approved in 2004 for use in colorectal cancer and subsequently for several other types; it is currently used off-label in the eye.

Lucentis. Genetech believed Avastin would not diffuse through the retina efficiently enough to reach the choroid, so it developed an alternative, shortened molecule, Lucentis.[6]

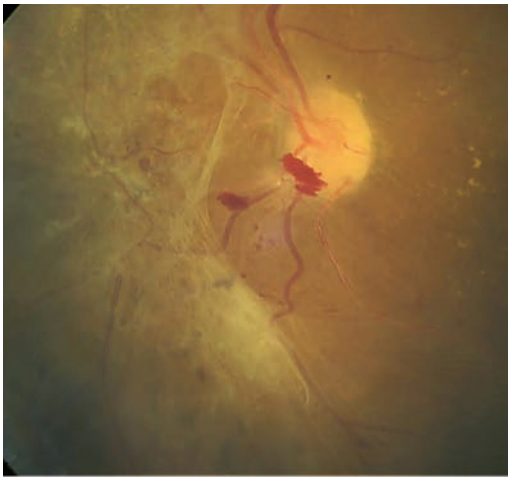


Photo: Erik Hanson, MD

This patient shows a large area of preretinal fibrosis and a small amount of preretinal hemorrhage. The macula demonstrates exudate with macular edema.

Eylea. This recombinant fusion protein functions as a decoy receptor for VEGF-A, developed with improved pharmacokinetics, stronger binding affinity, and a longer half-life than Lucentis and Avastin.[6,7]

The value of these agents in treating ocular disease continues to be realized and has led to FDA approvals for the treatment of DME and DR in patients with DME and macular edema associated with retinal vein occlusion *(Table 1)*.

The New Gold Rush

Recent years have seen a rapid reappraisal of the treatment protocols for diabetic eye disease, largely due to the success of anti-VEGF options. Long-cherished gold standards of care are being reconsidered, relegated and, in some cases, abandoned.

Diabetic macular edema. Focal laser photocoagulation has long been the standard of care for diabetic macular edema. The Early Treatment Diabetic Retinopathy Study (ETDRS) demonstrates the efficacy of focal macular laser treatment in reducing the risk of moderate vision loss by up to 50% in eyes with clinically significant macular edema.[8] However, this classic treatment regimen has now been demoted with the advent of intravitreal anti-VEGF agents. Several recent clinical trials show anti-VEGF therapy (AVT) achieves better visual outcomes vs. focal grid laser for DME.[9-14] Though once considered off-label, the FDA recently approved Lucentis and Eylea for DME *(Table 1)*.

While evaluating anti-VEGF agents for their efficacy and safety in DME, the RISE and VISTA studies also noted that diabetic retinopathy has a tendency to improve in patients undergoing AVT.[11-14] A second group of researchers demonstrated comparable results, with 39% of eyes improving equal to or greater than two steps on the diabetic retinopathy severity grading system (DRSS) through 36 months in Lucentis-treated eyes.[15] In a third instance, similar findings were seen by the Diabetic Retinopathy Clinical Research Network (DRCR), with almost half of the patients in the Lucentis group demonstrating an improvement in DR greater than two steps based on the DRSS.[16]

Diabetic retinopathy. The gold standard treatment of proliferative DR (PDR) has long been panretinal photocoagulation (PRP), since its evaluation in EDTRS.[17] Although there is no denying the efficacy of PRP, it can leave the patient with nyctalopia, reduced contrast sensitivity, visual field constriction and optic atrophy.[18] Several studies show Lucentis and Avastin improve PDR without permanent side effects.[11-14]

In April 2017, Lucentis gained FDA approval for treatment of all forms of DR, including eyes without DME. The approval stems from

DRCR's Protocol S in which Lucentis was compared to PRP for the treatment of PDR. The new Lucentis indication may prove useful for the prevention of visually disabling retinopathy, rather than simply the treatment of it.

Can AVT effectively treat PDR as well as PRP? A recent study sought to investigate this concept. The randomized trial evaluated the superiority of Lucentis vs. PRP for PDR and found no difference in active or regressed neovascularization through two years of follow-up.[16]

With the FDA approval of Lucentis and Eylea for DR in 2015, the standard of care for this disease is continually evolving. A simple web search on www.clinicaltrials.gov shows an abundance of trials involving AVT, which suggests the progressive nature of research in retinal treatment modalities.[19] Many retina specialists are transitioning towards intravitreal injection as the primary treatment modality over laser for a multitude of conditions, according to a worldwide annual survey conducted by the American Society of Retina Specialists (ASRS).[20] It found that 86.6% of respondents in the United States would choose either Avastin (62.2%) or Eylea (24.4%) as the treatment of choice in patients with decreased vision due to DME.[20] Respondents from all other continents also show a clear preference for AVT in DME, with 60% of respondents choosing AVT as their first-line treatment.[20] In addition, the survey found many retina specialists would consider using Lucentis over PRP as the primary treatment for PDR, consistent with results from DRCR's Protocol S study.[16,20] The principal exception: when patient follow-up is unreliable or uncertain.[20] Interestingly, only doctors in the United States felt the Lucentis's cost wasn't a pertinent factor in determining therapy choice.[20]

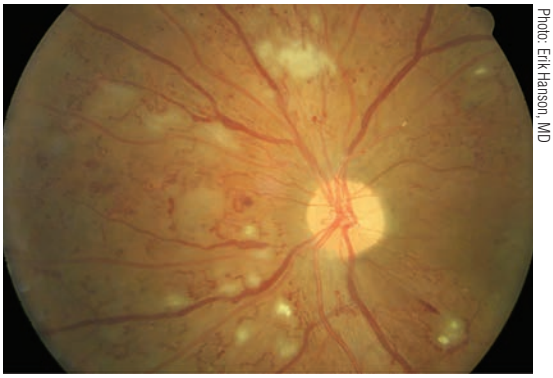
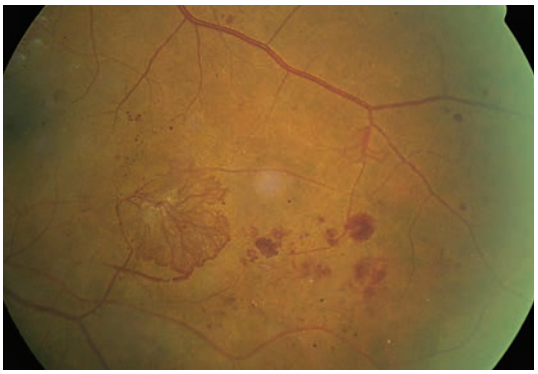


Photo: Erik Hanson, MD

Non-high risk PDR is evidenced by a small amount of neovascularization on the optic nerve. Large amounts of retinal hemorrhages, cotton wool spots and intraretinal microvascular abnormalities (IRMA) are present.



A large area of retinal neovascularization, also known as neovascular elsewhere (NVE), exists. The superotemporal vein demonstrates an omega loop, a strong indicator of retinal hypoxia.

Compare the Costs

A study by the DRCR (Protocol T) comparing Avastin, Lucentis and Eylea for DME showed similar visual acuity outcomes at one year for eyes with baseline acuity of 20/40 or better, but among eyes with baseline VA worse than 20/40, Eylea had a superior (clinically meaningful) visual acuity outcome.[22] Through two years of follow up Eylea remained superior to Avastin, however, there was no longer a significant visual difference when compared with Lucentis.[23] Additionally, the two-year results found a minimal difference in the number of injections needed in patients treated with Avastin, Lucentis and Eylea through two years of follow up for DME.[22,23]

The time has come to talk about the elephant in the room—money. A large price difference exists between the three commonly used AVTs. The 2015 wholesale acquisition costs for single doses of each drug were $60, $1,170 and $1,850 for Avastin, Lucentis and Eylea, respectively.[21] As AVTs are often injected multiple

Table 1. FDA Approval Date and Indication for Lucentis and Eylea[30, 31]

Drug	Date	Approval
Lucentis	2006	Neovascular macular degeneration
	2010	Macular edema following retinal vascular occlusion
	2012	Diabetic macular edema
	2015	Diabetic retinopathy in patients with DME
	2017	Myopic choroidal neovascularization
	2017	All forms of diabetic retinopathy
Eylea	2011	Neovascular macular degeneration
	2014	Diabetic macular edema
	2014	Macular edema following retinal vascular occlusion
	2015	Diabetic retinopathy with macular edema

times per year, this can result in a significant cost to both the patient and health care system.

Using the above wholesale cost numbers, patient expense in this study would have registered at $960, $17,550 and $27,750 for treatment with Avastin, Lucentis, and Eylea, respectively. Likewise, a post hoc analysis of the DRCR Comparative Effectiveness Trial indicated Eylea and Lucentis are not cost-efficient treatment options relative to Avastin for the treatment of DME.[21]

Consider the Risks

No doubt exists that anti-VEGF therapy is great for treating various posterior segment conditions. However, important drawbacks to the modality exist.

Complications. All of the trials required an intravitreal injection every four to eight weeks (depending on whether Avastin, Lucentis or Eylea was used). Patients treated with AVT for DME or other retinal conditions will often require multiple injections over several months or years. Secondly, AVT is not altogether benign.[24] Although serious complications such as endophthalmitis are rare (at approximately 0.028%), they can be visually disabling when they occur.[24] Investigators report that ocular side effects of AVTs may include: retinal tears, retinal detachment, vitreous hemorrhage, traumatic iatrogenic cataract, ocular hypertension, ocular hemorrhage and infectious uveitis.[24-27]

Reported adverse reactions to AVT affect many of the body's organ systems, including the gastrointestinal, cardiovascular, endocrine, immune and musculoskeletal and excretory systems.[23] Of particular interest, a post hoc analysis of the DRCR's Protocol T study found Lucentis is associated with a higher rate of APTC (Anti-Platelet Trialists Collaboration) events than Eylea and Avastin, including nonfatal strokes and vascular deaths. However, this trend has not been observed in other large randomized control trials including RISE and RIDE.[23]

While the AVT complication rate is quite low, the vision-threatening complications cannot be dismissed.

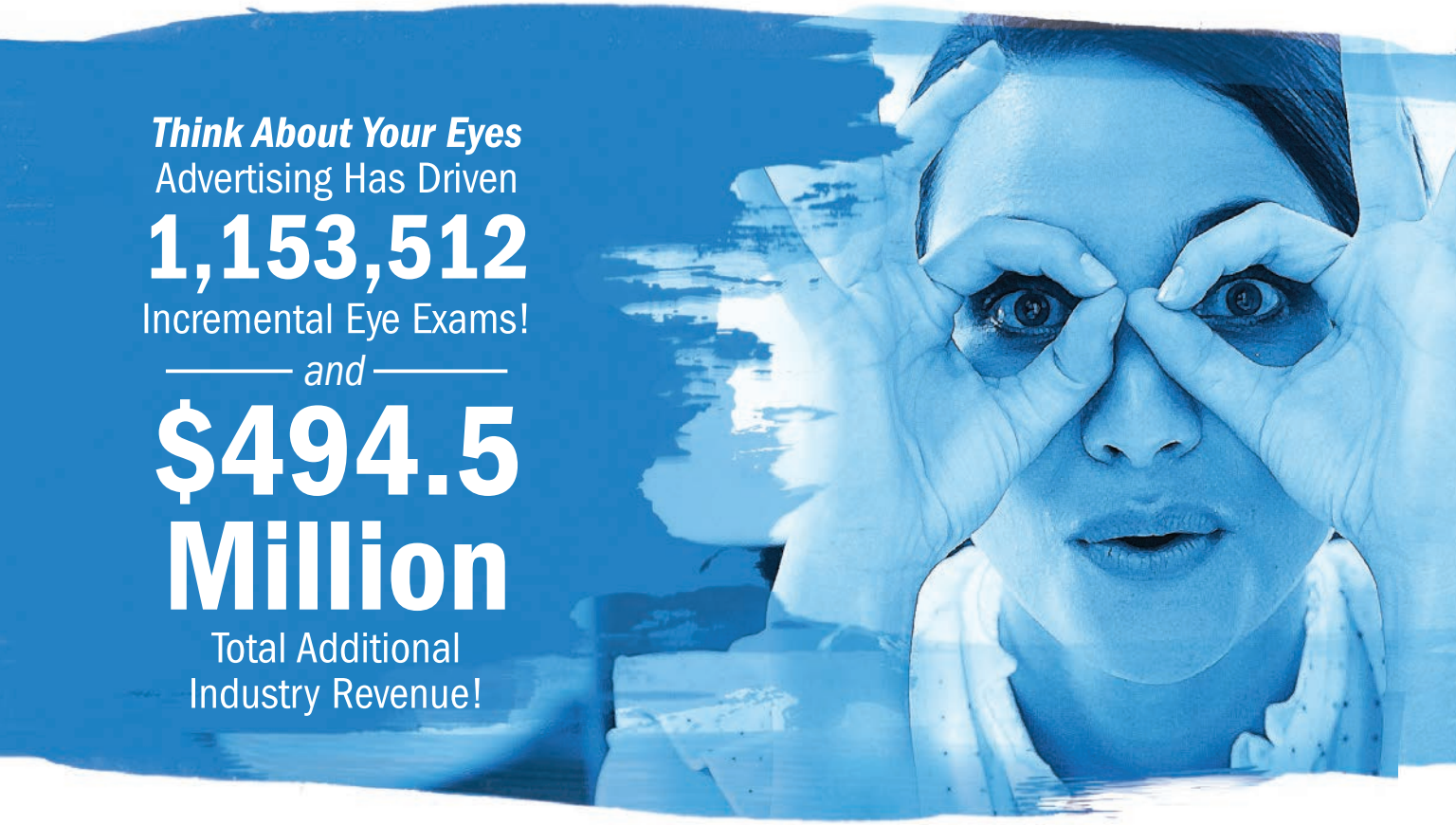
Half life. One of the primary detractors from widespread adoption of AVT is the limited half-life. Eylea aimed to change that with improved pharmacokinetics relative to the other two anti-VEGF drugs; however, it still requires dosing every four weeks for the first three months, followed by injections every eight weeks, if needed. Genentech is currently in the process of recruiting patients for a Phase II study that uses a port delivery system (PDS) to deliver Lucentis; the new delivery system aims to allow an ophthalmologist to refill an intraocular device while delivering the drug into the vitreous over an extended time period.[28] The study will compare the efficacy of the PDS vs. traditional Lucentis intravitreal injections for patients with AMD-related subfoveal choroidal neovascularization.[28] This novel therapeutic approach has the potential to improve the delivery of care not only for AMD, but myriad other retinal conditions, including diabetic eye disease.

We are left with several questions:

- Should diabetic retinopathy be treated earlier with AVT?
- Can we prevent the development of PDR with AVT?
- Is there still a place for laser?

As the face of ocular therapeutics continues to advance, we can expect pharmacologic agents to lead the way in answering these questions and likely change longstanding laser therapy practice patterns.

Strength in Numbers



Think About Your Eyes
Advertising Has Driven

1,153,512

Incremental Eye Exams!

and

$494.5 Million

Total Additional
Industry Revenue!

These incredible numbers tell the story!

Think About Your Eyes is motivating current patients to get eye exams more regularly, and driving new patients to get their first annual eye exam. This allows you to detect vision issues more quickly and prescribe proper treatment.

thinkaboutyoureyes.com®

There's Never Been a Better Time to Support Think About Your Eyes!

Please contact Jon Torrey for more information at **jtorrey@thinkaboutyoureyes.com**

Sit in Your Own Chair

Take a step back and consider your patient's perspective. You have just been diagnosed with DME and PDR. Would you rather receive a laser treatment that will leave you with permanent side effects or get repeated injections in the eye? Patients should be educated about the available treatment options and the benefits and risks of each. Is the patient reliable for follow up? Does the lack of transportation to appointments pose a concern? Is cost a factor for the patient? These discussions need to begin in the optometrist's chair. As the delivery methods change and scope of practice advances, we may be deciding the best AVT for our patients. ■

Dr. Zimbalist practices at Harry S. Truman Memorial Veterans Hospital in Columbia, MO, and is an American Academy of Optometry fellow.

Dr. Scharnweber practices at the St. Cloud VA Health Care System in St. Cloud, MN and is an adjunct faculty at the Pennsylvania College of Optometry.

1. National Eye Institute. Diabetic retinopathy. Available at: nei.nih.gov/eyedata/diabetic. Accessed January 28, 2017.
2. Leasher JL, Bourne RR, Flaxman SR, et al. Global estimates on the number of people blind or visually impaired by diabetic retinopathy: a meta-analysis from 1990 to 2010. Diabetes Care. 2016; Sep;39(9):1643-9.
3. Varma R, Bressler NM, Doan QV, et al. Prevalence of and risk factors for diabetic macular edema in the United States. JAMA Ophthalmology. 2014;132(11):1334-40.
4. Zhang X, Saaddine JB, Chou CF, et al. Prevalence of diabetic retinopathy in the United States, 2005-2008. JAMA. 2010;304(6):649-56.
5. Javitt JC, Aiello LP, Chiang Y, et al. Preventative eye care in people with diabetes is cost-saving to the federal government: implications for health-care reform. Diabetes Care. 1994 Aug;17(8):909-17.
6. Kim LA, D'Amore PA. A brief history of anti-VEGF for the treatment of ocular angiogenesis. Amer J Pathol. 2012 Aug;181(2):376-9.
7. Aflibercept (ophthalmic): Drug information. UpToDate. www.uptodate.com/contents/aflibercept-ophthalmic-drug-information?source=search_result&search=aflibercept&selectedTitle=1~30. Accessed February 14, 2017.
8. Photocoagulation for diabetic macular edema. Early treatment diabetic retinopathy study report number 1. Early treatment diabetic retinopathy study research group. Arch Opthalmology. 1985 Dec;103(12):1796-806.
9. Elman MJ, Qin H, Aiello LP, et al. Intravitreal ranibizumab for diabetic macular edema with prompt versus deferred laser treatment: three year randomized trial results. Ophthalmology. 2012;119(11):2312-18.
10. Elman MJ, Ayala A, Bressler NM, et al. Intravitreal ranibizumab for diabetic macular edema with prompt versus deferred laser treatment: 5-year randomized trial results. Ophthalmology. 2015;122(2):375-81.
11. Boyer DS, Nguyn QD, Brown DM, et al. Outcomes with as-needed ranibizumab after initial monthly therapy. Ophthalmology. 2015;122(12):2504-13.
12. Nguyen QD, Brown DM, Marcus DM, et al. Ranibizumab for diabetic macular edema: results from 2 phase III randomized trials: RISE and RIDE. Ophthalmology. 2012;119(4):789-801.
13. Heier JS, Korobelnik JF, Brown DM, et al. Intravitreal aflibercept for diabetic macular edema: 148-week results from the VISTA and VIVID studies. Ophthalmology. 2016 Nov;123(11):2376-85.
14. Boyer DS, Nguyen QD, Brown DM, et al. RIDE and RISE Research Group. Outcomes with as-needed ranibizumab after initial monthly therapy: long-term outcomes of the Phase III RIDE and RISE trials. Ophthalmology. 2015 Dec;122(12):2504-13.
15. Ip MS, Domalpally A, Sun JK, Ehrlich JS. Long-term effects of therapy with ranibizumab on diabetic retinopathy severity and baseline risk factors for worsening retinopathy. Ophthalmology. 2015 Feb;122(2):367-74.
16. Writing Committee for the Diabetic Retinopathy Clinical Research Network. Panretinal photocoagulation vs intravitreous ranibizumab for proliferative diabetic retinopathy: A randomized clinical trial. JAMA. 2015;314(20):2137-46.
17. Early Treatment Diabetic Retinopathy Study Research Group. Early photocoagulation for diabetic retinopathy. ETDRS report number 9. Ophthalmology. 1991 May;98(5 Suppl):766-85.
18. Fong DS, Girach A, Boney A. Visual side effects of successful scatter laser photocoagulation surgery for proliferative diabetic retinopathy: a literature review. Retina. 2007 Sep;27(7):816-24.
19. Clinical Trials.gov: A service of the U.S. National Institutes of Health. clinicaltrials.gov/ct2/results?term=retina&Search=Search. Accessed February 21, 2017.
20. Rezaei KA, Stone TW, eds. Global trends in retina survey. American Society of Retina Specialists. 2016. Chicago, IL. www.asrs.org/content/documents/2016_global_trends_in_retina_survey_highlights_for_website_2.pdf. Accessed February 1, 2017.
21. Ross EL, Hutton DW, Stein JD, et al. Cost-effectiveness of aflibercept, bevacizumab, and ranibizumab for diabetic macular edema treatment. JAMA Ophthalmology. 2016;134(8):888-96.
22. Wells, JA, Glassman AR, Ayala AR, et al. Aflibercept, bevacizumab, or ranibizumab for diabetic macular edema. N Engl J Med. 2015;372(13):1193-203.
23. Wells JA, Glassman AR, Ayala AR, et al. Aflibercept, bevacizumab, or ranibizumab for diabetic macular edema: Two-year results from a comparative effectiveness randomized clinical trial. Ophthalmology. 2016;123(6):1351-9.
24. Merani R, Hunyor AP. Endophthalmitis following intravitreal anti-vascular endothelial growth factor (VEGF) injection: a comprehensive review. Inter J Retina Vitreous. 2015; 21:1-9.
25. Moshfeghi AA. Safety of Intravitreal Anti-VEGF Agents. Review of Ophthalmology. 2014 Nov; 20(11):52-6.
26. Falavarjani KG, Nguyen QD. Adverse events and complications associated with intravitreal injection of anti-VEGF agents: a review of literature. Eye. 2013;27(7):787–94.
27. Nuzzi R, Tridico F. Local and systemic complications after intravitreal administration of anti-vascular endothelial growth factor agents in the treatment of different ocular diseases: a five-year retrospective study. Semin Ophthalmol. 2015 Mar;30(2):129-35.
28. Study of the efficacy and safety of the ranibizumab port delivery system for sustained delivery of ranibizumab in participants with subfoveal neovascular age-related macular degeneration (LADDER). Clinical Trials.gov: A service of the U.S. National Institutes of Health. Clinicaltrials.gov/ct2/show/NCT02510794. Updated October 3, 2016. Accessed February 22, 2017.
29. Lucentis approval history. Drugs.com. www.drugs.com/history/lucentis.html. Accessed February 22, 2017.
30. Eyelea approval history. Drugs.com. www.drugs.com/history/eylea.html. Accessed February 22, 2017.

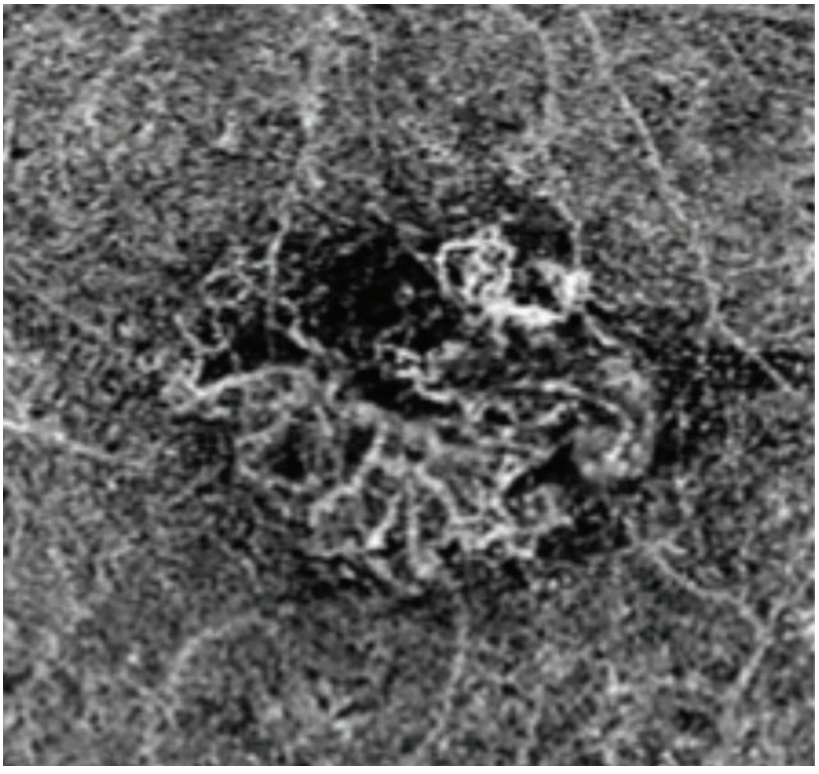
8th Annual Retina Report

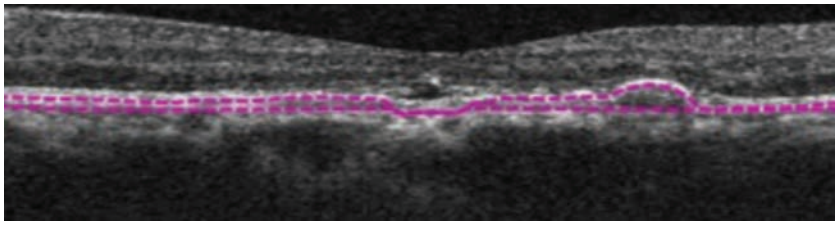
Advanced Imaging Techniques for Choroidal Disease

With new technology, optometrists can better target this previously stealthy structure.

By Rim Makhlouf, OD, Diana Shechtman, OD, and Sherrol Reynolds, OD

The choroid is the main source of blood to the eye and, as such, understanding its health can play a vital role in identifying and managing various ocular diseases. Traditionally, however, visualizing the choroid has been a near-impossible challenge, due to its positioning between the retinal pigment epithelium (RPE) and the sclera. This difficulty extends to the structures within the choroid, such as the choriocapillaris, a rich vascular supply that lies at the inner side of the choroid in a single plane beneath the retina. The outer layers of the choroid mainly consist of intermediate-sized vessels adjacent to the capillaries (Sattler's layer), followed by larger vessels (Haller's layer). The choriocapillaris is the main layer that provides nutrients to the outer retina, including the photoreceptor and RPE cells. Its integrity is crucial for visual function.





Above, this segmentation image of the choroid, obtained via OCT-A, demonstrates the hyper-flow "sea fan" pattern At left, this structural OCT image of the same eye shows RPE elevations and mild foveal subretinal fluid.

Luckily, these previously hidden structures are no longer out of optometrists' reach thanks to the latest imaging modalities. This article explains how these technologies can be used to better evaluate the choroid.

Yesterday's Limitations

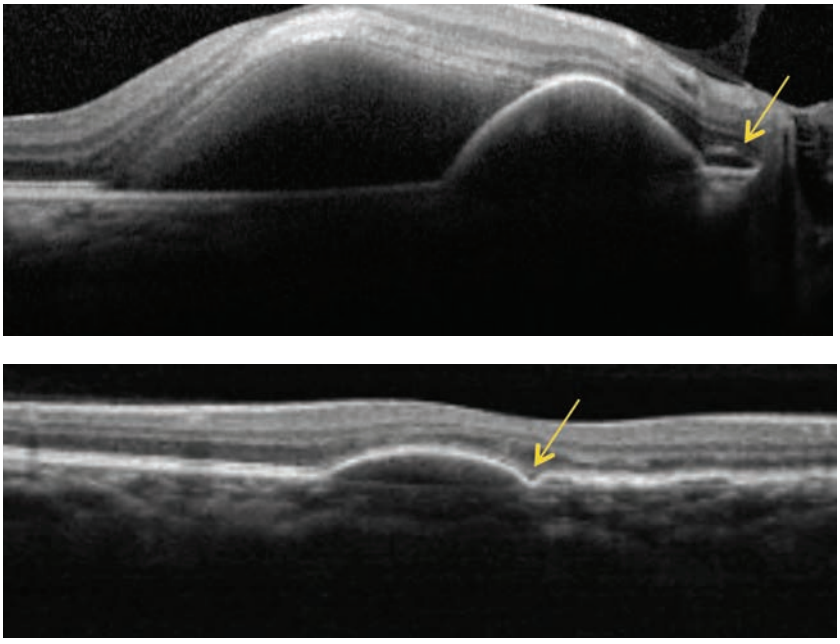
Indocyanine green angiography (ICGA) has traditionally been used to study choroidal circulation, but it does not allow three-dimensional visualization of the individual layers.[1,2] Conventional spectral-domain OCT (SD-OCT), while useful to visualize retinal layers with high resolution, is somewhat weaker in assessing the choroid, due to light scattering by the RPE layer, which results in decreased signal penetration through the retina into the choroid. This decreases the resolution at the choroid and visibility of the choroid-sclera interface. Although B-scan ultrasonography is helpful for visualizing some tumors and other features, it does not provide sufficient resolution to assess changes in choroidal thickness.

Today's Improvements

Advanced imaging modalities, such as enhanced-depth imaging optical coherence tomography (EDI-OCT), allow for the visualization of deep structures, including the choroid and the choroidal-sclera interface, while still retaining retinal details. EDI uses SD-OCT with the peak sensitivity placed posteriorly, towards the sclera.[3] It can penetrate up to 800µm and significantly improves the visualization of the choriocapillaris and choroidal vasculature, due to the proximity of these layers to the RPE.[3] Thanks to EDI-OCT's increased sensitivity in detecting the proper choroidal-sclera interface, we finally have a method by which we can measure choroidal thickness. Using this technology, researchers have found that the mean subfoveal choroidal thickness is approximately 287µm (plus or minus a standard deviation of 75.7µm).[4]

Knowing this has helped not only in the assessment of choroidal conditions, but also in the evaluation of the relationship between choroidal thickness and other ocular conditions. For example, we now know choroidal thickness typically decreases by approximately 16µm per decade of life.[4]

Choroidal thickness is now implicated in numerous retinal diseases, including age-related macular degeneration (AMD), adult-onset foveomacular dystrophy (AOFMD), central serous choroidopathy (CSC), diabetic retinopathy and retinitis pigmentosa, as well as in glaucoma. Choroidal thickness can also be affected by other factors, including diurnal variations, caffeine



Above, this SD-OCT shows a PCV patient. Note the dome-shaped elevation of RPE with an adjacent double layer sign (see arrow) and a serous retinal detachment. Below, this SD-OCT shows a different patient with PCV demonstrating a PED with a bola sign (see arrow).

consumption and water intake.[5] These changes are less prominent and usually don't exceed 30µm.[6]

The ability to see further into the choroidal structures using EDI-OCT has become increasingly helpful in assessing conditions such as choroidal nevi, choroidal melanomas and choroidal neovascularization. However, even when using EDI-OCT, direct visualization of choroidal vessels and assessment of the choroidal vascular density remain a challenge.

The newest technology, OCT-angiography (OCT-A), provides a detailed vascular map of the posterior segment. OCT-A uses motion contrast to construct detailed volumetric angiographic images, mapping the retinal and choroidal vasculature in a matter of seconds. To assess blood flow (erythrocytes), it compares decorrelation signal (differences in the backscattered OCT intensity) between sequential OCT B-scans, taken at the same cross-section.[3] It is a noninvasive imaging technique that has many potential applications in retinal and choroidal vascular diseases as it provides structural vascular information as well as functional blood flow information without the use of intravenous dye.

Our understanding of the choroid has vastly increased with the introduction of these imaging modalities. They have allowed the identification of conditions such as age-related choroidal atrophy (ARCA), pachychoroid and better assessment of choroidal neovascular membranes.

Disease Types

Choroidal neovascular membranes (CNVMs) are characterized by the presence of abnormal blood vessels that originate from the choroid and extend either to the area between Bruch's membrane and the RPE (as seen in type I), or into the subretinal space (as seen in type II). Funduscopic signs associated with CNVM include hemorrhages, retinal thickening, exudation and fibrosis, which often lead to photoreceptor damage and vision loss.[7]

CNVMs on OCT will generally appear as an area of hyper-reflectivity either below or above the RPE, depending on the type.[7] Other OCT features associated with CNVMs, such as RPE elevation and accumulation of fluid at various levels of the retina, provide invaluable information in the assessment of the activity of the lesion and for its clinical management.

Not all membranes leak, nor does leakage always signify the presence of a concomitant CNVM. For example, a CNVM secondary to pathological myopia, angioid streaks, histoplasmosis, CSC or inflammatory disease tend to have minimal leakage on traditional fluorescein angiography (FA). Furthermore, early changes may also go undetected and, although research shows SD-OCT and EDI-OCT are beneficial in the detection of CNVM, various entities may have similar location and reflectivity, which may cause challenges in interpretation.[7] The inability of these techniques to accurately visualize the lesion or its perfusion results in decreased sensitivity and specificity in the diagnosis of a CNVM.

Traditional angiography, FA or indocyanine green angiography (ICGA), remain the standard method of evaluating choroidal conditions, particularly for the evaluation of CNVM; however, newer methods have some major improvements. OCT-A, for instance, is dyeless and provides high-resolution imaging of the choroidal vasculature, enabling it to delineate the complex vasculature associated with a CNVM. Studies show that the sensitivity of OCT-A

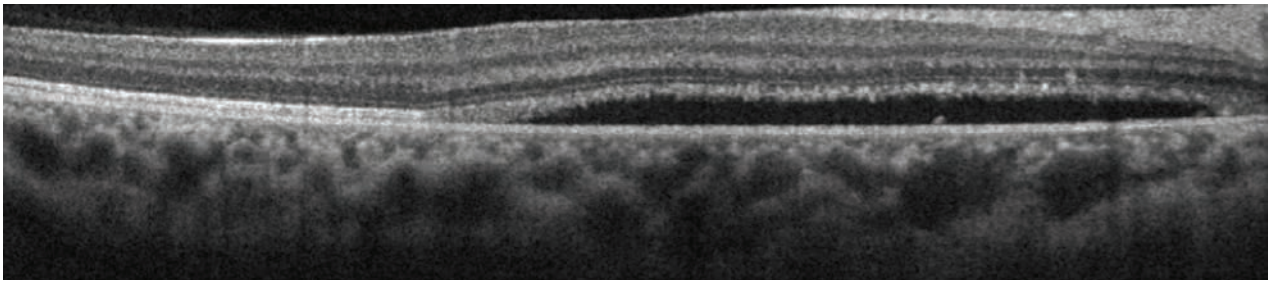
in detecting CNVMs when compared with FA ranges from 50% to 100%.[8-10] On the other hand, the sensitivity of CNVM diagnosis in diseases such as CSC and in cases of exudative AMD may be as high as 100% and 80%, respectfully.[11,12] The difference in sensitivity between conditions is probably due to the fact that an overlying massive hemorrhage is more likely to occur in AMD, as opposed to other conditions such as chronic CSC. The hemorrhage will block the signal and limit the visualization of CNVMs.[9,13]

OCT-A features associated with CNVM may include patterns described as small filamentous vessels forming anastomoses (lacy wheel or sea fan), as well as vessels associated with a central trunk (Medusa).[11,13] In contrast to FA, OCT-A allows a three-dimensional visualization of the fibrovascular network, by providing high-resolution depth-encoded images without any hindering effect associated with dye leakage. OCT-A is therefore invaluable in the qualitative assessment of CNVM, including its multiplanar location and morphology. This may aid in future assessment of morphological changes associated with treatment response.

Limitations of OCT-A include hindering effects from various artifacts, including projection artifacts which occur due to shadows created by moving erythrocytes in more superficial vessels, giving the false impression of blood flow in deeper layers. Another limitation is that OCT-A will detect a blood vessel only if its blood flow speed is higher than a minimum threshold, which is determined by the time between two sequential OCT B-scans.[14] CNVMs may show areas of reduced blood flow, potentially making them completely or partially undetectable on OCT-A.

The pachychoroid spectrum encompasses a multitude of macular disorders that share similar features, including increased choroidal thickness, dilation of the large choroidal vessels (Haller's layer) or "pachyvessels" and loss of choriocapillaris/Sattler's layer overlying the pachyvessels due to their compression by the latter.[15-17] The spectrum includes pachychoroid pigment epitheliopathy (PPE), CSC, pachychoroid neovasculopathy (PNV) and polypoidal choroidal vasculopathy (PCV). The classic and unifying feature for these conditions is the assessment of an increased choroidal thickness (pachychoroid), best evaluated through the use of EDI-OCT. This feature is helpful in the differential diagnosis of conditions within the pachychoroid spectrum from AMD.

Pachychoroid pigment epitheliopathy, researchers believe, is a precursor of CSC. It's characterized by



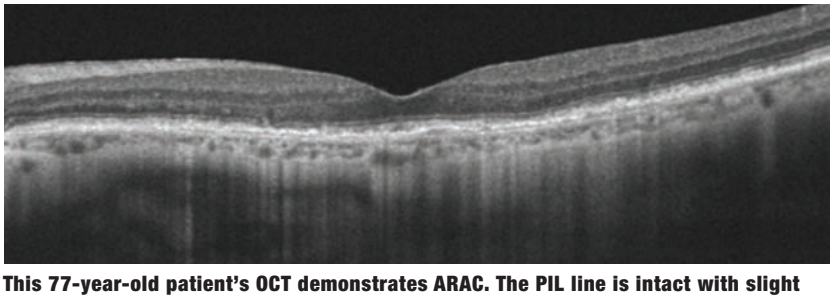
This SD-OCT shows a CSC patient demonstrating relatively thickened choroid (pachychoroid) with dilation of large choroidal vessels (pachyvessels) and relative absence of choriocapillaris and Sattler's layer in the area underlying the subretinal fluid.

the presence of RPE changes with absence of preceding or concurrent neurosensory detachment.[16,17] These RPE changes have similar appearance to findings often seen in the fellow eye of patients with unilateral CSC and may be mistaken for AMD changes. These include features associated with RPE hyperplasia and drusen-like deposits.[17] Classically, a thickened choroid is denoted on EDI-OCT, which is due to pachyvessels, with overlying attenuation of the choriocapillaris and Sattler's layer.[18] The pachyvessels, which run in close proximity to the RPE-Bruch membrane complex, incite pigment epitheliopathy changes.[18]

Central serous choroidopathy is characterized by an idiopathic serous neurosensory detachment often associated with serous pigment epithelial detachments (PEDs) and no evidence of inflammatory features or presence of a CNVM. SD-OCT shows a typical smooth and convex profile of neurosensory detachment with underlying hypo-reflectivity. EDI-OCT shows a thickened choroid and localizes the pachyvessels observed on ICGA to the outer choroid. This may help in the differential diagnosis with subretinal fluid associated with wet AMD. Chronic CSC (longer than three months) may result in RPE changes, thickened photoreceptor outer segments, ellipsoid zone disruption and, eventually, outer retinal atrophy, neovascularization formation (PNV), or both.[16]

Pachychoroid neovasculopathy is characterized by the presence of type I CNVM associated with a pachychoroid phenotype, such as a chronic CSC. The presence of shallow irregular RPE elevations on SD-OCT in eyes with pachychoroid should raise suspicion for neovascularization. PNV is distinguished from neovascular AMD by several features, including younger age at onset of neovascularization, a relative absence of drusen, and a thick choroid with pachyvessels as seen on EDI-OCT.[16] On the other hand, patients older than 60 years may exhibit some clinical features common to both PNV and neovascular AMD but with thinning of the choroid. Combining both EDI-OCT—to evaluate choroidal thickness—and OCT-A—to determine the presence of a CNVM—is critical in the diagnosis of PNV.

Polypoidal choroidal vasculopathy can be confirmed by the presence of polypoidal lesions with or without branching network on ICGA. Clinical findings include recurrent serosanguinous PEDs and may mimic those associated with wet AMD. Serous retinal detachments are often present. Two distinct signs are observed on OCT, the "double layer" sign described as two hyperreflective lines within the vicinity of the PED, representing the branching vascular network, and the "bolas sign" described as an RPE disruption, representing a small polyp adjacent to the PED.[19-21] PCV is highly suspect if funduscopic findings are noted in adults of Caribbean, Asian or African descent.[22-24] Unlike with wet AMD, EDI-OCT of a PCV will show a pachychoroid. Other distinguishing features on structural OCT include the increased height of the serous retinal detachment, which also occurs in higher frequency, and less intraretinal edema.[22-24] Although ICGA remains the standard to diagnose of PCV, OCT-A may help to identify the PCV complex. Using segmentation of the choriocapil-



This 77-year-old patient's OCT demonstrates ARAC. The PIL line is intact with slight RPE mottling.

laris, the branching vascular network will appear as a hyper flow lesion whereas the polypoidal lesion will demonstrate lower flow, and will appear either as a hyper flow round structure surrounded by a hypo-intense halo, or more frequently, as a hypo flow round structure. The lower flow of the polypoidal lesion is likely due to unusual blood flow within it in contrast with the branching vascular network.[25]

Age-related choroidal atrophy is a relatively newly described entity in patients older than 60 years with decreased visual acuity where the primary abnormality seems to be in the choroid.[26] It is characterized by markedly decreased choroidal thickness under the fovea and particularly nasal to the fovea, along with pigmentary changes and a rarefaction of visible choroidal vessels under the macula. In fact, since the choroid, and more specifically the choriocapillaris, plays a vital role by providing nutrients to photoreceptors and RPE cells, its decreased perfusion should lead to ischemic progressive photoreceptor and RPE cell death in the foveal area, eventually leading to decreased visual acuity. One study of 17 patients shows subfoveal choroidal thickness in ARCA is less than 125µm, with an average of 69.8µm.[26] However, this cut-off of 125µm of choroidal thickness does not represent an absolute demarcation.[26]

The main differential diagnosis with ARCA is AMD. Pathological features of AMD include drusen and pigmentary changes eventually progressing into geographic atrophy due to RPE and photoreceptor death. AMD itself has been linked with choroidal thinning and reduced vascular density, especially in the advanced stage of the disease. However, the differentiating characteristic of ARCA with AMD is that the main abnormality correlating with the decreased visual acuity seems to be severe choroidal thinning as opposed to the presence of geographic atrophy or changes characteristic of wet AMD.[26] In addition, the funduscopic presence of drusen are also not a common finding with ARCA but rather with AMD.[26] EDI-OCT is helpful in determining the thickness of the choroid, and therefore in the differential diagnosis.[26]

Our understanding of choroidal diseases has been augmented in recent years with the advent of advanced imaging techniques: EDI-OCT and OCT-A. These techniques allow for noninvasive, improved qualitative and quantitative analysis of the choroid and are invaluable in the diagnosis and management of a variety of chorioretinal diseases.

Up to 19 CE Credits*

NEW TECHNOLOGIES & TREATMENTS IN 2017 EYE CARE

REVIEW OF OPTOMETRY®
EDUCATIONAL MEETINGS OF CLINICAL EXCELLENCE

Philadelphia

November 3-5, 2017

Join *Review of Optometry*'s New Technologies & Treatments in Eye Care on November 3-5, 2017 in Philadelphia, PA. This meeting provides up to 19* COPE CE credits including interactive workshops!**

Photo: Loews Philadelphia Hotel

Loews Philadelphia Hotel
1200 Market Street,
Philadelphia, PA 19107
Phone: (215) 627-1200
Discounted room rate: $189/night†

EARLY BIRD SPECIAL
$75 OFF REGISTRATION
must register before Sept. 18, 2017

Program Chair:
Paul Karpecki, OD, FAAO

Registration Cost: $495
Receive $75 off if registered before September 18, 2017

Three Ways to Register

Online: www.reviewofoptometry.com/philadelphia2017
E-mail: reviewmeetings@jobson.com
Phone: 866-658-1772

Administered by *Review of Optometry*®

cope *Approval pending

SALUS UNIVERSITY Pennsylvania College of Optometry

Partially supported by an unrestricted educational grant from Shire Alcon

†Rooms limited. **Subject to change, separate registration required. See event website for complete details.

Incorporating these technologies also opens the doors to newer and more disease-specific treatment modalities. ■

Dr. Makhlouf is an assistant professor with a focus on primary eye care and ocular disease at Nova Southeastern University College of Optometry in Fort Lauderdale, Fla. She teaches the course in ophthalmic lasers and surgical comanagement.

Dr. Shechtman is a professor of optometry at Nova Southeastern University College of Optometry.

Dr. Reynolds is an associate professor of optometry at Nova Southeastern University College of Optometry.

1. Stanga P, Lim J, Hamilton P. Indocyanine green angiography in chorioretinal diseases: indications and interpretation: an evidence-based update. Ophthalmology. 2003;110:15–21.
2. Sohrab M, Wu K, Fawzi AA. A pilot study of morphometric analysis of choroidal vasculature *in vivo*, using *en face* optical coherence tomography. Prog Retin Eye Res. 2016 May;52:130-55.
3. Ferrara D, Waheed NK, Duker JS. Investigating the choriocapillaris and choroidal vasculature with new optical coherence tomography technologies. Exp Eye Res. 1973 Jan 1;15(1):15-29.
4. Margolis R, Spaide RF. A pilot study of enhanced depth imaging optical coherence tomography of the choroid in normal eyes. Am J Ophthalmol. 2009;147:811-5.
5. Arora KS, Jefferys JL, Maul EA, Quigley HA. Choroidal thickness change after water drinking is greater in angle closure than in open angle eyes. Invest Ophthalmol Vis Sci. 2012 Sep 21;53(10):6393-402.
6. Whitmore SS, Sohn EH, Chirco KR, et al. Complement activation and choriocapillaris loss in early AMD: implications for pathophysiology and therapy. Prog Retin Eye Res. 2015 Mar;45:1-29.
7. Giovannini A, Amato GP, Mariotti C, et al. OCT imaging of choroidal neovascularization and its role in the determination of patients eligibility for surgery. Br J Ophthalmol. 1999;83:438–42.
8. Jia Y, Bailey ST, Wilson DJ, et al. Quantitative optical coherence tomography angiography of choroidal neovascularization in age-related macular degeneration. Ophthalmology. 2014;121:1435-44.
9. De Carlo TE, BoniniFilho MA, Chin AT, et al. Spectral-domain optical coherence tomography angiography of choroidal neovascularization. Ophthalmology. 2015;122:1228–38.
10. Moult E, Choi W, Waheed NK, et al. Ultrahigh-speed swept-source OCT angiography in exudative AMD. Ophthalmic Surg Lasers Imaging Retina. 2014;45(6):496–505.
11. Lupidi M, Coscas G, Cagini C, Coscas F. Optical coherence tomography angiography of a choroidal neovascularization in adult onset foveomacular vitelliform dystrophy: pearls and pitfalls. Invest Ophthalmol Vis Sci. 2015 Dec;56(13):7638-45.
12. BoniniFilho MA, de Carlo TE, Ferrara D, et al. Association of choroidal neovascularization and central serous chorioretinopathy with optical coherence tomography angiography. JAMA Ophthalmol. 2015;133(8):899–906.
13. Novais, E, Baumal, C. The clinical utility of oct angiography. Review of Ophthalmology. 2017;24(1):51-5.
14. E de Carlo T, Romano A, Waheed NK, Duker JS. A review of optical coherence tomography angiography (OCTA). International Journal of Retina and Vitreous. 2015;1:5.
15. Warrow DJ, Hoang QV, Freund KB. Pachychoroid pigment epitheliopathy. Retina. 2013;33(8):1659-72.
16. Dolz-Marco R, Dansingani KK, Freund SB. Identifying the pachychoroid phenotype. Review of Ophthalmology. 2016;23(7):60-4.
17. Gallego-Pinazo R, Dolz-Marco R, Gómez-Ulla F, et al. Pachychoroid diseases of the macula. Med Hypothesis Discov Innov Ophthalmol. 2014;3(4):111-5.
18. Dansingani KK, Balaratnasingam C, Naysan J, Freund KB. *En face* imaging of pachychoroid spectrum disorders with swept-source optical coherence tomography. Retina. 2016;36:499-516.
19. Sato T, Kishi S, Watanabe G, et al. Tomographic features of branching vascular networks in polypoidal choroidal vasculopathy. Retina. 2007 Jun;27(5):589-94.
20. Imamura Y, Engelbert M, Iida T, et al. Polypoidal choroidal vasculopathy: a review. Surv Ophthalmol. 2010 Nov-Dec; 55(6):501-15.
21. Lim TH, Laude A, Tan CS. Polypoidal choroidal vasculopathy: an angiographic discussion. Eye (Lond). 2010 Mar; 24(3):483-90.
22. Shantha JG, Kokame GT. Polypoidal choroidal vasculopathy. Retina Today. 2016;11(4):64-8.
23. Ozawa S, Ishikawa K, Ito Y, et al. Differences in macular morphology between polypoidal choroidal vasculopathy and exudative age-related macular degeneration detected by optical coherence tomography. Retina. 2009;29(6):793-802.
24. Liu R, Li J, Li Z, et al. Distinguishing polypoidal choroidal vasculopathy from typical neovascular age-related macular degeneration based on spectral domain optical coherence tomography. Retina. 2016;36(4):778-86.
25. Srour M, Querques G, Souied EH. Optical coherence tomography angiography of idiopathic polypoidal choroidal vasculopathy. Dev Ophthalmol. 2016;56:71-6.
26. Spaide RF. Age-related choroidal atrophy. Am J Ophthalmol. 2009;147(5):801-10.

PEER • REVIEWED PRA ARTICLE

The Larry Alexander Resident Case Report Contest:

Acute Syphilitic Posterior Placoid Chorioretinitis

After an outbreak, ODs must consider the differential diagnosis for syphilis.

By Kathryn Dailey, OD

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

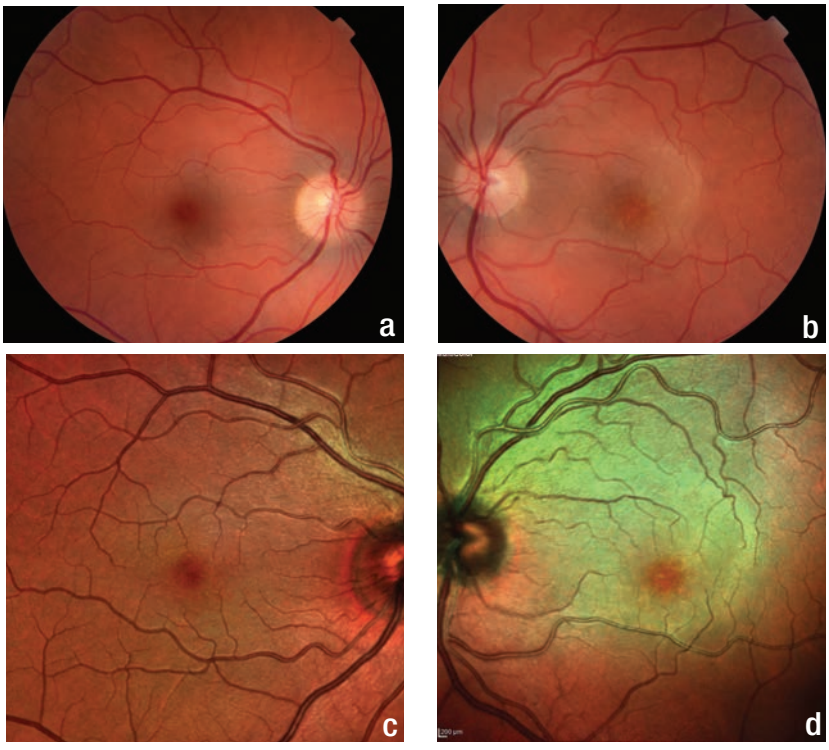


Fig. 1. The fundus and multicolor images of the patient's right eye (a, c) show no abnormalities, but the same images of her left eye (b, d) show a yellow, plaque-like lesion extending from the optic nerve into the superior macula.

In March 2016, the Centers for Disease Control and Prevention (CDC) issued a clinical advisory for ocular syphilis due to a 12-case cluster reported in San Francisco and Seattle over four months.[1] Although ocular involvement from syphilis is rare, increasing incidence rates suggest clinicians must consider syphilis on their list of differential diagnoses.

This article describes a rare case of acute syphilitic posterior placoid chorioretinitis (ASPPC) in an immunocompetent female secondary to syphilis. It also provides a brief review of systemic syphilis and an in-depth review of the literature regarding ASPPC, as well as discusses the laboratory evaluation and treatment of confirmed systemic syphilis.

Back in the States

A 46-year-old female presented to the clinic with concerns of a black spot in the central vision of her left eye. She reported the spot appeared upon awakening the day before and had gotten worse. The patient's ocular history was unremarkable. Her last eye exam was one year prior at a different clinic. She had recently noted swollen lymph nodes on her abdomen after a trip to Mexico. Further imaging showed cysts on her ovary near the swollen

CATHY CATARACTS & ANDY ASTIGMATISM

2 EYE CONDITIONS 1 PROCEDURE

GET **TWO BIRDS WITH ONE STONE**.
HELP YOUR PATIENTS CORRECT
CATARACTS & ASTIGMATISM
WITH **ONE PROCEDURE**.

Talk to your astigmatic patients about toric IOL options earlier, and help them see cataract surgery as an opportunity to correct two eye conditions at once.

mycataracts.com: online patient resources
1-844-MYCATARACT (1-844-692-2827): cataract counselors

Alcon A Novartis Division

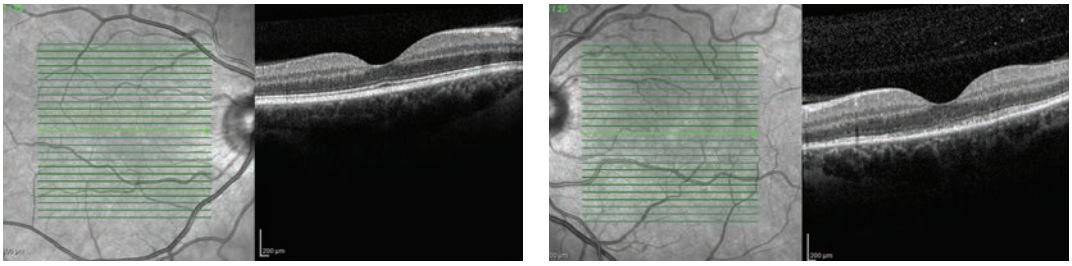


Fig. 2. While our patient's right macula appeared normal on OCT, her left eye demonstrated a disruption of the PIL.

lymph nodes and she was scheduled for an MRI the next week. She acknowledged high levels of stress secondary to the recent death of her best friend, but denied recent illness and neurologic symptoms.

Her medical history was significant for post-traumatic stress disorder, anxiety, chronic neck pain, gestational diabetes and gastroesophageal reflux disease. Her medications included methocarbamol, omeprazole, oxybutynin chloride and venlafaxine.

Diagnostic Data

Her entering visual acuities (VA) were 20/20 OD and 20/70+2 OS with no improvement on pinhole. Extraocular motilities, confrontation visual fields and pupils were all normal. During pupil testing, the patient noted the light was dimmer in the left eye than the right. Amsler grid testing showed a large central scotoma in her left eye.

Slit lamp examination of the anterior segment was normal with quiet anterior chambers in both eyes. Intraocular pressures (IOP) were normal. There was trace vitreous cell in the right eye and grade one vitreous cell in the left. Retinal evaluation revealed a normal retina in the right eye and a yellow plaque-like lesion in the outer retina of her left eye extending superiorly from the nerve (*Figure 1*).

Macular optical coherence tomography (OCT) revealed a normal foveal contour with significant disruption of the photoreceptor integrity line below the fovea and extending through the superior nasal macula in the left eye (*Figure 2*). Fundus autofluorescence (FAF) demonstrated hyperautofluorescence around the optic disc and superior macula in the patient's left eye (*Figure 3*).

Due to the patient's age and the acute nature of the vision loss, we recommend she undergo fluorescein angiography (FA) that day for further evaluation. The FA showed early mild hypofluorescence around the optic disc and superior macula with mottled hyperfluorescence in the area of the plaque in the late phase in the left eye (*Figure 4*).

Based upon the FAF, FA and OCT results, the retinal specialist who completed the FA was concerned about the unknown etiology of the posterior uveitis.

We ordered a comprehensive lab work-up, which included a complete

Syphilis Basics

Syphilis is a sexually transmitted systemic infection caused by the spirochete bacterium *Treponema pallidum*. Despite the common perception that syphilis is a disease of the past, the CDC reports that 2015 saw the highest rate of cases of primary and secondary syphilis since 1994.[2,3] Ocular involvement in syphilis—though rare—is notoriously difficult to diagnose, due to the disease's ability to affect almost any part of the eye and mimic other inflammatory disorders.[4-7]

In a minority of patients with ocular syphilis, a distinctive oval, yellow, plaque-like chorioretinitis in the outer retina of the macular or juxtapapillary areas develops. This disease entity—acute syphilitic posterior placoid chorioretinitis— is estimated to occur in only 3% of patients with ocular syphilis.[6-8]

A study in 2012 reported only 60 known cases of ASPPC, of which only 13% occurred in females.[7]

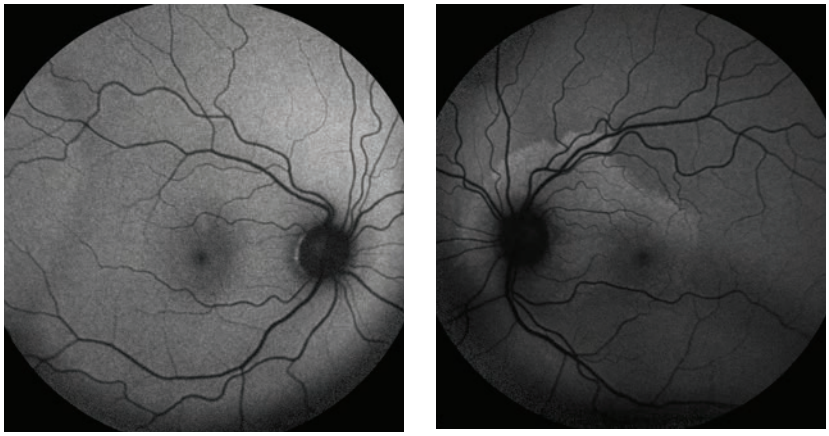


Fig. 3. Fundus autofluorescence demonstrates a normal right fundus, but the left shows mottled hyperfluorescence around the optic nerve and superior macula, indicating lipofuscin accumulation in the RPE.

blood count, a comprehensive metabolic panel, syphilis serologies, Quantiferon test for tuberculosis, toxoplasmosis serology and toxocariasis serology and a chest x-ray. A follow-up was scheduled in three days.

Diagnosis

At follow-up, the patient reported improved vision and a decrease in the size of the black spot. Uncorrected VA was 20/20 OD and 20/30 OS. Entrance testing, IOP and anterior segment evaluation were normal in both eyes. No change was seen in the appearance of either fundus. OCT of the left macula showed outer retinal disturbances with new hyperreflective areas at the RPE layer and extension of these changes into the inferior macula since the previous scan (*Figure 5*). FAF imaging revealed enlargement of the hyperfluorescent area (*Figure 6*).

All of the lab tests ordered at the initial presentation were normal, except for a positive enzyme immunoassay (EIA) used to assess for syphilis. Confirmatory testing for syphilis with the rapid plasma reagin (RPR) and *T. pallidum* particle agglutination assay tests were positive, and the RPR test showed a titer of 1:512. HIV titers, as well as screenings for gonorrhea and chlamydia, were all negative.

Based on the clinical and laboratory findings, we diagnosed the patient with ASPPC. At this point the patient revealed a history of unprotected intercourse with a promiscuous sexual partner who had a small, round, painless lesion on his genitals for the past month. She also reported several painful ulcers on her tongue and inside of her lower lip and a sore throat.

Treatment

The patient was referred to an infectious disease specialist, who completed a lumbar puncture was completed. A Venereal Disease Research Laboratory (VDRL) test on her cerebrospinal fluid (CSF) was negative. Although the standard treatment for syphilis is penicillin, the patient had a significant penicillin allergy and reported a blistering rash, mouth sores and difficulty breathing within 30 minutes of taking the drug on two separate occasions. Therefore, she was started on two grams of intravenous (IV) ceftriaxone per day for 14 days.

The patient was kept in the hospital for her first dose due to the small risk of cross-reactivity between penicillin and cephalosporin drugs. She reported throat itching and pain during the initial dose, but there were no objective signs of allergic reaction and

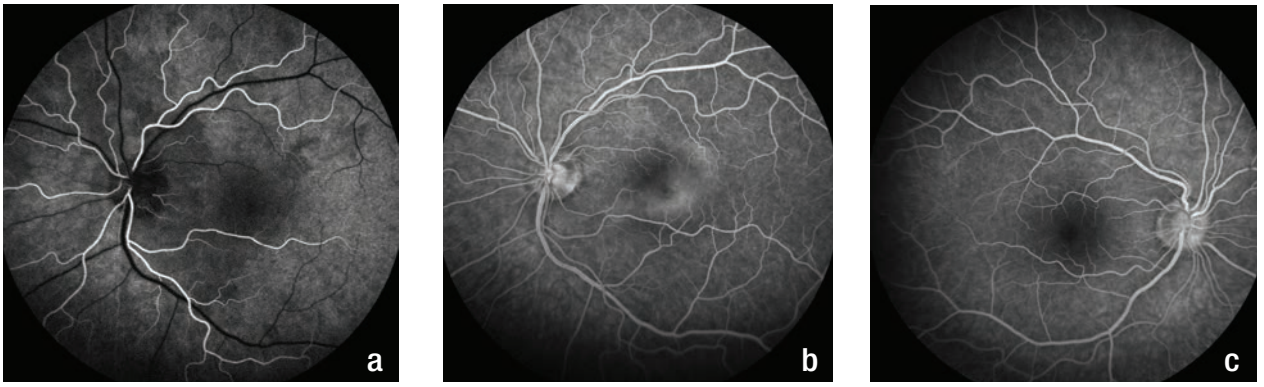


Fig. 4. Fluorescein angiography shows (a) hypofluorescence superior to the optic disc and around the left macula in the early phase, (b) mottled hyperfluorescence, known as "leopard spotting," in the area of the plaque lesion in the late phase in the patient's left eye and (c) normal fundus appearance during the late phase in the right eye.

the two-week treatment was completed at home.

At her six week follow-up, the patient felt her vision had improved further. VA was 20/20 OD and OS. Retinal examination showed mild RPE mottling in the macula OS with resolution of the yellow-plaque lesion. OCT showed resolved PIL disruption subfoveally, with remaining mild outer retinal granularity in the inferior macula and FAF imaging was normal (*Figures 5 and 6*). Despite the mild vitreous cell in her right eye at the initial exam, there were no signs of further ocular involvement.

Discussion

In cases of acquired syphilis, transmission of *T. pallidum* occurs through direct contact with an infectious lesion during sex. Four stages of disease have traditionally been described: primary, secondary, latent and tertiary. The primary stage is characterized by a painless lesion, known as a chancre, at the site of inoculation after an average incubation period of 21 days.[5,9] The secondary stage begins a few weeks to months after the chancre develops in 25% of untreated patients and is associated with a wide range of symptoms.[10] Latency may follow the secondary stage. The patient is asymptomatic during latency, and may remain in latency indefinitely, relapse into the secondary stage or develop tertiary syphilis. Tertiary syphilis can occur from one to 30 years after primary infection and is when the most serious systemic manifestations occur.[5,9]

Neurosyphilis is a distinct category of the disease that can occur at any time during the course of infection, and is regarded as any central nervous system manifestation. Distinction of neurosyphilis is important due to unique diagnostic and treatment guidelines.[11] Eye care providers should be aware that any ocular involvement is considered a manifestation of neurosyphilis.

Ocular involvement in syphilis most frequently occurs during the secondary and tertiary stages of the infection, but has been reported in all stages.[4-7] On average, 1% to 2% of cases of ocular inflammation are estimated to be secondary to syphilis, and the ocular manifestations are extremely varied.[5,12] Only 3% of cases of ocular syphilis result in the distinctive oval, plaque-like chorioretinitis known as ASPPC.

ASPPC has been described as bilateral and unilateral with a mean age of 40 at presentation.[7] ASPPC is significantly more rare in women than men (13% vs. 87%).[7] Presenting visual acuity ranges from 20/20 to count fingers, with a median of 20/80.[7] Mucocutaneous manifestations are the most common associated systemic finding with ASPPC, while anterior chamber or vitreous inflammation are the most common concurrent ocular findings.[6,7,13-16]

Identification

ASPPC has distinct clinical, angiographic, autofluorescence, multifocal ERG (mfERG) and OCT features. On funduscopic exam, all eyes with ASPPC exhibit a yellow, circular or oval placoid lesion in the outer retina, rarely extending beyond the posterior pole.[6] FA shows early hypofluorescence within the lesion followed by progressive punctate hyperfluorescence, known as "leopard spotting," in the late stage.[6,7] Although FA has been used more widely for evaluation of ASPPC, some authors suggest indocyanine green angiography (ICGA) may be better suited for evaluation to detect the full extent of choroidal inflammation.[25]

FAF is gaining popularity in eye care due to its ability to provide a qualitative measure of the structure and function of the RPE.[25]

FAF imaging during the acute stage of ASPPC shows punctate hyperautofluorescent spots within the lesion, consistent with lipofuscin accumulation at the RPE-photoreceptor junction.[6,7] Resolution of hyperautofluorescence on FAF, indicating improved RPE functioning, correlates to improvement in visual acuity.[25] This correlation suggests FAF may be a useful noninvasive tool for monitoring progressive inflammatory disorders such as ASPPC.[25]

OCT findings in the acute phase of ASPPC show thickening and hyperreflective nodularity of the RPE with disruption of the overlying photoreceptor integrity line (PIL).[18] In a minority of cases, OCT has revealed subretinal fluid.[23,24] Post-treatment, OCT imaging shows resolution of the RPE nodules with remaining mild disruption of the PIL.[18] Just as with FAF, return of the PIL on OCT correlates to improvement of visual acuity.

One study used serial mfERGs throughout the course of ASPPC in a single patient.[23] Their findings showed a reduced mfERG during the acute phase of the disease followed by a delay in the improvement of the mfERG compared with the improvement in visual acuity.[23] The authors concluded that their results suggest full photoreceptor recovery may actually be delayed compared with the apparent anatomical recovery on OCT.[23]

Good visual outcome after ASPPC is possible with and without prompt antibiotic therapy.[6,7,16,20] One study acheived improvement in VA to 20/25 or better in approximately 90% of cases.[6,7,15,16] Decreased vision after treatment was associated with persistent, significant disruption of the outer retinal layers, although mild disruption of these layers may remain in patients whose acuity returns to baseline.[6,23]

Differential diagnoses for ASPPC should include most posterior uveitides (*Table 1*).[7,26-28] Differential diagnosis of the posterior uveitides is often possible based upon history, examination and imaging (particularly FA, FAF and ICGA).[25,28]

Although resolution of ASPPC has been documented without treatment, appropriate diagnosis and treatment is critical to prevent subsequent systemic or ocular manifestations. Imaging tools can help raise suspicion of ASPPC, but accurate diagnosis of syphilis requires laboratory testing. Lab testing for syphilis is notoriously complex due to multiple testing algorithms and myriad tests available. All lab tests can be divided into treponemal specific vs. non-treponemal specific. Tests from both categories must be used to make a diagnosis.

Table 1. Differential Diagnosis of the Posterior Uveitides[7,26-28]

Cause	Clinical setting	Ancillary diagnostic testing	
		Ocular	**Systemic**
Idiopathic	Diverse	Diagnosis of exclusion	
Infectious			
Toxoplasmosis	Patch of white retinochoroiditis with speckled borders next to a pigmented scar	FA: hypofluorescence in the area of an old chorioretinal scar, early hypofluorescence and late hyperfluorescence with persistent leakage during active infection	Serum antibodies; High frequency false positives
CMV	Immunosuppressed patient with broad white patches of retinitis, often associated with hemorrhages	None	None required; HIV screening if no current diagnosis
Toxocariasis	Child with a white, round vitreous/inner retinal mass often extending from the optic nerve	None	Serum ELISA for anti-*Toxocara* antibodies
Tuberculosis	One or multiple slightly raised yellow, deep retinal patches with indistinct borders	None	PPD or Quantiferon gold
Syphilis	Any posterior uveitis	Varies depending upon presentation	VDRL, RPR, FTA-ABS
ARN	Patches of peripheral, white retinal necrosis	FA: peripheral areas of retinal arteriolar capillary nonperfusion	Aqueous PCR for VZV or HSV DNA
PORN	Immunosuppressed patient with deep, multifocal retinal lesions sparing retinal vasculature; often bilateral	FA: confirms lack of vasculitis	Aqueous PCR for VZV or HSV DNA
Inflammatory			
Sarcoidosis	Yellow-orange deep fundus lesions or larger peripheral full thickness white nodules; sometimes accompanied by an exudative vasculitis	FA: hypofluorescence in area of ischemia, venular leakage in area of inflamed vessels	ACE, chest x-ray
POHS	Classic triad of peripapillary atrophy, mid-peripheral histo spots, and macular lesions; vitreous cell will be absent	None	None
APMPPE	Creamy yellow deep retinal patches with indistinct borders	FA: early hypofluorescence with late hyperfluorescence in the area of active disease; ICGA: perfusion of the large choroidal vessels in the area of hypofluorescence in the early phase and marked choroidal hypofluorescence in the late phase	None
Serpiginous choroidopathy	Bilateral, well-circumscribed patches of yellow choroiditis that begin near the disc and extend in any direction	FA: early hypofluorescence with later hyperfluorescence in the area of active disease; hyperfluorescence begins at the margins of the lesion and spreads inward; ICGA: hypofluorescence through early and late phases; may be areas of hyperfluorescence outside the clinical lesion	None
Birdshot retino-choroidopathy	Multiple white/yellow oval spots with indistinct borders; often mostly nasally	FA: lesions aren't visible until late in the disease process, when they stain; ICGA: late phase hypofluorescence. Detects a greater number of spots than seen clinically; ERG: delayed implicit time and b-wave amplitude	HLA-A29
PIC	Few clustered white spots with distinct borders, no vitritis, usually women	FA: punctate hyperfluorescent late staining	None
MEWDS	Multiple subtle, deep, gray spots	FA: early hyperfluorescence that persists into late phase; ICGA: hypofluorescence in the area of the white dots, extensive area of hypofluorescence surrounding the optic disc; VF: enlarged blind spot; ERG: depressed A-wave, recovers during resolution of the disease	None
VKH Syndrome	Nonrhegmatogenous retinal detachments, often with deep, gray choroidal spots; patients often note tinnitus or severe headaches	FA: diffuse, pinpoint hyperfluorescent leaks at the level of the RPE, late pooling in the subretinal space; ICGA: multiple hypofluorescent spots; Ultrasound: shifting exudative RD	Often involves multi-system work-up including LP, audiogram, pathology, and genetic testing for HLA-DR405

CMV: cytomegalovirus, ARN: acute retinal necrosis, PORN: progressive outer retinal necrosis, POHS: presumed ocular histoplasmosis syndrome, APMPPE: acute posterior multifocal placoid pigment epitheliopathy, PIC: punctate inner choroidopathy, MEWDS: multiple evanescent white dot syndrome, VKH: Voyt-Koyanagi-Harada.

Testing

Non-treponemal tests assess the reactivity of serum to a cardiolipin-cholesterol-lecithin antigen and are, therefore, nonspecific for syphilis.[3] Traditionally, these tests were used for initial screenings, due to their low cost, ease of performance and quantifiability.[3] Results from these tests are given in a titer, which gives an indirect indication of activity of the infection and allows treatment response to be monitored. Our patient had a titer of 1:512, meaning her serum could be diluted 512 times and still be reactive.

Treponemal specific tests are traditionally used as confirmatory tests after a positive non-treponemal test because of their higher complexity and cost.[3] These tests are specific for serum antibodies against treponemal antigens, but are qualitative only. Newer, more automated versions of treponemal tests now exist, and are increasingly being used as the initial screening rather than a non-treponemal test.[29] These modern tests have flipped the traditional testing algorithm on its head, but positive tests from both the non-treponemal and treponemal categories are still required for diagnosis. New point-of-care tests that detect both treponemal and non-treponemal antibodies may become the future of syphilis diagnostic testing.[30]

All patients with suspected neurosyphilis require examination of the CSF to make a definitive diagnosis, and should also undergo testing for HIV.[2,3,31,32] A positive CSF-VDRL is highly specific for neurosyphilis, but sensitivity is very poor. The test may be negative in up to 70% of people with neurosyphilis, as was the case in our patient.[32] Of the patients in the case studies of ASPPC reviewed, only 50% to 75% had positive CSF testing.[6,7] Regardless of the results of CSF testing, patients with ocular involvement should be treated according to neurosyphilis guidelines. If CSF testing is positive, treatment response should be monitored via CSF rather than serum testing.

Medical Intervention

Penicillin has been the treatment of choice for syphilis since its introduction in the mid-1900s. Prolonged levels of penicillin are required to eliminate *T. pallidum*, and the route of administration and duration varies depending on the stage of the disease. In cases of neurosyphilis due to poor penetration of the ocular structures and central nervous system via IM administration, IV penicillin G is given.[33] Adequate treatment response is considered a fourfold decline in the non-treponemal titer.[7] Most patients remain serofast after treatment with stable titers for life.[33,34]

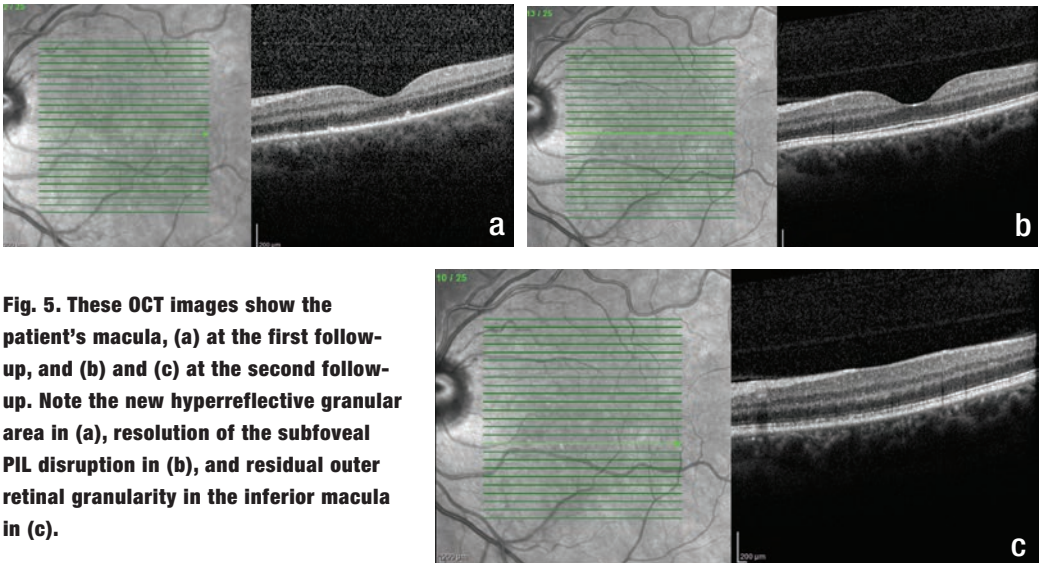


Fig. 5. These OCT images show the patient's macula, (a) at the first follow-up, and (b) and (c) at the second follow-up. Note the new hyperreflective granular area in (a), resolution of the subfoveal PIL disruption in (b), and residual outer retinal granularity in the inferior macula in (c).

Treatment of patients who are severely allergic to penicillin involves an alternative agent such as the tetracyclines or ceftriaxone. There is limited data to support the dosage, duration and efficacy of these alternatives.[3,33] This was the case in our patient, and treatment with IV ceftriaxone resulted in a good clinical response.

ASPPC is a rare manifestation of syphilis with distinctive clinical and angiographic features. The disease seems to follow a course of onset-aggravation-resolution, sometimes prior to treatment. The pathophysiology of ASPPC is not well understood, but recent studies of FAF and mfERG findings indicate choroidal and RPE inflammation with significant, often long-lasting, disruption of the photoreceptors.

Because the CDC has issued a clinical advisory for ocular syphilis, more patients may be presenting with manifestations of the disease. Since only 50% of patients with ocular syphilis have any systemic signs, eye care providers must maintain a high index of clinical suspicion to make a diagnosis. Syphilis is much more likely to present in men, but as shown in this report, women may also be affected. Patient history, clinical examination, imaging characteristics and targeted laboratory testing are critical for diagnosis in cases of posterior uveitis of unknown etiology. All patients diagnosed with ocular syphilis should be tested for both neurosyphilis and HIV coinfection. In addition, comanagement with an infectious disease specialist is appropriate to ensure proper treatment and fulfillment of reporting and counseling requirements set forth by the CDC. ■

Dr. Dailey practices in the Optometry Department of the Veteran's Administration Puget Sound Health Care System – American Lake. She is the winner of the First Annual Larry Alexander Resident Case Report Contest. *She would like to acknowledge Jeffrey Hiett, OD, and Judith Oh, OD, for their guidance and expertise during the writing process.*

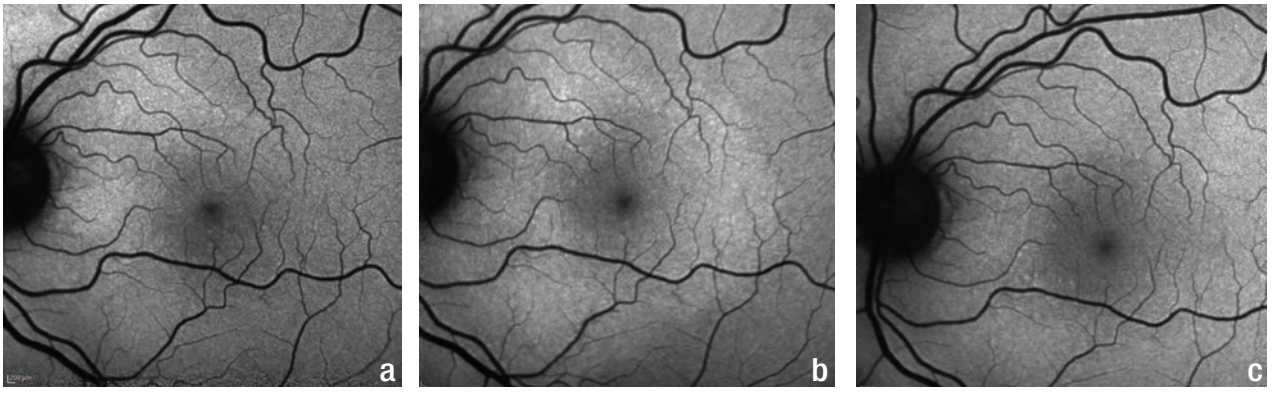


Fig. 6. Serial FAF photos of the left eye at (a) initial presentation, (b) first follow-up, and (c) second follow-up. Note the extension of the hyperfluorescence inferior to the optic nerve and macula in (b) and resolution of hyperfluorescence in (c). These findings were consistent with the changes in the PIL shown in Figure 5.

1. Centers for Disease Control and Prevention. Clinical Advisory: ocular syphilis in the United States 2015. Atlanta, GA: US Health and Human Services, CDC; 2016. http://cdc.gov/std/syphilis/clinicaladvisoryos2015.htm. Accessed January 8, 2017.
2. Kenyon CR, Osbak K, Tsoumanis A. The global epidemiology of syphilis in the past century – a systematic review based on antenatal syphilis prevalence. PLoS Negl Trop Dis. 2016;10(5):e0004711.
3. Centers for Disease Control and Prevention. Sexually Transmitted Disease Surveillance. http://www.cdc.gov/std/stats15/std-surveillance-2015-print.pdf. Updated 2015. Accessed on January 8, 2017.
4. Moradi A, Salek S, Daniel E, et al. Clinical features and incidence rates of ocular complications in patients with ocular syphilis. Am J Ophthalmol. 2015;159:334-43.
5. Sahin O, Ziaei A. Clinical and laboratory characteristics of ocular syphilis, co-infection, and therapy response. Clin Ophthalmol. 2016;10:13-28.
6. Pichi F, Ciardella AP, Cunningham ET, et al. Spectral domain optical coherence tomography findings in patients with acute syphilitic posterior placoid chorioretinopathy. Retina. 2014;34(2):373-84.
7. Eandi CM, Neri P, Adelman RA, et al. Acute syphilitic posterior placoid chorioretinitis. Report of a case series and comprehensive review of the literature. Retina. 2012;32(9):1915-41.
8. Gass JD, Braunstein RA, Chenoweth RG. Acute syphilitic posterior placoid chorioretinitis. Ophthalmology. 1990;97(10):1288-97.
9. Sparling PF. Natural history of syphilis. In: Sexually Transmitted Diseases. Holmes KK, Mardh PA, Sparling PF et al (Eds), New York;McGraw-Hill 1990:213.
10. Chapel TA. The signs and symptoms of secondary syphilis. Sex Transm Dis. 1980;7(4):161-4.
11. Workowski KA, Bolan GA, Centers for Disease Control and Prevention. Sexually transmitted diseases treatment guidelines 2015. MMWR Recomm Rep. 2016;64:1-137.
12. Tamesis RR, Foster CS. Ocular syphilis. Ophthalmology. 1990;97(10):1281-7.
13. Yap SC, Tan YL, Chio MT, et al. Syphilitic uveitis in a Singaporean population. Ocul Immunol Inflamm. 2014;22(1):9-14.
14. Anshu A, Cheng CL, Chee SP. Syphilitic uveitis: an Asian perspective. Br J Ophthalmol. 2008;92(5):594-7.
15. Puech C, Gennai S, Pavese P, et al. Ocular manifestations of syphilis: Recent cases over a 2.5-year period. Graefes Arch Clin Exp Ophthalmol. 2010;248(11)1623-9.
16. de Souza EC, Jalkh AE, Trempe CL, et al. Unusual central chorioretinitis as the first manifestation of early secondary syphilis. Am J Ophthalmol. 1988;105(3):271-6.
17. Burkholder BM, Leung TG, Ostheimer TA, et al. Spectral domain optical coherence tomography findings in acute syphilitic posterior placoid chorioretinitis. J Ophthalmic Inflamm Infect. 2014;4:2.
18. Franco M, Noguiera V. Severe acute syphilitic posterior placoid chorioretinitis with complete spontaneous resolution: the natural course. GMS Ophthalmol Cases. 2016: 6:Doc02.
19. Zamani M, Garfinkel R. Corticosteroid-induced modulation of acute syphilitic posterior placoid chorioretinitis. AJO. 2003:135(6):891.
20. Ji YS, Yang JM, Park SW. Early resolved acute syphilitic posterior placoid chorioretinitis. Optom Vis Sci. 2015; 92:S55.
21. Song JH, Hong YT, Kwon OW. Acute syphilitic posterior placoid chorioretinitis following intravitreal triamcinolone acetonide injection. Graefes Arch Clin Exp Ophthalmol. 2008;246:1775-8.
22. Erol N, Topbas S. Acute syphilitic posterior placoid chorioretinitis after an intravitreal triamcinolone acetonide injection. Acta Ophthalmol Scand. 2006;84:435.
23. Joseph A, Rogers S, Browning A, et al. Syphilitic acute posterior placoid chorioretinitis in non-immunocompromised patients. Eye (Lond). 2007; 21:1114.
24. Yoo C, Kim SK, Huh K, et al. Atypical acute syphilitic posterior placoid chorioretinitis. Korean J Ophthalmol. 2009;23(2):108.
25. Malamos P, Masaoutis P, Georgalas I, et al. The role of fundus autofluorescence imaging in the study of the course of posterior uveitis disorders. Biomed Res Int. 2015; 247469.
26. Huang JJ, Gaudio PA. Posterior uveitis and panuveitis. In:Ocular Inflammatory Disease and Uveitis Manual: Diagnosis and Treatment, Huang JJ, Gaudio PA. Lippincott Williams & Wilkins. Philadelphia;2010:70.
27. Nakamoto D, Gaudio P. Systemic diseases associated with ocular inflammation. In: Ocular Inflammatory Disease and Uveitis Manual: Diagnosis and Treatment, Huang JJ, Gaudio PA. Lippincott Williams & Wilkins;Philadelphia 2010:162.
28. Jabs D, Busingye J. Approach to the diagnosis of the uveitides. Am J Opthalmol. 2013;156(2):228-36.
29. Sena AC, White BL, Sparling PF. Novel treponema pallidum serologic tests: a paradigm shift in syphilis screening for the 21st century. Clin Infect Dis. 2010;51:700-8.
30. Castro AR, Esfandiari J, Kumar S, et al. Novel point-of-care test shows good performance in simultaneous detecting nontreponemal and treponemal antibodies in patients with syphilis. J Clin Microbiol. 2010;48:4615-9.
31. Doris JP, Saha K, Jones NP, et al. Ocular syphilis: the new epidemic. Eye. 2006;20:73-5.
32. Sparling PF. Diagnosis of neurosyphilis: new tools. Sex Trans Dis. 2010;37:288-9.
33. Clement ME, Okeke NL, Hicks CB. Treatment of syphilis: a systematic review. JAMA 2014;312:1905-17.
34. Romanowski B, Sutherland R, Fick GH, et al. Serologic response to treatment of infectious syphilis. Ann Intern Med. 1991;114:1005-9.
35. Centers for Disease Control and Prevention. 2015 Sexually Transmitted Disease Treatment Guidelines: Syphilis. Atlanta, GA: US Health and Human Services, CDC. 2016. https://www.cdc.gov/std/tg2015/syphilis.htm. Accessed January 25, 2017.

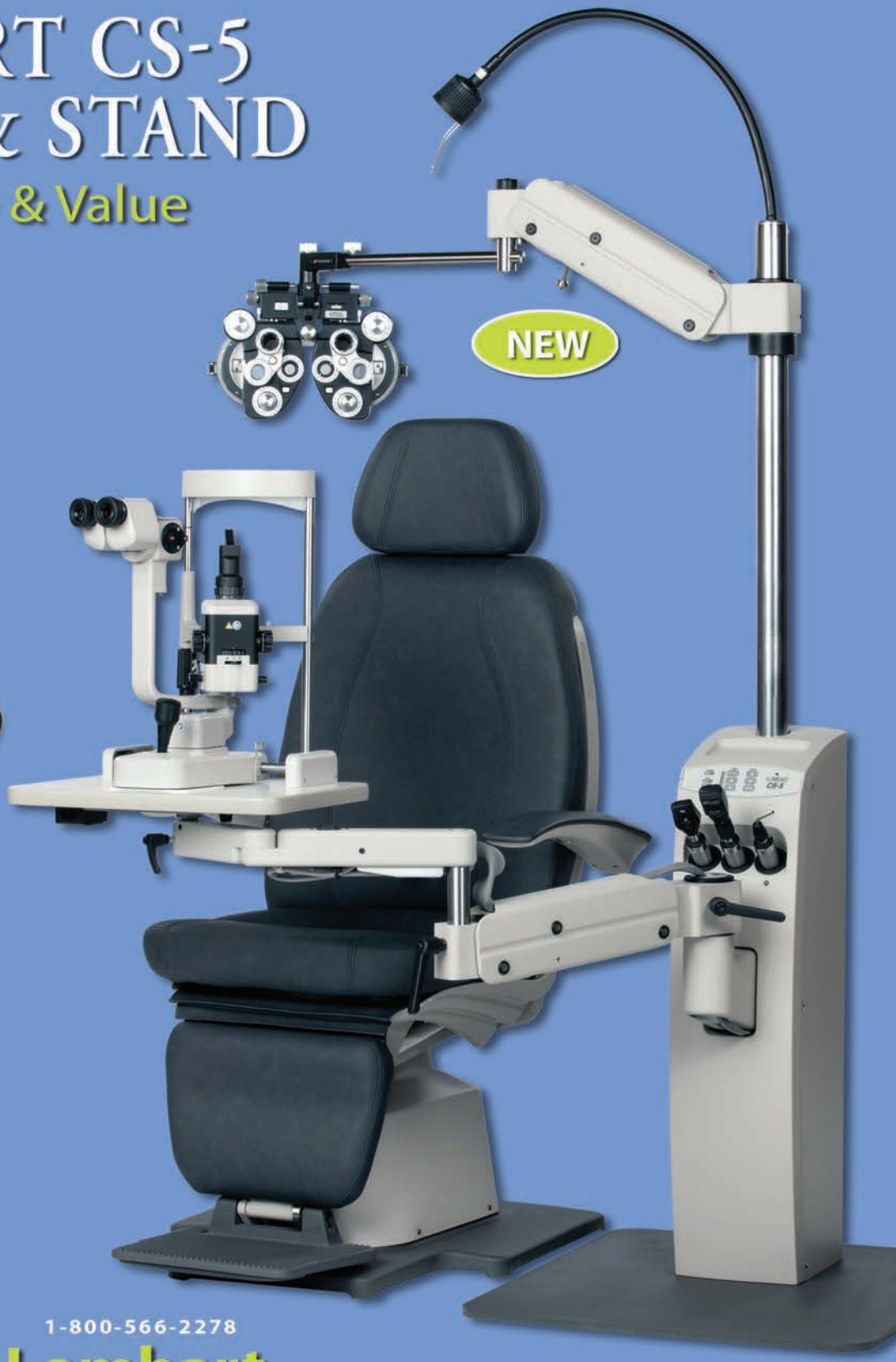
LOMBART CS-5 CHAIR & STAND

Quality, Style & Value

Package includes:

- The ***Lombart CS-5*** Chair & Stand
- ***Topcon VT-10*** Refractor
- ***Topcon SL-2G*** Slit Lamp

- ***Lombart CVS Essential Visual Acuity System*** with RF Remote Control
- Additional upgrades & configurations available.



$13,995

Or lease for $277/mo. for 60 months*

*Lease rate subject to credit approval, 1st payment is due at signing with 59 remaining rental payments of $265 and a $1.00 purchase option. Taxes, freight and installation additional. Hand Instruments optional. Subject to change without notice.

1-800-566-2278

Call 1-800-Lombart

Or Your Local Lombart Representative

Corporate Office - 5358 Robin Hood Road, Norfolk, VA 23513-2430
757-853-8888 | FAX 757-855-1232 | 800-566-2278
www.lombartinstrument.com
lombart@lombartinstrument.com

Sales and Service Centers Coast to Coast

ATLANTA • BALTIMORE/WASHINGTON D.C. • BOSTON • BOYNTON BEACH/MIAMI • BRADENTON • CHARLOTTE • CHICAGO • CINCINNATI • DALLAS • DENVER • DETROIT • GREENSBORO • HOUSTON
JACKSON • KANSAS CITY • KNOXVILLE • LOS ANGELES • MILWAUKEE • MINNEAPOLIS • NEW JERSEY/NEW YORK/PENNSYLVANIA • NORFOLK • PORTLAND • SAN ANTONIO • SAN DIEGO • SAN FRANCISCO

2 CE Credits (COPE approved)

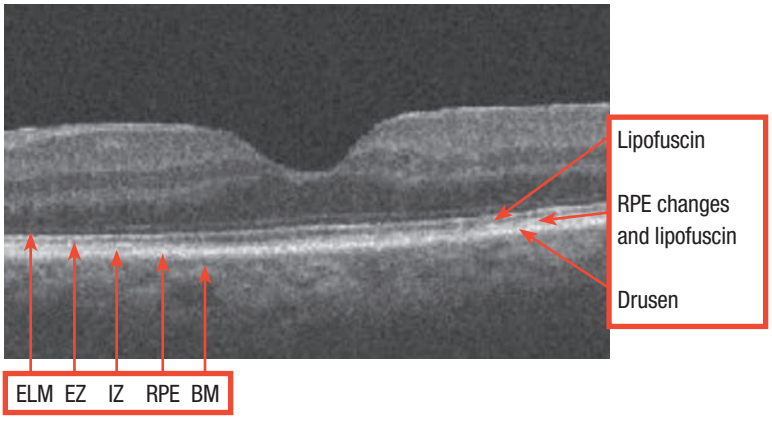
8th Annual Retina Report

PEER • REVIEWED • PRA • ARTICLE •

AMD Mimickers: When to Suspect Macular Dystrophy

Some presentations are different than they appeared at first glance. Here's how to spot the differences. **By Sara Weidmayer, OD**

Age-related macular degeneration (AMD) is a progressive disease of the central retina and the leading cause of blindness in the developed world.[1] While drusen are the trademark finding, AMD may present with any combination of drusen, retinal pigment epithelial (RPE) changes, geographic atrophy (GA) and choroidal neovascularization (CNV).[1] Given this variety of clinical presentations, several other diseases look similar to AMD, such as macular dystrophies. Here, we will explore how to differentiate AMD from some of the more commonly seen macular dystrophies so we can appropriately diagnose and manage the right disease and provide accurate prognostic information to our patients.



Normal retina with relevant structures noted. ELM: external limiting membrane. EZ: ellipsoid zone. IZ: interdigitation zone. RPE: retinal pigment epithelium. BM: Bruch's membrane.

Release Date: June 2017

Expiration Date: June 15, 2020

Goal Statement: Since many maculopathies appear quite similar clinically, this article is designed to educate practitioners about similarities and differences between several types of maculopathies, as well as explain how imaging devices and other tools can be used to differentiate these entities.

Faculty/Editorial Board: Sara Weidmayer, OD

Credit Statement: This course is COPE approved for 2 hours of CE credit. Course ID is **53907-PS**. Check with your local state licensing board to see if this counts toward your CE requirement for relicensure.

Disclosure Statements:

Authors: The author has no relationships to disclose.

Editorial staff: Jack Persico, Rebecca Hepp, William Kekevian, Michael Riviello and Michael Iannucci all have no relationships to disclose.

Table 1. How Dystrophies Compare with AMD

Dystrophy	Similarities with AMD	Differences from AMD	Helpful Ancillary Studies
ML	• Drusen • Possible GA or CNV	• Earlier onset (3rd-4th decade) • Likely (+)family history (autosomal dominant) • Smaller radial macular drusen, larger drusen around disc; confluent drusen later on	• Large drusen may hyper-AF (unlike drusen in AMD)
Stargardt's, fundus flavimaculatus	• Yellowish flecks • Possible GA	• Typically early onset (<3rd decade), but rarely can have late onset (>50 years) • Flecks are irregularly shaped and typically extend beyond macula • Flecks may spare the macula • Rarely develops CNV • Likely (+) family history (autosomal recessive)	• Yellowish flecks intensely hyper-AF on FAF and may have surrounding hypo-AF halo • OCT shows flecks as RPE thickening (not sub-RPE as with drusen)
Butterfly pattern dystrophy	• Pigmentary changes • Possible GA or CNV	• Mid-life onset (20-50 yrs) • Likely (+) family history • Butterfly pattern of pigmentary changes including lipofuscin	• Butterfly pattern of pigmentary changes on FAF, hyper-AF if lipofuscin present
AFVM	• Yellowish macular lesion • Atrophic changes in later stages • Possible CNV	• Mid-life onset (20-50 yrs) • No drusen • Vitelliform lesion is usually solitary	• Vitelliform lesion is anterior to RPE (between RPE and retina) with intense hyper-AF on FAF
BVMD	• Yellowish macular lesion • Atrophic changes in later stages	• Earlier onset (<40 yrs) • No drusen • Likely (+) family history (autosomal dominant) • Associated with hyperopia • Rarely develops CNV	• Vitelliform lesion is anterior to RPE (between RPE and retina) with intense hyper-AF on FAF
CACD	• Looks like GA	• Typically earlier onset (5th-6th decade), can be later (>55 years) • Usually no drusen • Likely (+) family history (autosomal dominant) • Multiple oval atrophic patches	• Very sharply demarcated hypo-AF atrophic patches on FAF
Cone dystrophy	• Pigmentary changes • Possible GA	• Most often earlier onset (30-60 years) • Possible (+) family history • No drusen • Possible peripheral bone-spicule pigmentation and/or temporal optic disc pallor	• Atrophic changes on FAF • Outer retinal thinning on OCT with loss of hyperreflective outer bands (EZ, IZ) • Significant decrease in photopic response on ERG • Color testing abnormal

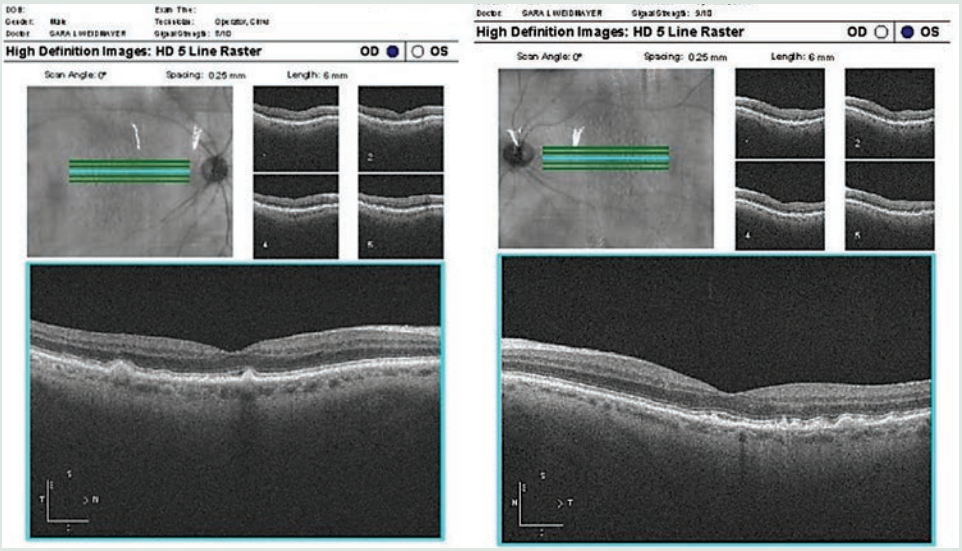
Clinical Features of AMD

The best way to detect a counterfeit dollar bill is not to know all of the possible fraudulent features of a counterfeit, but rather, to know the details of a true dollar bill. Similarly, the best way to detect AMD mimickers is to understand the features of AMD itself.

Drusen, the hallmark of AMD, are small extracellular byproduct deposits that accumulate posterior to the RPE, between the RPE and Bruch's membrane.[1,2] They appear yellowish and vary from small (<63µm) to large (>125µm).[1,3] While small drusen may appear simply due to aging and do not necessarily indicate early AMD, medium drusen without pigmentary changes fits the criteria for early AMD. Intermediate stage AMD includes any large drusen, or any macular pigmentary changes associated with medium or large drusen. Late AMD shows GA, CNV or disciform scarring (*Case 1*).[1,4]

The pigmentary changes seen in AMD occur as a result of RPE degeneration or migration.[1] Several factors contribute to the degeneration of RPE cells, including oxidative stress and inflammation.[1] Oxidative stress may occur due to the RPE's physiologic role in outer segment phagocytosis and environmental factors including chemical oxidative stress related to smoking, high fat diets and photo-oxidative stress from sunlight exposure. These oxidative stressors trigger an immune response, resulting in both acute and chronic regional inflammation, ultimately injuring the tissue.[5] RPE degeneration clinically appears as areas of RPE mottling or atrophy; migratory changes may look like pigment clumps, typically at the level of the RPE, but it is not uncommon to

Case 1. Drusen in Dry AMD Patient



These OCT images show typical drusen in dry AMD. Notice that the drusen are under the RPE.

see pigment migration into the outer, or even more anterior, layers of the retina; this is particularly evident on optical coherence tomography (OCT). GA shows larger, defined patches of choriocapillaris, RPE and subsequently outer retinal atrophy (*Case 2*).[1]

Any area of disruption in the Bruch's membrane-RPE complex, including drusen, RPE mottling or atrophy, can compromise the blood-retina barrier and predispose it to the development of CNV. This condition is caused by new blood vessels that form from the choriocapillaris, through Bruch's membrane and into the sub-RPE or subretinal space; exudation of blood and plasma from CNV causes tissue disruption and later leads to the fibrovascular scar formation that is typical in exudative AMD. CNV may develop in patients with macular dystrophies, but this is infrequent. Fortunately, CNV associated with macular dystrophies generally has a better prognosis than CNV associated with AMD.[1]

Symptoms typical of AMD are progressive central visual acuity loss, central scotomas and metamorphopsia. Slowed dark adaptation is common in patients with AMD, and patients may be symptomatic for this before there is any loss of central visual acuity.[1]

Though AMD diagnosis seems straightforward enough, mimickers can trip ODs up. Macular dystrophies in particular have considerable overlap with AMD in the clinical features and symptoms. Luckily, today's diagnostic tools provide the enhanced ability to detect and differentiate imposters.

Ancillary Tools

Color testing. This is a simple way to get a glimpse at the patient's central cone function. Color discrimination can be reduced in AMD, but usually not by any substantial amount. Color vision is generally more notably reduced in macular dystrophies, particularly those specifically affecting cone function, such as cone dystrophy.[1] Serial color vision testing may assist in monitoring functional disease progression in any maculopathy.

Visual fields. While AMD certainly will show variable degrees of central visual field (VF) loss, and VFs aren't typically routinely repeated in AMD patients, some macular dystrophies in their early stages may cause patient symptoms before clinical signs. Especially in patients with complaints of qualitative vision loss but an unremarkable fundus, a central VF (such as 10-2) or microperimetry may aid in the detection and monitoring of functional progression of early macular dysfunction more precisely than standard high-contrast acuity measurements. Both mesoptic and scotopic microperimetry can be performed, and the functional sensitivity results may be correlated to—and tracked along with—the structural findings seen simultaneously on OCT.[6]

Fundus autofluorescence (FAF). This can serve as one of the clinician's best tools to help differentiate AMD from macular dystrophies. On FAF, there is normally a low level of autofluorescence, which is derived from the autofluorescence of lipofuscin that is normally found at the level of the RPE.[1] These cells phagocytose the outer segment of photoreceptors as they are shed during the normal visual cycle; the phagocytosed material is stored in liposomes, which subsequently form lipofuscin that remains stored in the RPE.[7] Lipofuscin precursors can also accumulate between the RPE and retina, and these precursors also autofluoresce.[1] Relative to the normal background autofluorescence, any areas

of excess lipofuscin accumulation will appear as hyperautofluorescent (hyper-AF) on FAF, and funduscopically appears as a yellowish deposit. Conversely, areas of photoreceptor or RPE atrophy, thus with less lipofuscin, will appear hypo-autofluorescent (hypo-AF) or dark. There may be a junctional ring of hyper-AF around areas of RPE atrophy; this indicates the RPE is likely metabolically stressed in that zone, burdened with an abundance of shed outer segments and is generally indicative of impending atrophy in that area.[7]

Except for large or confluent drusen, which may be somewhat hyper-AF, drusen associated with AMD tend to be relatively isoautofluorescent (iso-AF), uniform with the low level of background autofluorescence.[1] While AMD may have some associated lipofuscin accumulation and may show variable autofluorescence patterns based on the clinical phenotype, specific macular dystrophies often show fairly characteristic distribution patterns of FAF findings. Also, many macular dystrophies begin earlier in life, so end up with higher amounts of lipofuscin accumulation over time, which ultimately intensifies the hyper-AF signal.[1]

OCT. The anatomic structure of the retina-to-choroid complex can be readily visualized using OCT (*Figure 1*). In differentiating AMD from mimickers, this offers extremely helpful additional information. Key points to note:

1. Drusen form posterior to the RPE, between the RPE and Bruch's membrane.

2. RPE changes are just that: RPE *changes*. RPE clumping or thickening can be visualized with OCT. RPE migration, often into the outer retina, can occur with AMD.

3. Lipofuscin or lipofuscin precursors are either within the RPE, or accumulate anterior to the RPE, between the RPE and retina.

Case 2. Geographic Atrophy

Notice the hypo-AF on FAF associated with the GA, and some patchy surrounding hyperf-AF surrounding the GA in the OCT, color photo and FAF image of this patient with AMD with GA and drusen. On OCT, the obvious drusen is posterior to the RPE, as expected, and the atrophy involves the RPE and outer retina.

Electroretinogram (ERG) can also help differentiate AMD from its mimickers. It may not be readily available to the primary eye care provider, but ERG may prove helpful in a number of these cases (particularly for deciphering cone dystrophy where there will be an obviously reduced photopic response).[1] As in any situation, when in doubt, investigate with further ancillary studies or refer the patient, as appropriate.

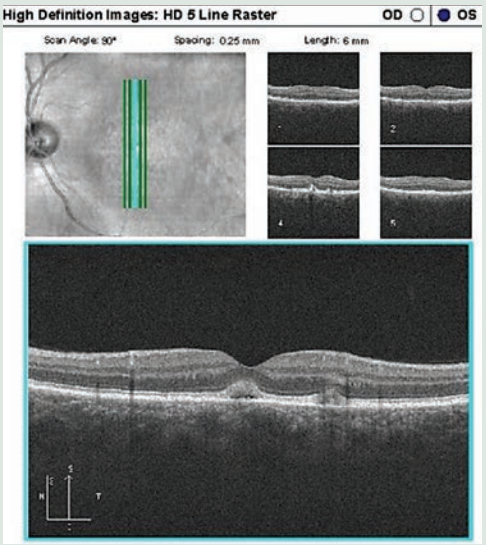
Family History

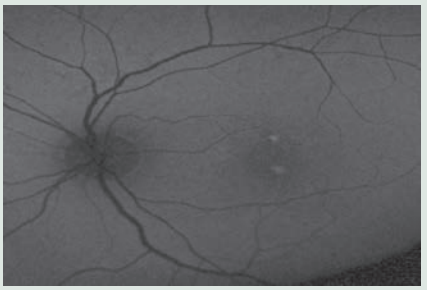
Clearly known environmental risk factors exist for AMD, such as ultraviolet exposure and smoking, and though there is some genetic contribution and AMD may run in families, the bulk of cases are sporadic.[1] Macular dystrophies, on the other hand, are largely genetic.[1] There can be variable expression and penetrance of macular dystrophies, but remember, relatively symmetric retinal findings suggest heredity, and by-and-large a family history will likely be able to be elicited in macular dystrophies. Referral for genetic testing may be quite helpful for patients with macular dystrophy, certainly for diagnosis, but likely also for the purposes of disease prognosis, and genetic counseling.

Types of Dystrophies

Malattia leventinese (ML) (also known as Doyne honeycomb dystrophy or sometimes, non-specifically, as dominant drusen or familial drusen), is characterized by radial drusenoid deposits of varying sizes around the macula and optic nerve that over time can coalesce into large drusenoid plaques.[1,2] These deposits are sub-RPE, as with drusen in AMD, but unlike AMD, the smaller drusen tend to be more elongated and orient radially temporal to the macula, and the larger round drusen are in the perimacula and around the disc. Also unlike drusen in AMD, in some cases the larger drusen in ML tend to hyper-AF (the smaller drusen are iso-AF as generally expected for drusen).[2]

Case 3. Adult-onset Vitelliform Dystrophy





At left, OCT in an AFVM patient showing two lesions with vitelliform material anterior to the RPE, the larger (more central) of the two with an underlying lucent space. Above, FAF of the same patient demonstrating the two hyper-AF vitelliform lesions.

ML is an autosomal-dominant disease that starts typically in the third to fourth decade, with more rapid progression in the mid-40s; however, there is wide variability in both onset and severity of disease.[1,2] Symptoms may include loss of central acuity, central metamorphopsia or scotoma, photosensitivity or color vision decline. These changes occur due to pigmentary and atrophic changes associated with confluent drusen, which can then lead to the development of CNV.[1,2]

Fundus flavimaculatus (a variation of Stargardt's disease), an autosomal-recessive dystrophy, usually develops before the third decade of life (Stargardt's), but rarely can manifest in later years after age 50, fundus flavimaculatus, somewhat mimicking AMD.[1,8-10] It shows yellowish flecks of lipofuscin accumulation on clinical exam, similar to drusen but more irregularly shaped and are at the level of the RPE.[8,10] The flecks may vary in size and shape and often are fish-shaped, and generally extend beyond and even may spare the central macula.[1,8,11] These flecks intensely hyper-AF on FAF, and may have a surrounding hypo-AF ring.[1] GA may develop, but CNV is rare; however, CNV in fundus flavimaculatus carries a poor prognosis.[11] The juvenile form may begin with symptoms of acuity decline or a reduced sense of color discrimination that seem out of proportion to the clinical exam, where the patient may initially only have a blunted foveal light reflex.[8] The juvenile form becomes more visually devastating, while the adult-onset form usually has more widespread flecks but more slowly progressive disease.[8,9]

Pattern dystrophies include butterfly-shaped pattern dystrophy, adult-onset foveomacular vitelliform dystrophy (AFVM) and others.[12] The pattern dystrophies are generally symmetric and include lipofuscin deposits or various patterns of pigmentary changes or atrophy. However, pattern dystrophies can vary substantially within families and even within one patient from eye to eye, and can even shift from one pattern to another over time.[1,12] These generally develop mid-life with mild symptoms early on.[1,12]

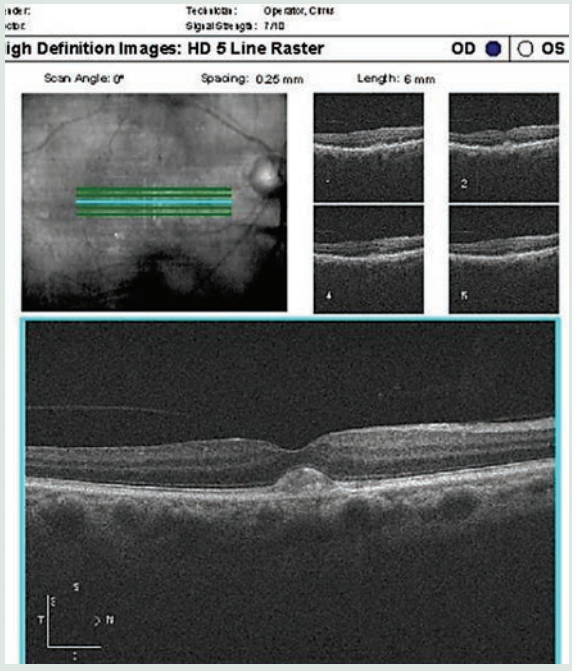
Butterfly-shaped pattern dystrophy is a progressive disorder that appears in earlier mid-life with central RPE pigmentary changes and lipofuscin accumulation, often surrounded by adjacent atrophic changes, usually in a patchy radial or butterfly-wing pattern; these RPE changes and atrophy lead to photoreceptor loss with time.[1,12,13] Patients are usually asymptomatic at diagnosis and usually progress slowly to mild or moderate acuity loss (unless GA or CNV ensue), while maintaining normal color vision and dark adaptation.[12,13]

AFVM is classified as a pattern dystrophy, but predominantly manifests early in the disease with vitelliform (yellow egg yolk-like) rather than pigmentary changes. The foveal vitelliform lesion is made of hyperreflective lipofuscin material and other debris between the retina and RPE.[12,14] These vitelliform lesions are generally solitary but may be multifocal, are usually symmetric bilaterally and intensely hyper-AF on FAF.[1,3,11] Symptoms with AFVM generally develop mid-life and are often reported as only mild visual acuity changes (*Cases 3 and 4*).[1]

Similarly, ***Best vitelliform macular dystrophy (BVMD)*** begins with only mild foveal pigmentary changes, then evolves to also show a foveal vitelliform lesion that becomes quite large (at least one disc diameter).[9] With time, the vitelliform material begins to clump, settle inferiorly and reabsorb, and later leads to chorioretinal atrophy; this evolution in BVMD is not as typical or as dramatic as is seen in AFVM.[9,11] However, the vitelliform material can still reabsorb

as RPE atrophy develops in AFVM, as well, which can lead to a lucid or optically clear space on OCT within or under the vitelliform lesion.[12] In both BVMD and AFVM, the vitelliform lesion develops between the RPE and retina, which is a helpful differentiator from drusen on OCT and shows intense hyper-AF on FAF, whereas associated atrophy would show hypo-AF.[15] CNV is possible with AFVM and BVMD, particularly during the reabsorption phase.[14] BVMD is autosomal-dominant, but with variable penetrance and expression; central vision loss ensues typically before age 40, but ranges widely.[1,9] Interestingly, BVMD is often associated with hyperopia, which might be a helpful clinical consideration.[1]

Case 4. Vitelliform Lesion



A typical, solitary vitelliform lesion in a patient with AFVM. Notice the vitelliform material is anterior to the RPE.

Central areolar choroidal dystrophy (CACD) is another autosomal dominant retinal dystrophy.[1,9] Visual symptoms—due to central pigmentary changes then eventual atrophy—usually start in the fourth to fifth decades, but up to one-third can begin later in life (>55 years), mimicking AMD.[1] CACD may start with subtle pigmentary changes, but the patches of RPE, choriocapillaris and outer retinal atrophy that develop are very sharply demarcated ovals, which expand to coalesce with one another; unlike AMD, CACD rarely has associated drusen.[1,12] Patients may experience photophobia along with visual decline.[1]

Cone dystrophy is a disease of progressive cone dysfunction, which may be inheritable or sporatic.[11,16] Signs and symptoms associated with cone dystrophy can develop at any age, and though it is less common to first manifest later in life (>60 years), late-onset cone dystrophy does occur and is often initially associated with central or paracentral vision loss, color vision dysfunction or photophobia without any clear funduscopic findings to substantiate the patient's symptoms; this can make the initial clinical diagnosis challenging.[16] Later in the disease, cone dystrophy may present with a subtle foveal granularity, pigmentary changes, a bull's-eye appearance, or frank atrophy.[1,11,16] Unlike AMD, patients with late-onset cone dystrophy may show peripheral bone-spicule pigmentary changes, particularly with cone-rod dystrophy, and rarely, temporal optic disc pallor.[1,11] When retinal findings appear normal or nonspecific, ancillary testing can assist: early atrophic changes may be detectable on FAF, and OCT will show outer retinal thinning with loss of the ellipsoid line due to photoreceptor atrophy, and loss of the interdigitation zone between the photoreceptors and RPE complex.[1,17] ERG shows a significant reduction or even absence in photopic response and likely a reduced scotopic response, as well, as many with cone dystrophy also develop rod dysfunction with time, referred to as cone-rod dystrophy.[11] This is associated with nyctalopia due to rod dysfunction. ERG changes are evident before symptoms and signs develop, so can be tremendously helpful in these patients.[9,16]

Many macular dystrophies share features with AMD, such as drusen or drusenoid deposits, pigmentary changes or atrophy.

When patients present with decline in acuity or color vision, or if what looks like AMD doesn't fit the bill, be sure to further investigate. Color vision testing is simple, yet quite helpful, particularly with cone dystrophy. If a patient reports qualitatively reduced visual function, even if not measurable in office or if the fundus looks normal, get photos with FAF. Central visual field testing may also help lead to early detection of macular disease. OCT can offer substantial additional structural information to differentiate AMD from mimickers. And, as always: when it doubt, refer the patient to a medical retina or retinal dystrophy specialist; use their expertise to the benefit of your patients. ■

Dr. Weidmayer practices at the VA Ann Arbor Healthcare System in Ann Arbor, MI. She is also a clinical instructor for the University of Michigan Department of Ophthalmology and Visual Sciences.

1. Saksens NT, Fleckenstein M, Schmitz-Valckenberg S, et al. Macular dystrophies mimicking age-related macular degeneration. Progress in Retinal and Eye Research. 2014;39:23-57.
2. Querques G, Guigui B, Leveziel N, et al. Multimodal morphological and functional characterization of Malattia Leventinese. Graefes Arch Clin Exp Ophthalmol. 2013;251:705-14.
3. Lima LH, Laud K, Freund KB, et al. Acquired vitelliform lesion associated with large drusen. Retina. 2012;32:647-561.
4. Ferris FL III, Wilkinson CP, Bird A, et al. Clinical classification of age-related macular degeneration. Ophthalmol. 2013;120:844-851.
5. Datta S, Cano M, Ebrahimi K, et al. The impact of oxidative stress and inflammation on RPE degeneration in non-neovascular AMD. Progress in Retinal and Eye Research. 2017;1-18.
6. Midena E, Pilotto E. Microperimetry in age:related macular degeneration. Eye. 2017:1-20. [Epub ahead of print]
7. Stuart A. The nuts and bolts of fundus autofluorescence imaging. AAO EyeNet Magazine. Sept 2012;35-37.
8. Sun JP, Chen MS, Jou JR, et al. Clinical characteristics and visual function tests with retinal tomographic correlation in patients with Stargardt's disease in Taiwan. J Formosan Medical Assoc 2013; 112: 79-86.
9. Roosing S, Thiadens A, Hoyng CB, et al. Causes and consequences of inherited cone disorders. Progress in Retinal and Eye Research. 2014;42:1-26.
10. Strauss R, Mu oz B, Wolfson Y, et al. Assessment of estimated retinal atrophy progression in Stargardt macular dystrophy using spectral-domain optical coherence tomography. Br J Ophthalmol. 2016;100:956-62.
11. Kanski J. Fundus dystrophies. In: Clinical Ophthalmology: A Systemic Approach. 6th ed. Oxford: Butterworth Heinemann Elsevier, 1994:663-79.
12. Hannan SR, de Salvo G, Stinghe A, et al. Common spectral domain OCT and electrophysiological findings in different pattern dystrophies. Br J Ophthalmol. 2013;97:605-10.
13. Zhang K, Garibaldi DC, Li Y, et al. Butterfly-shaped pattern dystrophy: a genetic, clinical and histopathological report. Arch Ophthalmol. 2002;120:485-90.
14. Balaratnasingam C, Hoang QV, Inoue M, et al. Clinical characteristics, choroidal neovascularization and predictors of visual outcomes in acquired vitelliform lesions. Am J Ophthalmol. 2016. 172;28-38.
15. Kay CN, Abramoff MD, Mullins RF, et al. Three-dimensional distribution of the vitelliform lesion, photoreceptors, and retinal pigment epithelium in the macula of patients with best vitelliform macular dystrophy. Arch Ophthalmol. 2012;130(3):357-64.
16. Zahlava J, Lestak J, Karel I. Optical coherence tomography in progressive cone dystrophy. Biomed Pap Med Fac Univ Palacky Olomouc Czech Repub 2014;158(4):628-34.
17. Lima L, Sallum J, Spaide R. Outer retina analysis by optical coherence tomography in con-rod dystrophy patients. Retina. 2013;33:1877-80.

OSC QUIZ

You can obtain transcript-quality continuing education credit through the Optometric Study Center. Complete the test form and return it with the $35 fee to: Jobson Medical Information, Dept.: Optometric CE, 440 9th Avenue, 14th Floor, New York, NY 10001. To be eligible, please return the card within one year of publication.

You can also access the test form and submit your answers and payment via credit card at *Review of Optometry* online, www.reviewofoptometry.com/ce.

You must achieve a score of 70 or higher to receive credit. Allow eight to 10 weeks for processing. For each Optometric Study Center course you pass, you earn 2 hours of transcript-quality credit from Pennsylvania College of Optometry and double credit toward the AOA Optometric Recognition Award—Category 1.

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

1. What is the trademark finding in AMD?
a. Drusen.
b. RPE mottling.
c. CNV.
d. GA.

2. Where, anatomically, are drusen found?
a. Under/posterior to Bruch's membrane.
b. Under/posterior to the RPE, between the RPE and Bruch's membrane.
c. Above/anterior to the RPE, between the RPE and the neurosensory retina (subretinal space).
d. Within the outer retina.

3. Which of the following findings alone would qualify as intermediate stage AMD?
a. Small drusen (<63µm).
b. Medium drusen (between 63µm and 125µm).
c. Large drusen (>125µm).
d. GA.

4. GA ultimately shows atrophy of all of the following layers except:
a. Choriocapillaris.
b. RPE.
c. Outer retina.
d. Inner retina.

5. What typically shows hyper-autofluorescence?
a. Drusen.
b. RPE atrophy.
c. Lipofuscin.
d. CNV.

6. What typically show hypo-autofluorescence?
a. Drusen.
b. RPE atrophy.
c. Lipofuscin.
d. CNV.

7. Where, anatomically, are lipofuscin found?
a. Under/posterior to Bruch's membrane.
b. Under/posterior to the RPE, between the RPE and Bruch's membrane.
c. Above/anterior to the RPE, between the RPE and the neurosensory retina (subretinal space).
d. Within the outer retina.

8. In many cases of macular dystrophies, there will be:
a. A positive family history.
b. Relative symmetry between right and left eyes.
c. Helpful genetic testing results.
d. All of the above.

9. Malattia leventinese (ML) is different from AMD in that:
a. ML does not show drusen.
b. ML cannot develop GA or CNV.
c. ML typically shows an earlier onset.
d. ML only shows small drusen.

10. The yellow flecks in Stargardt's and fundus flavimaculatus:
a. Intensely hyper-AF on FAF.
b. Are iso-AF on FAF.
c. Are hypo-AF on FAF.
d. Disappear on FAF.

11. The yellow flecks in Stargardt's and fundus flavimaculatus:
a. Are all of intermediate size.
b. Are all round in shape.
c. Accumulate under/posterior to the RPE.
d. Are irregularly shaped.

12. Which is true of pattern dystrophies:
a. They are usually asymmetric between right and left eyes.
b. They can vary substantially within families.
c. They cannot change pattern over time.
d. They are always in a butterfly pattern.

13. The vitelliform lesion in AFVM develops where?
a. Under/posterior to Bruch's membrane.
b. Under/posterior to the RPE, between the RPE and Bruch's membrane.
c. Above/anterior to the RPE, between the RPE and the neurosensory retina (subretinal space).
d. Within the outer retina.

14. The dramatic evolution of vitelliform development and subsequent reabsorption is seen in:
a. Stargardt's disease.
b. BVMD.
c. AFVM.
d. ML.

OSC QUIZ

15. CACD is similar to AMD in that:
a. It typically has associated drusen.
b. Choriocapillaris and outer retinal atrophy develop.
c. There are usually multiple oval patches of GA.
d. There is usually a positive family history.

16. Cone dystrophy differs from AMD in that:
a. Onset is typically earlier.
b. There are no drusen.
c. There may be peripheral bone-spicule pigmentary changes.
d. All of the above.

17. In cone dystrophy, the ERG shows:
a. Normal photopic response.
b. Decreased or absent photopic response.
c. Normal photopic and scotopic responses.
d. Normal photopic but decreased scotopic responses.

18. In cone dystrophy, the FAF usually shows:
a. Macular hypo-AF consistent with atrophic changes.
b. Macular hyper-AF consistent with lipofuscin.
c. Macular iso-AF consistent with a normal macula.
d. Macular hyper-AF consistent with cone loss.

19. In what macular dystrophy would you most expect frankly abnormal color vision testing?
a. BVMD.
b. CACD.
c. ML.
d. Cone dystrophy.

20. Which of the following referrals would be least helpful to patients with a macular dystrophy?
a. Genetic testing and counseling.
b. Retinal dystrophy specialist.
c. Low vision specialist.
d. Contact lens specialist.

TAKE THE TEST ONLINE TODAY!
www.reviewofoptometry.com/continuing_education/

Examination Answer Sheet

AMD Mimickers: When To Suspect Macular Dystrophy

Valid for credit through June 15, 2020

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

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

Mail to: Jobson Medical Information, Dept.: Optometric CE, 440 9th Avenue, 14th Floor, New York, NY 10001.

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

Credit: This course is COPE approved for 2 hours of CE credit. Course ID is **53907-PS**.

Sponsorship: This course is joint-sponsored by the Pennsylvania College of Optometry.

Processing: There is an eight- to 10-week processing time for this exam.

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 cause, and significance of, drusen formation. ① ② ③ ④ ⑤
22. Become familiar with the various types of macular dystrophies and their presentations. ① ② ③ ④ ⑤
23. Better understand which retinal layers are affected by the various maculopathies. ① ② ③ ④ ⑤
24. Better read OCT, FAF, AF, ERG and other imaging devices associated with macular health. ① ② ③ ④ ⑤
25. Better understand which ancillary studies should be used to diagnose particular dystrophies. ① ② ③ ④ ⑤
26. Better understand the functional symptoms that indicate the presecence of macular dystrophy. ① ② ③ ④ ⑤

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

27. The content was evidence-based. ① ② ③ ④ ⑤
28. The content was balanced and free of bias. ① ② ③ ④ ⑤
29. The presentation was clear and effective. ① ② ③ ④ ⑤
30. Additional comments on this course:

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

First Name
Last Name
E-Mail

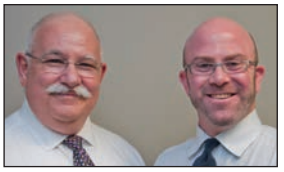
The following is your: ☐ Home Address ☐ Business Address

Business Name
Address
City State
ZIP
Telephone # - -
Fax # - -

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 114847 RO-OSC-0617



Putting Pen to Paper

You don't always need expensive proprietary systems to address your patient's aniseikonia. **By Marc B. Taub, OD, MS, and Paul Harris, OD**

Early in our optometric education, we all learned about aniseikonia and the equations for modifying ophthalmic lenses to compensate for perceived size differences. As full-fledged optometrists, we even think we know when it becomes clinically relevant. In school, we were taught how to differentiate between cases that could benefit from contact lenses vs. glasses based on keratometry readings and axial length measurements; we may have been shown a space eikonometer (though few of us have access to one in practice).

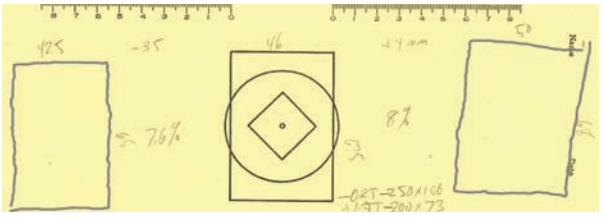


Fig. 1. A patient's cheiroscopic tracings show a significant size difference.

However, some literature calls into question many of the assumptions about aniseikonia we were taught as students.[1] The research, as far back as 1999, demonstrates the flaws in assuming keratometry reading and axial length are key factors. Often, distortions in spatial perception trump the more purely optical considerations and must be tested directly and responded to, rather than being driven by the prescription alone. One of our recent cases reminded us of the importance of using those fundamental equations, not necessarily fancy technology, to solve the patient's problems.

The Case

J.F., a current student at Southern College of Optometry, was undergoing vision therapy, but hit a wall as we attempted to build binocularity. Despite admirable compliance, he failed to make enough progress and saw little relief of his symptoms, including intermittent double vision, which he often closed an eye to avoid. He tilted his head to one side slightly, but was aware of it whenever he was fusing. He also experienced frequent headaches and asthenopia whenever he had to read for more than five to 10 minutes.

With the following prescription he could see 20/20+ with each eye:

- OD -0.25 -2.50 x 108
- OS +1.50 -2.00 x 75

The difference in one meridian is about 1.75D and in the other meridian about 2.25D—this is hardly an amount that shouts aniseikonia.

Student interns and residents repeatedly noted the presence of a large exophoria, which J.F. could converge to overcome, but fusion was just beyond reach. J.F. was experiencing significant near point symptoms, so we asked him to perform a cheiroscopic tracing. Using a stereoscope, we asked him to trace a picture presented in front of one occluded eye, using the other eye to help trace. Though the pencil point and picture are in different physical places, the patient perceives the pencil as tracing directly over the picture. We have the patient first perform this with the picture in front of the right eye, with the pencil tracing on the left of the recording paper. This is then repeated with the left eye with everything reversed.

When J.F. performed the tracings, the figures were separated more than expected, in correlation with exophoria *(Figure 1)*. The left tracing was lower than the central reference picture and the right one was shifted upward. This was consistent with the measured right hyperphoria. Another revelation: The left picture was about 7.6% narrower than the reference picture and the right tracing was about 8% wider—a possible explanation for J.F.'s fusion issues. We also noted

$$SM = \underbrace{\left(\frac{1}{1-\frac{t}{n}F_1}\right)}_{\text{shape}}\underbrace{\left(\frac{1}{1-hF_V}\right)}_{\text{power}}$$

Fig. 2. A colleague shared the spectacle magnification equation from his lecture notes. F_1 is the front surface power of the lens, F_V is the back vertex power, *t* is the lens thickness, *n* is the index of refraction and *h* is the distance from the lens' back vertex to the entrance pupil.

that the right tracing looks twisted slightly in a clockwise direction but the floor of the tracing is nearly flat; the left tracing was not torqued at all. We tested for a cyclophoria using a subjective Double Maddox Rod test. It showed a three- to four-degree cyclophoria at distance. Generally, this is not perceived as a problem for the binocular system to overcome. However, a 7.6% to 8% perceptual size difference is often just at the point that it does cause a problem. We also noted the vertical size differences were not a big as the horizontal differences.

Closing the Distance

The first approach was to get J.F. fit in contact lenses. He mentioned that he had been in and out of lenses and had a long-term problem with dry eye, which precludes his ability to wear contact lenses for any significant amount of time. Instead, it was time to break out the equations to compute a new set of spectacles for J.F. *(Figure 2)*.

To make things a little more convenient, we used a calculation tool at opticampus.com (64.50.176.246/tools/magnification.php). Putting this formula into practice prompted a phone call to our lab to clarify a few parameters:

- What is a practical range of front base curves to which we can have lenses ground?
- What is the practical thickest lens you could accommodate?

The practical base curve range was from +0.50 to +8.00, and the thickest lens we'd explore would have a 6mm center thickness.

When we put J.F.'s current glasses Rx into the equation, we saw they were producing about 3% of the 7.6% to 8% size difference noted by the tracing. His original glasses were done on a +3.50 front surface base curve.

We then explored a range of lenses and materials to come up with the following Rx:

- OD -0.25 -2.50 x 108 with a +8.00 base curve and 6.0mm center thickness.
- OS +1.50 -2.00 x 75 with a +0.50 base curve and a 2.2mm center thickness.

This achieved a 2.8% increase in the size of the right eye image and a 0.5% decrease in size in the left eye for a net 3.3% decrease in size difference *(Figures 3 and 4)*. We hoped it would be enough.

The Difference is Clear

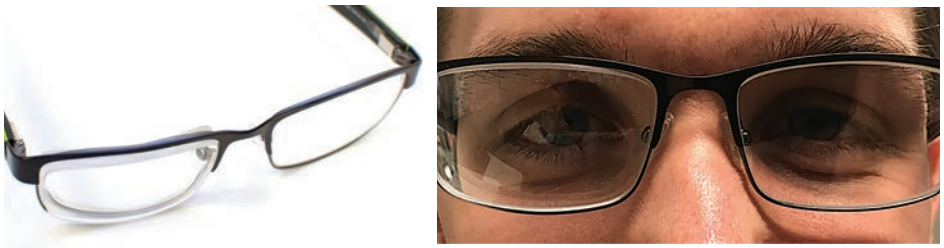
After wearing the new glasses for about 25 minutes, J.F. completed a second cheiroscopic tracing *(Figure 5)*. The horizontal size in the right tracing matched the reference figure. The left tracing was less stable, as the top was wider and the bottom was narrower than the reference. Also, the figures are a bit closer to the center and the vertical deviation is somewhat reduced *(Figure 5)*.

Did the new prescription make a difference? J.F. wrote the next morning, "Usually, I end up closing my left eye reading my scriptures in the morning. I was able to comfortably read with both eyes open with the new glasses on. Woohoo!"

Oh, and these lenses only cost $38.85.

As J.F. reminded us, we can diagnose and treat aniseikonia without the need for a space eikonometer; treatment need not cost a fortune, nor do we need to fall prey to expensive proprietary systems to address the clinical problems. Sometimes, in ophthalmic prescribing, size differences do matter. ■

1. Romano PE, Von Noorden GL. Knapp's law and unilateral axial high myopia. Bin Vis Strab Quart. 1999;14:215-22.



Figs. 3 and 4. At left, J.F.'s lenses had significantly different thickness, yet they made a huge difference in his aniseikonia. At right, note the amount of the shift inwards of the right side of J.F.'s head in the temporal portion of his glasses and the lack of shift in the left side of his face.

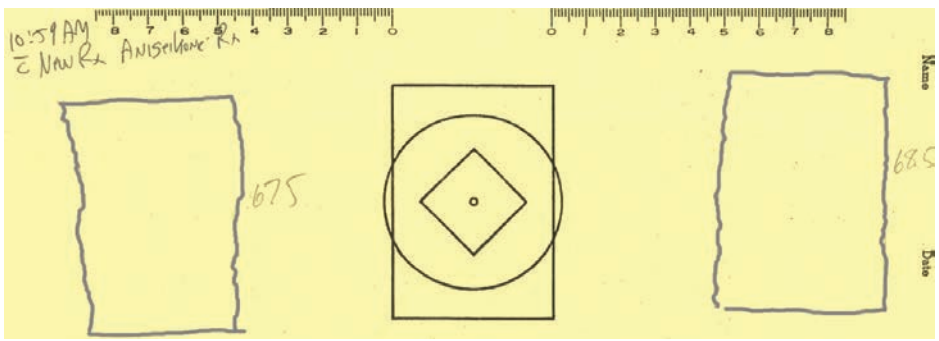


Fig. 5. J.F.'s second tracing showed significantly less difference in size.

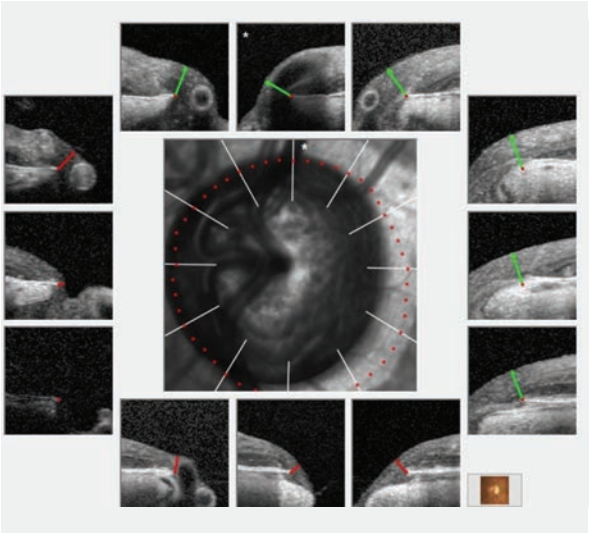
Through Thick and Thin

Don't be afraid to be the last doctor the patient sees before they go blind.

By James L. Fanelli, OD

A 76-year-old white female presented in late April for optical coherence tomography (OCT) and Heidelberg retinal tomography (HRT 3) images of her optic nerves, related to her long-standing glaucoma.

Her previous visit was five months earlier, at which time visual fields, intraocular pressure (IOP) readings and a slit lamp evaluation of her optic nerves were all stable.



The most recent images of this patient's left eye demonstrate significant cupping and a markedly reduced ganglion cell layer overlying Bruch's membrane opening, consistent with advanced glaucomatous disease.

History

Her medications included aspirin, Lipitor (atorvastatin, Pfizer), Lopressor (metoprolol, Novartis), Paxil (paroxetine, GSK), K+ supplementation, Nasonex (mometasone, Merck) and Claritin (loratadine, Bayer). She has allergies to multiple medications, including all penicillins and several antihypercholesterolemia medications. She has an extensive history of cardiovascular disease with coronary artery bypass grafting x4 and subsequent coronary artery stenting x7 over the past 10 years.

Her most recent cardiovascular event, in 2015, required surgical intervention. Three months prior to that, she underwent an appendectomy and recovered well.

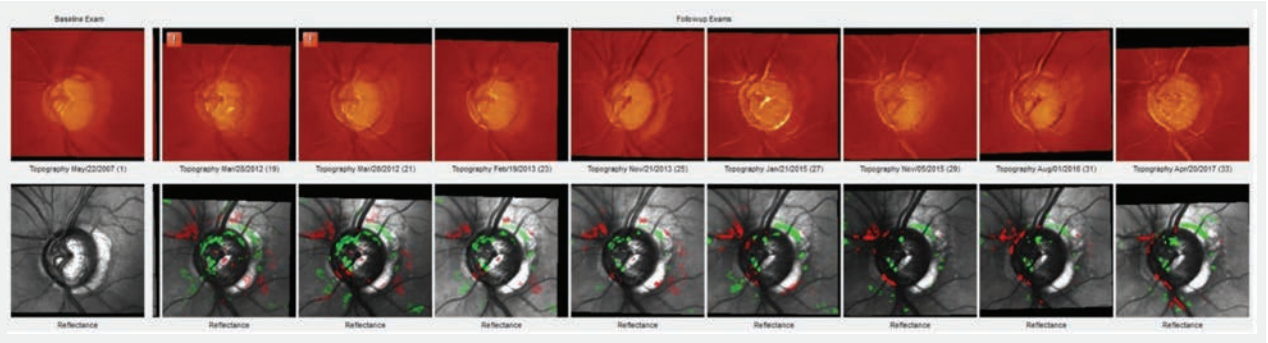
She presented as a new patient in 2006, carrying with her a diagnosis of open-angle glaucoma, at which time she was medicated with Xalatan (latanaprost, Pfizer) HS OU and 0.5% timolol BID OU. Her central corneal thicknesses were 517µm OD and 514µm OS. In the first few visits in late 2006 and early 2007, she was deemed to be relatively stable insofar as her glaucoma was concerned, and her glaucoma state was graded as severe at that time, with optic nerves appearing similarly to the current presentation.

Her current glaucoma medications include Azopt (brinzolamide, Novartis) BID OU and Lumigan (bimatoprost, Allergan) HS OU. Best-corrected visual acuities were 20/20 OD and 20/25- OS through hyperopic astigmatic and presbyopic correction. She was pseudophakic and underwent cataract surgery in 2009.

Testing

The slit lamp exam of her anterior segments was essentially unremarkable. There was mild corneal arcus noted in both eyes, along with minimal endothelial cell loss. The anterior chamber angles were open, and the chambers were deep and quiet with no cells or flare. Applanation tensions were 13mm Hg OD and 14mm Hg OS, consistent with previous visits. Pupils were equal, round, responsive to light and accommodation with no afferent pupil defect. Her intraocular lenses were in their capsular bags and both posterior capsules had been opened following implantation. Through dilated pupils, she had long-standing bilateral peripheral vascular diseases. Her cup-to-disc ratios were 0.75 x 0.85 OD and 0.85 x 0.95 OS, which were consistent with previous evaluations.

The remaining neuroretinal rims were plush and well perfused, but there was significant thinning of the rim temporally, moreso in the left eye than the right. Both maculae were characterized by fine RPE mottling and a few drusen, with no



The patient's HRT 3 scans of her left eye over the past 10 years showing marked stability of her neuroretinal rims in the presence of advanced glaucoma.

evidence of subretinal neovascular membrane in both eyes.

The patient's retinal vasculature was characterized by grade 2 arteriolarsclerotic retinopathy and minimal hypertensive retinopathy in both eyes. Her peripheral retinal evaluations were unremarkable.

Scans

HRT 3 and OCT imaging of both optic nerves was obtained, with good image quality. Both the HRT 3 scans, as well as the OCTs, demonstrated no progression of the glaucomatous damage in the neuroretinal rims, the perioptic retinal nerve fiber layer and the macular ganglion cell layers.

She has remained stable for 10 years—save for the cataracts, and a gradual creep in her IOP which required bilateral SLTs and, ultimately, a change in her topical glaucoma medications.

Discussion

This long-term patient initially presented with advanced glaucoma, and, over the past 10 years, developed expected changes, such as gradual worsening.

When establishing care with new patients, it's important to evaluate their overall stability within the first couple of visits and begin to develop a doctor/patient relationship. Of course, obtaining old records at that time is helpful in determining stability, but as you manage these patients over the course of their lives, you will ultimately be repeating all of those same studies. The data gathered over decades of care becomes the patient's own reference data base, upon which future stability is continually evaluated. IOP ranges, structural details of the optic nerve (as obtained by CLSO, OCT and stereo photos), functional details of the optic nerve (as obtained by threshold visual fields), compliance issues (if any), medication tolerability and ancillary ophthalmic findings are all elucidated over time.

Given a relatively compliant patient, it becomes easy to see trends over time. We know that ultimately, glaucoma patients are going to follow one of two paths: they will either remain stable or get worse. Our primary responsibility to the patient is to determine whether they are indeed stable. If so, then maintaining the status quo is prudent. If not, a course change is required. As captain of that ship, determining when to change course and in what direction to change course is critical in allowing the patient to continue seeing well enough to live out the rest of their lives. Clinicians who manage glaucoma must be comfortable in being the "captain" of that ship. Certainly, there will be instances where consultation will be necessary but ultimately if you are planning on managing glaucoma, you need to understand, accept and be comfortable with managing glaucoma patients of all different types—even those with advanced disease.

How you handle the doctor-patient relationship can either soothe or discomfort the patient. Our patient was a new patient with advanced disease who has chosen me to be her provider of glaucoma care. Eleven years have lapsed since we first met. She has undergone selective laser trabeculoplasty and modification of therapy because of some instability in her disease, yet we were able to wrest control of the situation. Of course, the other way that this might go is that she progresses, and loses vision.

With advanced glaucoma patients, the unfortunate reality is they may lose their vision, but with the right kind of care, the optometrist can be the hand the patient holds across that threshold. ■

He Came By it Naturally

Can these images help explain this patient's blurry vision?

By Celina Ann Diego, OD, and Mark T. Dunbar, OD

An 11-year-old male with complaints of gradually blurring vision in both eyes was referred to our office after a year of experiencing symptoms. The accompanying parent noted that the child had a normal birth, growth and development. His pertinent medical history was remarkable for asthma. Family history was remarkable for unknown vision loss suffered by his maternal grandfather and a maternal cousin. Otherwise, the boy's ocular history was noncontributory.

Examination

Upon examination, visual acuities with his current spectacle correction were 20/70 OD and 20/100 OS with no improvement with pinhole OU. Pupils were equally round and reactive to light. There was no afferent defect. Confrontation visual fields were full to careful finger counting in both eyes. Extraocular motility testing was normal. Anterior segments of both the right and left eye were unremarkable. Applanation tonometry measured 19mm Hg OD and 17mm Hg OS.

Fundus examination of the right eye revealed normal vitreous and optic nerve, with tortuous vessels, and a poor foveal light reflex. Other changes were noted in fundus images (*Figure 1*). Similar and more extensive finding were seen in the left eye (*Figure 2*). A fluorescein angiogram (FA) and OCT were obtained and available for review (*Figures 3-6*).

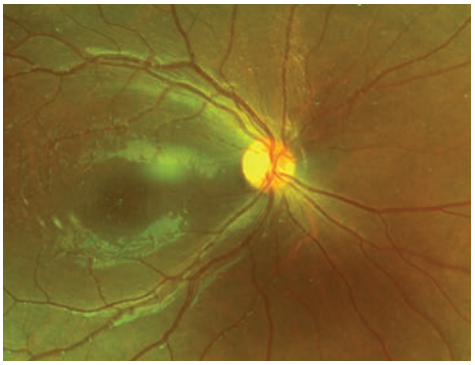


Fig. 1 At left, this fundus photograph shows an 11-year-old patient's right eye.

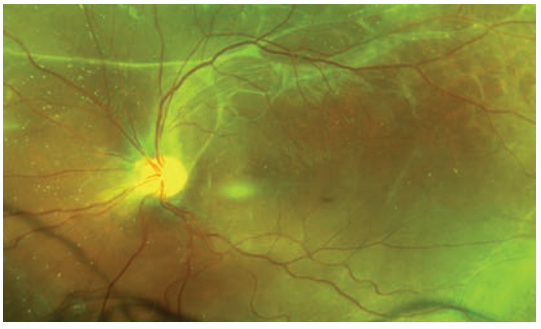


Fig. 2. Pertinent findings were also discovered in his left eye, shown below. Look carefully at the maculae of both eyes and the peripheral changes that are seen in the left.

Take the Retina Quiz

1. What is the most likely diagnosis?
a. Retinitis pigmentosa.
b. Juvenile X-linked retinoschisis.
c. Cystoid macular edema.
d. Goldmann-Favre vitreoretinal degeneration.

2. What is the appropriate interpretation of the OCT imaging of the right eye as seen in *Figure 5*?
a. IS/OS disruption.
b. Epiretinal membrane (ERM).
c. Cystic changes.
d. Vitreomacular traction.

3. What is the inheritance pattern of this condition?
a. Autosomal dominant.
b. Autosomal recessive.
c. X-linked dominant.
d. X-linked recessive.

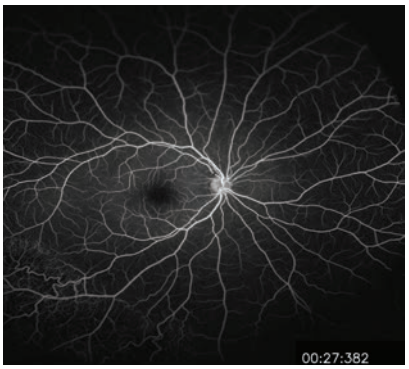
4. Aside from surgical intervention, what else is warranted in the management of this patient?
a. Genetic testing of patient and family members.
b. Yearly observation.
c. Anti-VEGF injection.
d. Vitamin A supplementation.

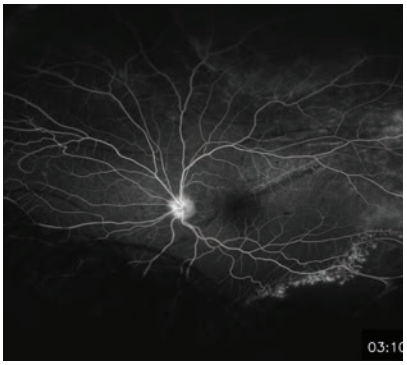
Diagnosis

The macula of the right eye showed cystic spaces within the sensory retina. This was confirmed using OCT, where macular cysts can be seen (*Figure 5*). In the left eye, there was an obvious macular detachment, which was also confirmed with OCT imaging (*Figure 6*). The right eye showed prominent white without pressure and fibrotic bands extending into

the vitreous. A peripheral retinoschisis was present inferotemporally. The fundus exam of the left eye revealed pigmented cells in the vitreous, retinal folds and fibrotic bands extending superiorly and superonasally. The peripheral retina was superiorly detached.

The FA revealed vascular abnormalities in the patient's inferotemporal peripheral retina in the right eye and inferonasal peripheral retina in left eye (*Figures 3 and 4*). After careful review of the patient's familial ocular history, ancillary testing and clinical presentation, the young boy was diagnosed with juvenile X-linked retinoschisis (JXRS).





Figs. 3 and 4. These midphase FA images show the patient's right (at left) and left eyes. Do they reveal any pathology?

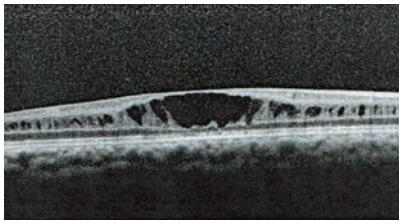
Discussion

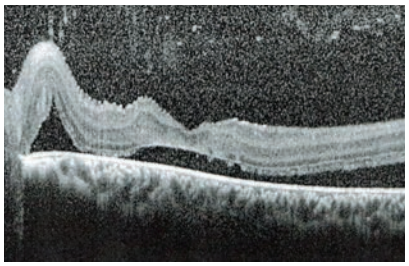
This disease typically affects young males in their first decade of life. Prevalence of JXRS has been estimated to be anywhere from 1/7000 to 1/2800.[2] It is passed on in an X-linked recessive pattern. In other words, only males are affected by the disease while female counterparts are asymptomatic carriers.[3] The only known disease gene associated with JXRS to date is Retinoschisin 1 (RS1).[2] Generally, entering visual acuities are usually in the range of 20/40 to 20/60 and continue to deteriorate into the third decade of life.[2] However, different strands of JXRS exist with varying levels of severity.

Those affected by JXRS will have characteristic cystic changes in the fovea. A stellate pattern of cystic spaces surrounding the foveal area is considered pathognomonic for this condition. This change is secondary to the schisis of the inner retinal region or the nerve fiber layer.[2] With time, the cystic spaces begin to coalesce, giving the appearance of foveal atrophy with associated pigmentary changes. Also, peripheral schisis is common in the inferotemporal area. With the coalescence of peripheral schisis areas, vitreous hemorrhages, retinal holes and retinal detachments can occur. These are often secondary to traction in the retinal bands or areas of non-schitic retina.[2] If the schisis becomes severe and the vitreous condenses, leukocoria may manifest.[1,2] Nonspecific pigmentary changes occur later in the disease as the outer retinal regions become affected by chronic schisis.[2]

Ancillary testing is extremely helpful and is performed to assist in distinguishing the diagnostic features of JXRS. Scotopic ERG demonstrates an electronegative response with deep negative a-wave and a b-wave that is substantially attenuated and does not return to baseline.[1,2] Electronegative ERG has a sensitivity of 100%, as all affected males produce the same results despite age or stage of disease.[1] However, there are no reliable or identifying ERG findings in female carriers.[1] Electro-oculogram testing is initially normal, but with disease progression affecting the outer retinal layers and RPE, results become abnormal.[2] Color vision often reveals proton or tritan deficits.[2] Visual field testing can elicit a relative central scotoma, which is related to the foveal schisis, as well as absolute scotomas in regions that correspond to the areas of peripheral retinal schisis (i.e., superonasal scotomas in the presence of inferotemporal schisis).[2] Lastly, FA often shows no signs of leakage or staining within the fovea despite apparent cystic changes. In late-stage JXRS, a window defect appears due to the pigment in the fovea area, and capillary nonperfusion in schisis areas of peripheral retina, which may also have associated pigmentary changes.[2] As seen in the FA results of our patient, only vascular telangiectatic abnormalities, retinal schisis and retinal detachment were noted; leaking of the vessels was not an issue.

Currently, no treatment exists for either the retinal degeneration or the schisis that occurs from JXRS. However, should a patient experience serious complications from the condition such as a vitreous hemorrhage or retinal detachment, surgical intervention may be warranted.[5] As primary care physicians, it is in the best interest of the patient that we best correct vision with polycarbonate lenses





Figs. 5 and 6. These SD-OCT slices show the macula in the right (above) and left eyes. How do you explain these findings?

and employ the use low vision aids.[5]

Lately, researchers have studied new treatments for the complications of JXRS. Most notably, one study shows dorzolamide 2% decreases foveal thickness as well as the appearance of the cystic spaces.[6] Gene replacement therapy has also been considered as another treatment option. This therapy would aim to replace RS1 and research shows it is successful in knock-out mouse models.[5]

Regardless, it is ideal for the family of a diagnosed JXRS patient to be genetically tested for RS1 to determine the likelihood of the disease affecting future children and to determine which particular strand of JXRS the patient suffers from, as different strains have varying levels of severity.[5]

In the case of our patient, he suffered from inferior schisis of the right eye and retinal detachment of the left eye. He underwent a pars plana vitrectomy with scleral buckle in the left eye and experienced no complications. At his most recent visit, the vision in his left eye was best corrected to 20/100. The plan is to proceed with a scleral buckle in the right eye once his left eye is deemed stable. In the meantime, he is to continue using polycarbonate spectacle lenses for correction and protection. ■

Dr. Diego is a resident at Bascom Palmer in Miami.

1. Spaide R. Diseases of the Retina and Vitreous. London: W.B. Saunders;1999.
2. Reynolds J, Olitsky S. Pediatric Retina. Berlin, Heidelberg: Springer Berlin Heidelberg;2011.
3. Hollyfield J, Ash J, Grimm C, et al. Retinal Degenerative Diseases. Springer. 2012.
4. Alexander L. Primary care of the posterior segment. New York: McGraw-Hill;2002.
5. Sikkink S, Biswas S, Parry N, et al. X-linked retinoschisis: an update. Journal of Medical Genetics. 2007;44(4):225-32.
6. Apushkin M, Fishman G. Use of dorzolamide for patients with x-linked retinoschisis. Retina. 2006;26(7):741-5.

Tonometry Done Right

new

D-KAT Digital
Keeler quality.

Pulsair Desktop
Smallest footprint and simple to use!

Purchase a Pulsair Desktop by September 30, 2017 and get a $1,326 Instant Rebate!

Intellipuff
The standard for hand held mobility.

Buy Online!
keelerusa.com

Keeler
OPTICS

Keeler Instruments, Inc. • 3222 Phoenixville Pike #50 • Malvern, PA 19355 • Tel: (800) 523-5620 • Fax: (610) 353-7814 • email: keeler@keelerusa.com


A Pop Fly Straight to the Eye

A refresher on hyphema management, just in time for wiffleball season.

By Joseph W. Sowka, OD, and Alan G. Kabat, OD

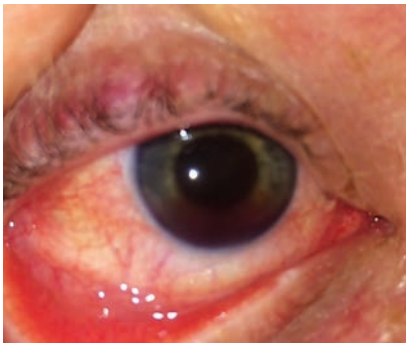
A 55-year-old man presented urgently after being struck in the right eye. Playing wiffleball with his son the day before, the patient took a direct, high-speed hit to his right globe. He noted no pain to the eye initially, but some discomfort to the periorbital region with associated bruising. He did note that his vision was immediately blurry, but waited to seek medical care, expecting that "it would clear up."

Examination

He felt that his vision did improve and he presented with acuity of 20/25 in the eye. His right pupil was mid-dilated and unreactive to light and accommodation. He had moderate conjunctival injection in his right eye and a moderately heavy anterior chamber reaction with both white and red blood cells. He also had a mild accumulated hyphema. Gonioscopy was deferred due to the recent nature of the trauma. His intraocular pressure (IOP) was 15mm Hg in the right eye and 12mm Hg in the left. His crystalline lens was clear and centered and a dilated fundus exam showed no abnormalities.

Diagnosis

He was diagnosed with a traumatic uveitis and hyphema, as well as a tonic pupil due to iris sphincter damage. He was cyclopleged with atropine 1% BID and treated with difluprednate 0.05% (Durezol, Alcon) QID. He was instructed to relax and stay in bed as much as possible. By the next day, his vision improved to 20/20 and his anterior chamber reaction was greatly diminished with near-complete resolution of hyphema. Medications were tapered accordingly over the ensuing week and the patient had a great outcome, though he has a small degree of angle recession and a permanently dilated pupil from the iris sphincter tear.



After his son confused the meaning of "keep your eye on the ball," this dad's traumatic ocular injury developed into uveitis and hyphema.

Discussion

Hyphema is accumulated blood in the anterior chamber (AC).[1-4] The presence of non-layered red blood cells circulating in the anterior chamber is referred to as microhyphema. Hyphema can occur as a result of:

- Blunt or lacerating ocular or adnexal trauma
- Following intraocular surgery
- Secondary to conditions that induce iris neovascularization, such as diabetes
- Venous occlusion or iris melanoma
- Secondary systemic conditions, such as juvenile xanthogranuloma or myotonic dystrophy
- As a complication of keratouveitis (e.g., herpes zoster)
- As a complication of other blood disorders such as leukemia, hemophilia, von Willebrand disease and in association with the use of substances that alter platelet or thrombin function (e.g., ethanol, aspirin, warfarin).[1-3]

Complications of traumatic hyphema include increased IOP with secondary glaucoma, peripheral anterior synechiae, decreased visual acuity and corneal dysfunction (blood being in the AC and corneal blood staining) and rebleeding with secondary hemorrhaging.[1-3]

Patients may present with the classic signs and symptoms of associated uveitis, including conjunctival hyperemia, blurred vision, eye and orbital pain, photophobia, lacrimation and blepharospam as well as blood in the AC.[1-3] Any time IOP is elevated following blunt traumatic ocular injury, hyphema should be suspected whether blood is visible in the anterior chamber or not. The most common concurrent ocular injuries associated with traumatic hyphema is corneal

injury and uveitis; however, adnexa ecchymosis and lacerations of the eyelid are also possible.[4-6]

Hyphemas are classically graded by the amount of visible blood occupying the AC. Less than 33% of the AC is grade 1, 33% to 50% is grade 2 and greater than 50%, but less than 100%, is grade 3. Complete AC filling is grade 4. The term "8-ball hemorrhage" connotes complete filling of the AC with blood, appearing black like a billiard 8-ball.

There are two postulated mechanisms regarding traumatic hyphema formation. Either direct, concussive forces cause mechanical tearing of the fragile vasculature of the iris or angle, or concussive trauma creates rapidly rising intravascular pressure within these vessels, resulting in their rupture.[6] The most common traumatic cause of hyphema is a tear in the anterior face of the ciliary body.

A minor quantity of blood in the AC is not, by itself, necessarily harmful to the ocular environment. However, when quantities are sufficient, macrophages ingest the hemoglobin from the lysed red blood cells. These hemoglobin-laden macrophages obstruct the outflow of aqueous humor by physically blocking access to the drainage area, resulting in IOP rise.[7] This is known as hemolytic glaucoma.[7]

Patients with the sickle trait have a greater risk for elevated IOP.[8] Sickled red blood cells are not as malleable as normal red blood cells. Hyphema involving any sickled cells further impedes the flow of aqueous humor, slowing both aqueous and oxygen transfer. The hypoxic environment encourages red blood cells encoded with the sickle trait to undergo the sickle transformation, which further obstructs aqueous outflow.[8]

Circumstances surrounding the event and current medicines are important pieces of data. Bleeding in the eye warrants concern for systemic blood disorders such as antiphospholipid antibody disease (protein S and protein C), hyperhomocysteinemia, dysfunction of the clotting factors, sickle cell anemia, hemophilia and von Willebrand's disease.[1,2] Testing for sickle cell anemia is also a consideration.

Ocular examination should include evaluation of the adnexa. Imaging should be ordered when appropriate to rule out fracture or entrapment (x-ray, CT scan).

The cornea should also be evaluated for abrasion, laceration or penetrating injury. A ruptured globe usually includes poor visual acuity, ocular hypotony, and shallowing of the AC.[9] The iris should be inspected for iridodialysis and sphincter tears, and the lens for luxation. A dilated fundus exam should be completed to rule out vitreous hemorrhage and retinal tears or detachments. If a clear view of the fundus is obstructed by the hyphema or vitreous hemorrhage, B-scan ultrasonography should be attempted to best assess posterior segment damage.[1,2] Eventually, gonioscopy should be performed to look for angle recession. This is typically performed four to six weeks after the initial trauma.

Treatments

Management of hyphema is directed towards enhancing blood resorption and minimizing complications such as glaucoma and corneal blood-staining. Controversy is ongoing whether these individuals should be managed as in- or outpatients.[10] While some advocate strict bed rest and even hospital admission, there is no evidence that outcomes for such measures are better than quiet ambulation. Most practitioners manage microhyphema and uncomplicated grade 1 and 2 hyphema conservatively without hospitalization. Hospitalization is typically reserved for severe hyphema, sickle cell trait/disease, noncompliant patients, children and those with bleeding disorders. Rest with head elevation allows blood to settle inferiorly and prevents clot formation and blood-staining over the visual axis.[10] Patients should be instructed to wear a protective shield at all times, especially while sleeping, to prevent inadvertent repeat trauma. Aspirin and nonsteroidal anti-inflammatory medications increase the risk of rebleeding and should not be used if at all possible.[10]

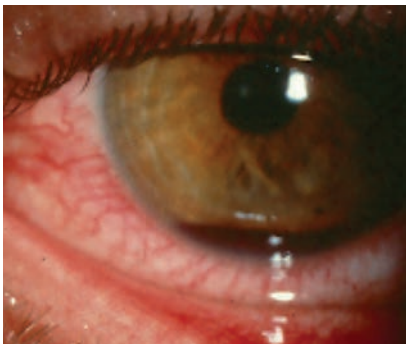
Although studies have not found cycloplegic usage to affect final visual acuity, their use is considered advantageous by reducing the risk of secondary hemorrhage by immobilizing the iris and ciliary body. Also, their use reduces risk of posterior synechiae, increases uveoscleral outflow and enhances patient comfort.[10] Adequate cycloplegia is accomplished using atropine 1%, BID–TID for two weeks, though less frequent dosing may achieve adequate cycloplegia for mild hyphema.

Local inflammation is controlled via topical prednisolone acetate 1%, Q2H-QID or difluprednate 0.05% BID-QID.[10] Additionally, they stabilize the blood-ocular barrier and may decrease the frequency of secondary hemorrhage.[10] However, in a recent study topical steroids failed to demonstrate any benefit over supportive therapy consisting simply of bed rest, head elevation and hydration.[11]

If IOP is elevated above 24mm Hg, it can be reduced with the use

Advertisers Index

Therapeutic Review



Traumatic hyphema patients may suffer from increased IOP, secondary glaucoma, corneal dysfunction and other compounding factors.

of topical aqueous suppressants such as an alpha-2 agonist, beta-blocker or carbonic anhydrase inhibitor in standard dosing. When IOP is exceptionally high (greater than 35mm Hg) requiring acute attention, acetazolamide tablets, 500mg (2 x 250mg) BID can be prescribed along with topical aqueous suppressants. Acetazolamide lowers plasma pH, which promotes sickling of red blood cells; thus, methazolamide is preferable in patients with sickling disorders.[10] Follow up should be set no later than a week for uncomplicated cases and should be set for consecutive days, as necessary, in cases where there are vision-threatening issues.[1,3]

Referral for surgical evacuation is indicated in the following cases:

(1) if there is corneal blood staining;

(2) if the hyphema fails to reduce to less that 50% of AC volume within eight days;

(3) if IOP is greater than 60mm Hg for two days;

(4) if there is an 8-ball hemorrhage;

(5) if the IOP remains greater than 25mm Hg for five or more days (to prevent blood-staining); or

(6) if IOP remains greater than 24mm Hg over 24 hours in a patient with a sickling disorder.[1-3,10]

The most common surgical procedure is limbal paracentesis and blood aspiration. AC washout can be done if the blood has not yet layered.[10]

The use of oral antifibrinolytic medications such as tranexamic acid tablets have demonstrated superior properties for stabilizing bleeding and maintaining clot performance, reducing the risk for rebleeding and injury worsening.[12,13] Topical tranexamic acid is as effective as the oral version in preventing secondary hemorrhage.[13]

Though hyphema is a common entity, there exists no consensus on management. The use of hospitalization, bed rest, head elevation and use of cycloplegic steroid and antifibrinolytic agents must be decided on a case-by-case basis. ■

The authors would like to thank Dr. Lindsay Baker-Howse for her contribution of the case and suggestion of topic.

1. Walton W, Von Hagen S, Grigorian R, et al. Management of traumatic hyphema. Surv Ophthalmol. 2002;47(4):297-334.
2. Recchia FM, Saluja RK, Hammel K, et al. Outpatient management of traumatic microhyphema. Ophthalmology. 2002;109(8):1465-70.
3. Rocha KM, Martins EN, Melo LA Jr, et al. Outpatient management of traumatic hyphema in children: prospective evaluation. J AAPOS. 2004;8(4):357-61.
4. Rocha KM, Martins EN, Melo LA Jr, et al. Outpatient management of traumatic hyphema in children: prospective evaluation. J AAPOS. 2004;8(4):357-61.
5. Mowatt L, Chambers C. Ocular morbidity of traumatic hyphema in a Jamaican hospital. Eur J Ophthalmol. 2010;20(3):584-9.
6. Ashaye AO. Traumatic hyphaema: a report of 472 consecutive cases. BMC Ophthalmol. 2008;8(11):24.
7. Wilensky JT. Blood induced secondary glaucomas. Ann Ophthalmol. 1979;11(11):1659-62.
8. Pandey P, Sung VC. Gonioaspiration for refractory glaucoma secondary to traumatic hyphema in patients with sickle cell trait. Ophthalmic Surg Lasers Imaging. 2010;41(3):386-9.
9. Rao LG, Ninan A, Rao KA. Descriptive study on ocular survival, visual outcome and prognostic factors in open globe injuries. Indian J Ophthalmol. 2010;58(4):321-3.
10. Bansal S, Gunasekeran DV, Ang B, et al. Controversies in the pathophysiology and management of hyphema. Surv Ophthalmol. 2016;61(3):297-308.
11. Türko lu EB, Celik T, Celik E, et al. Is topical corticosteroid necessary in traumatic hyphema? J Fr Ophtalmol. 2014;37(8):613-7.
12. Gharaibeh A, Savage HI, Scherer RW, et al. Medical interventions for traumatic hyphema. Cochrane Database Syst Rev. 2013 Dec 3;(12):CD005431.
13. Jahadi Hosseini SH, Khalili MR, Motallebi M. Comparison between Topical and oral tranexamic acid in management of traumatic hyphema. Iran J Med Sci. 2014;39(2 Suppl):178-83.

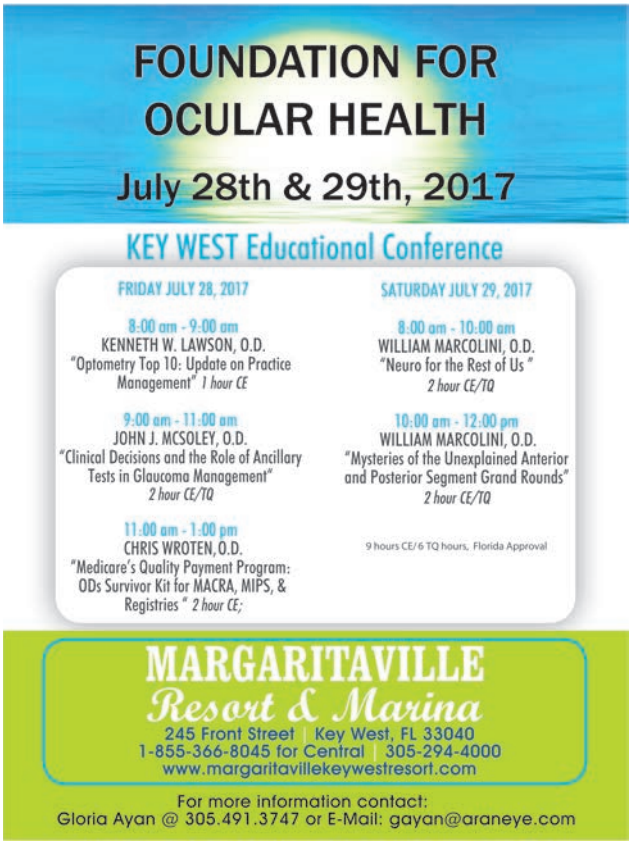
Merchandise Offered

Merchandise Offered

Contact Lenses

FACULTY

Continuing Education



Career Opportunities

OPTOMETRIST OPPORTUNITY
ANCHORAGE, AK.

Anchorage, AK. - Full time Optometrist Opportunity with Alaska's PREMIER Eye Health Care Practice – Alaska Eye Care Centers, APC – voted Best of Alaska since 2007. A privately owned, two location practice with 7 doctors is seeking a new graduate or experienced O.D. for employment opportunity.

Each location has 4-5 full exam rooms and the appropriate equipment- special testing equipment, optometric pre-testers, and full administrative support. The main office offers full lens manufacturing for both locations. Each office has optical dispensaries consisting of over 2,500 frames and is serviced by licensed opticians. In addition, each office has full contact lens services, including licensed CL technicians.

This opening is for our Wasilla location rotating into Anchorage at least once a week. Wasilla is a semi-rural setting approximately 40 miles from Anchorage, Alaska. The population of the surrounding area is about 98,000 and housing and utilities costs are comparable to West Coast levels. Entertainment, fine dining and the arts are easily available in Anchorage, a city of more than 291,000 residents.

Alaska is one of the most beautiful places to live and practice. For further information, please contact:

Alaska Eye Care Centers
1345 W. 9th Avenue, Anchorage, AK. 99501
Contact: Deb Foster, Administrator
deb@alaskaeyecare.com
(907) 272-2557 X 1604
(907) 274-4932 (Fax)
Website: www.alaskaeyecare.com

Career Opportunities

OPTOMETRY ASSOCIATE
Leading to Partnership

Full or Part Time Mid Hudson Valley- Kingston, NY 90 miles North of New York City Skiing, Hiking, Biking and Much More Dr. Chasin and Guarente, Optometrists 240 Lucas Avenue Kingston, NY 12401

EMAIL: DRMC@EYES-OK.COM

Staff Optometrist Wanted

Bard Optical is a family owned full-service retail optometric practice with 22 offices (and growing) throughout Central Illinois. Bard Optical prides itself on having a progressive optometric staff whose foundation is based on one-on-one patient service. We are currently accepting CV/resumes for Optometrists to join our medical model optometric practice that includes extended testing. The practice includes but is not limited to general optometry, contact lenses and geriatric care. Salaried, full-time positions are available with excellent base compensation and incentive programs and benefits. Some part-time opportunities may also be available.

Current positions are available in Bloomington/Normal, Decatur/Forsyth, Peoria, Sterling and Canton as we continue to grow with new and established offices.

Please email your information to **mhall@bardoptical.com** or call Mick at **309-693-9540 ext 225**.
Mailing address if more convenient is:
Bard Optical
Attn: Mick Hall, Vice President
8309 N Knoxville Avenue
Peoria, IL 61615

Bard Optical is a proud Associate Member of the Illinois Optometric Association.

BARD OPTICAL

www.bardoptical.com

Practice For Sale

Products and Services

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

Diabetes Gone Wild

New technology helps surgeons remove dense preretinal membranes in an eye with advanced PDR. **By Alan Franklin, MD, PhD**

Diabetic retinopathy (DR) is the leading cause of blindness in the United States among working age adults and the most common disease to require a vitrectomy.[1] While careful management can delay, or even eliminate, the need for a vitrectomy, in our experience, the majority with catastrophic vision loss present with little to no history of routine eye care. Even with diligent follow-up, some diabetic patients progress rapidly.

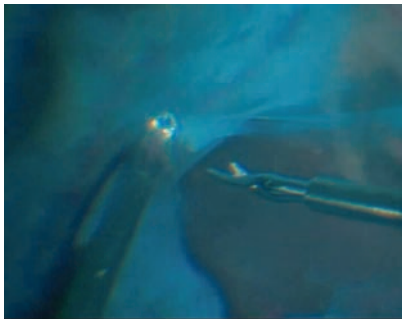
When to Refer

Most optometrists are comfortable with routine DR evaluations in mild to moderate cases. But the longer a patient has diabetes, and especially with a lack of blood sugar control, the greater the suspicion that they may develop more advanced DR. And when damage becomes severe—beyond primary diabetic macular edema (DME)—patients may need surgical intervention. The indications for pars plana vitrectomy are far more serious than primary DME and will require immediate referral. These indications include: vitreous hemorrhage, tractional retinal detachment, pre-macular hemorrhage and DME caused by traction.

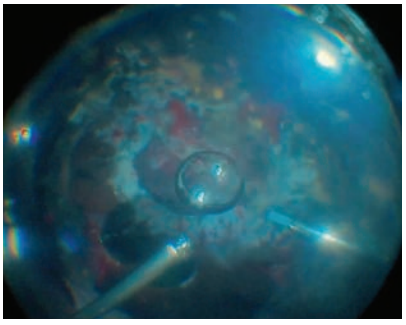
Repairing the Retina

Patients with proliferative diabetic retinopathy who suffer from a tractional retinal detachment may need specialized surgical intervention.

The resulting inflammation and bleeding within the retina and subretinal space lead to excessive scaring in both the retina and vitreous. Removing this scar tissue becomes the challenge and is often the main variable for success. Removal begins with bimanual forceps and scissors to separate and access underneath the fibrous membranes. The surgeon then removes the membranes using a beveled vitrectomy instrument that can cut up to 10,000 times per minute. Next, a laser can help tack down any retinal tears or breaks and even prophylactically treat areas at risk or were under traction. The surgery usually concludes with an air/fluid exchange or placement of silicone oil in the vitreous cavity.



The surgeon first uses forceps and scissors to separate the membranes.



At the conclusion of the surgery, the surgeon adds air and silicone oil.

Post-op Complications

Surgeons always attempt to minimize postoperative inflammation, but other complications include: elevated IOP, cataract formation, rubeosis and neovascular glaucoma, postoperative vitreous hemorrhage, anterior hyaloidal fibrovascular proliferation and retinal redetachment.

Comanaging optometrists can manage elevated IOP with aqueous suppressants, not prostaglandins due to risk of further inflammation. If a patient develops cataracts, clinicians can monitor for progression and visual significance. All other complications should be referred back to the retinal surgeon for evaluation.

Because these eyes have poor inflammation control, which led to the initial surgery, it is not uncommon for post-surgical complications or further pathology progression. ■

Dr. Franklin practices at the Diagnostic & Medical Clinic in Mobile, AL.

New Tools

With new 25-gauge and 27-gauge retinal surgeries, specialists can more precisely dissect retinal tissue and cause fewer complications. These smaller instruments typically remove tissue efficiently, 25-gauge better than 27-gauge, so size does matter. In addition, both can cut through relatively thick fibrotic membranes.

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

1. Spraul CW, Grossniklaus, HE. Vitreous Hemorrhage. Surv Ophthalmol. 1997;42:3-39.

Diagnostic **Quiz**

A Suspicious Freckle

By Andrew S. Gurwood, OD

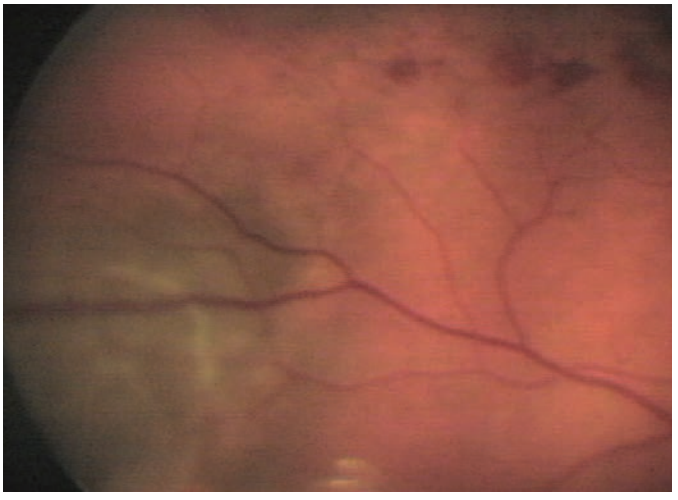
History

A 59-year-old male presented to the office with a chief complaint of blurry vision, with and without his correction at distance and near, in his right eye for three weeks. He denied having any previous ocular injuries or surgeries. His systemic history, however, was remarkable for a 10-year history of hypertension, appropriately managed with lisinopril. The patient reported no history of allergies of any kind.

Diagnostic Data

His best-corrected entering visual acuities were 20/50 OD and 20/20 OS at distance and near with no improvement upon pinhole. His external examination was normal with smooth extraocular muscle movements, normal color vision, full confrontation visual fields and no evidence of afferent pupil defect. Biomicroscopic examination of the anterior chamber found normal structures and tissues with open angles and normal intraocular pressures measuring 15mm Hg OU via Goldmann applanation.

The pertinent posterior segment finding—pictured—was discovered during dilated fundus exam.



This 59-year-old hypertensive patient's right eye shows findings that may explain his blurry vision. Can you diagnose him?

Your Diagnosis

Does this case require additional tests? How would you manage this patient? What is the likely prognosis? To find out, please visit www.reviewofoptometry.com. ■

Retina Quiz Answers *(from page 94):* 1) b; 2) c; 3) d; 4) a.

Next Month in the Mag

In July, *Review of Optometry* will present its 23rd annual glaucoma report.

Topics include:

- *How Up-And-Coming Glaucoma Medications Will Fit Into Your Armamentarium*
- *The State of Minimally Invasive Glaucoma Surgery, Today and Tomorrow*
- *Comanaging the Glaucoma Surgical Patient*
- *10-2 Visual Field Testing—A Tool for All Glaucoma Stages*

Also in this issue:

- *Caring for Patients with Traumatic Brain Injuries* (earn 2 CE credits)
- *The Connection Between Dry Eye Disease and Systemic Conditions*
- *Antiviral Therapy: Differential Diagnosis of Herpes Simplex Virus*

REVIEW OF OPTOMETRY (ISSN 0147-7633) IS PUBLISHED MONTHLY, 12 TIMES A YEAR BY JOBSON MEDICAL INFORMATION LLC, 440 9TH AVENUE, NEW YORK, NY 10013-1678. PERIODICALS POSTAGE PAID AT NEW YORK, NY AND ADDITIONAL MAILING OFFICES. POSTMASTER: SEND ADDRESS CHANGES TO REVIEW OF OPTOMETRY, PO BOX 81, CONGERS, NY 10920-0081. SUBSCRIPTION PRICES: US: ONE YEAR $56; TWO YEARS $97, CANADA: ONE YEAR $88, TWO YEARS $160, INT'L: ONE YEAR $209, TWO YEARS $299. FOR SUBSCRIPTION INFORMATION CALL TOLL-FREE (877) 529-1746 (USA); OUTSIDE USA, CALL (845) 267-3065. OR EMAIL US AT REVOPTOMETRY@CAMBEYWEST.COM. PUBLICATIONS MAIL AGREEMENT NO: 40612608. CANADA RETURNS TO BE SENT TO BLEUCHIP INTERNATIONAL, P.O. BOX 25542, LONDON, ON N6C 6B2.

THE BAUSCH + LOMB ULTRA® CONTACT LENS FAMILY IS NOW COMPLETE

Now for Astigmatism

Provide a wider range of patients with all-day comfort and consistently clear vision[1]

Featuring
MoistureSeal™ TECHNOLOGY

Helps lenses maintain
95% of their moisture for a full 16 hours[2]

FOR MORE INFORMATION, CONTACT YOUR REPRESENTATIVE OR CALL 1-800-828-9030.

REFERENCES: 1. Results from a 7-investigator, multi-site 2-week study of Bausch + Lomb ULTRA® for Astigmatism contact lenses on 157 current soft contact lens wearer. **2.** Data on file. Bausch & Lomb Incorporated. Rochester, NY; 2013.

BAUSCH + LOMB
See better. Live better.

INTRODUCING AIR OPTIX® PLUS HYDRAGLYDE CONTACT LENSES

2 UNIQUE TECHNOLOGIES 1 OUTSTANDING LENS

SMARTSHIELD® TECHNOLOGY **EXCELLENT DEPOSIT PROTECTION**[1,2] + HYDRAGLYDE MOISTURE MATRIX **LASTING LENS SURFACE MOISTURE**[3,4]

FOR A LIMITED TIME, NEW WEARERS CAN

SAVE UP TO $100

ON AN ANNUAL SUPPLY VIA MAIL-IN REBATE*
WITH THE AIR OPTIX® CHOICE PROGRAM!

Visit AIROPTIXCHOICE.com to learn more

PERFORMANCE DRIVEN BY SCIENCE®

*Rebate is in the form of an Alcon VISA® Prepaid Card. Certain criteria must be met to be eligible for the full rebate. Must be a new patient to the AIR OPTIX® family of contact lenses or an existing patient that is switching lenses within the AIR OPTIX® family. Must purchase an annual supply (four 6-ct boxes) of AIR OPTIX® brand contact lenses (excluding AIR OPTIX® AQUA lenses) within 90 days of eye exam or contact lens fitting. Rebate submission must be postmarked (or submitted electronically) within 60 days of lens purchase date. Valid on purchases made at participating retailers through 6-30-17. Visit AIROPTIXCHOICE.com for complete terms and conditions.

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.

References: 1. Nash W, Gabriel M, Mowrey-Mckee M. A comparison of various silicone hydrogel lenses; lipid and protein deposition as a result of daily wear. *Optom Vis Sci.* 2010;87:E-abstract 105110. **2.** Nash WL, Gabriel MM. Ex vivo analysis of cholesterol deposition for commercially available silicone hydrogel contact lenses using a fluorometric enzymatic assay. *Eye Contact Lens.* 2014;40(5):277-282. **3.** *In vitro* study over 16 hours to measure wetting substantivity; Alcon data on file, 2015. **4.** *In vitro* wetting analysis: out-of-pack and wetting substantivity; Alcon data on file, 2014.

Alcon A Novartis Division

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

 1/17 US-AOH-16-E-4693a

Rx only