REVIEW OF OPTOMETRY • VOL. 160, NO. 6 • JUNE 15, 2023 • Tips for Better AMD Diagnosis • Wh

Focus on Refraction: Kinetic Testing in TBI, P. 28 • Therapeutic Review: Hypotony Hits Home, P. 80

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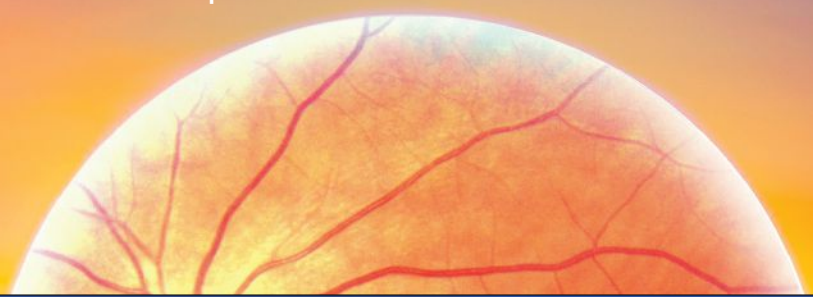
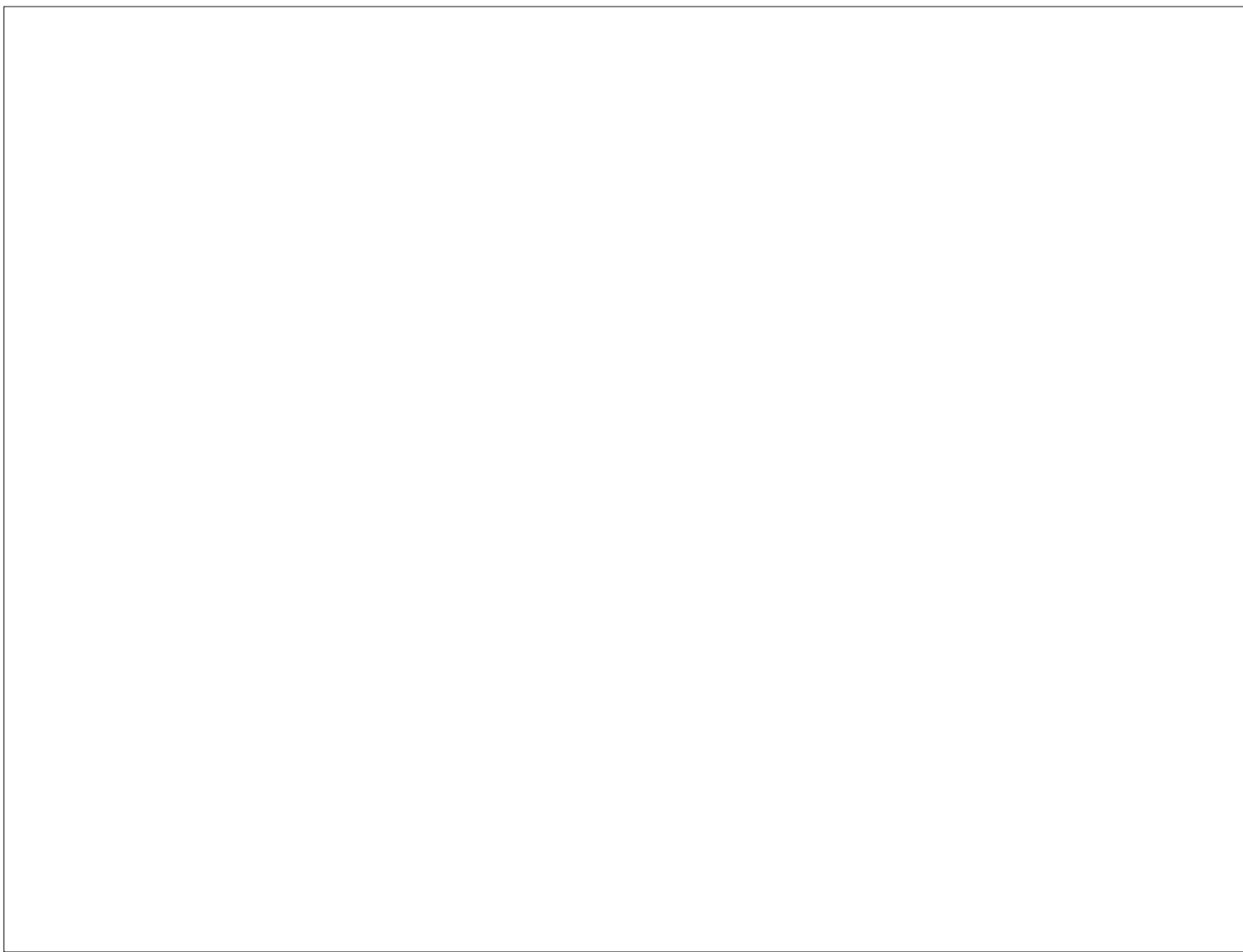
14TH ANNUAL RETINA REPORT

RETINA ON THE RISE





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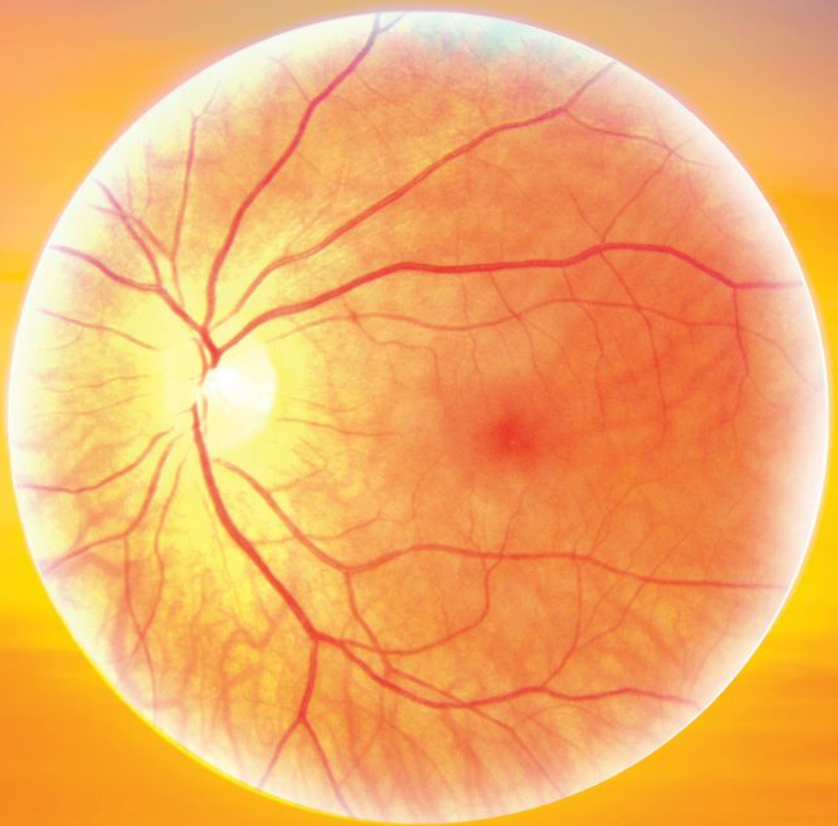
Leadership in clinical care

14TH ANNUAL RETINA REPORT

RETINA ON THE RISE

New meds, methods and motivations are bringing it to the forefront of optometric care.



REVIEW OF OPTOMETRY • VOL. 160, NO. 6 • JUNE 15, 2023 • Tips for Better AMD Diagnosis • When to Make Retinal Referrals • Assessing Vitreous Opacities • Nutrition and the Retina • Choroidal Folds: What Do They Signify?

BRIEF SUMMARY OF PRESCRIBING INFORMATION

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

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

Initial U.S. Approval: 2017

1 INDICATIONS AND USAGE

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

4 CONTRAINDICATIONS

None

5 WARNINGS AND PRECAUTIONS

5.1 Pigmentation

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

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

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

5.2 Eyelash Changes

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

5.3 Intraocular Inflammation

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

5.4 Macular Edema

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

5.5 Bacterial Keratitis

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

5.6 Use with Contact Lens

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

6 ADVERSE REACTIONS

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

6.1 Clinical Trials Experience

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

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

8 USE IN SPECIFIC POPULATIONS

8.1 Pregnancy

Risk Summary

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

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

The background risk of major birth defects and miscarriage for the indicated population is unknown. However, the background risk in the U.S. general population of major birth defects is 2 to 4%, and of miscarriage is 15 to 20%, of clinically recognized pregnancies.

Data

Animal Data

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

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

8.2 Lactation

Risk Summary

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

8.4 Pediatric Use

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

8.5 Geriatric Use

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

13 NONCLINICAL TOXICOLOGY

13.1 Carcinogenesis, Mutagenesis, Impairment of Fertility

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

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

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

13.2 Animal Toxicology and/or Pharmacology

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

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

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VYZULTA®
(latanoprostene bunod ophthalmic solution), 0.024%

A BRANDED FIRST-LINE TREATMENT, RIGHT OUT OF THE GATE

IOP reduction for your open-angle glaucoma patients—with more horsepower

VYZULTA delivered up to **9.1 mmHg** mean IOP reduction from baseline—a greater reduction vs timolol—in pivotal studies[1,2]*

Excellent tolerability with <1% discontinuation rates due to ocular AEs[3,4]

Now with ~70% coverage for Medicare Part D and commercially insured patients nationwide†

LEARN MORE ABOUT VYZULTA HORSEPOWER AT VYZULTAHCP.COM

*Pivotal study designs: Two Phase 3, randomized, multicenter, parallel-group studies, APOLLO and LUNAR, evaluating noninferiority of once-daily VYZULTA vs twice-daily timolol maleate 0.5% in patients with open-angle glaucoma or ocular hypertension. Primary endpoint was IOP measured at 9 assessment time points in study eye. APOLLO (VYZULTA, n=284; timolol, n=133) and LUNAR (VYZULTA, n=278; timolol, n=136).[1,2]

†MMIT Analytics3, December 2022.

AE=adverse event; IOP=intraocular pressure.

INDICATION

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

IMPORTANT SAFETY INFORMATION

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

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

References: 1. Weinreb RN, Scassellati Sforzolini B, Vittitow J, Liebmann J. Latanoprostene bunod 0.024% versus timolol maleate 0.5% in subjects with open-angle glaucoma or ocular hypertension: the APOLLO study. *Ophthalmology*. 2016;123(5):965-973. **2.** Medeiros FA, Martin KR, Peace J, Scassellati Sforzolini B, Vittitow JL, Weinreb RN. Comparison of latanoprostene bunod 0.024% and timolol maleate 0.5% in open-angle glaucoma or ocular hypertension: the LUNAR study. *Am J Ophthalmol*. 2016;168:250-259. **3.** VYZULTA Prescribing Information. Bausch & Lomb Incorporated. **4.** Weinreb RN, Liebmann JM, Martin KR, Kaufman PL, Vittitow JL. Latanoprostene bunod 0.024% in subjects with open-angle glaucoma or ocular hypertension: pooled phase 3 study findings. *J Glaucoma*. 2018;27(1):7-15.

BAUSCH + LOMB

Washington Passes Minor Surgery Scope Bill, Alabama and California Retool for 2024 Effort

Trained ODs in the Evergreen State will now be authorized to perform chalazion removal, certain injections and non-cosmetic eyelid procedures, among other added privileges.

Since January, at least 10 states have introduced bills proposing to expand optometry's scope of practice to better reflect the profession's current education and training. Here are updates on a few states with scope bills in play.

Washington Gov. Signs Scope Bill

For the first time in two decades, the scope of practice in Washington state has finally been updated. On May 9, Governor Jay Inslee signed SSB 5389, otherwise known as The Access to Eyecare Act, which now authorizes Washington optometrists with the proper training to perform the following procedures:

- Incision and excision of chalazion
- Injections (subconjunctival, subcutaneous and intramuscular [epinephrine])
- Eyelid surgery (excluding cosmetic surgery or those requiring the use of general anesthesia)
- Use of topical and injectable anesthesia
- Prescribing of oral steroids

The win marks the first for US optometrists in 2023, with the last scope expansion taking place in Colorado a year ago in June 2022. Several other states also still have scope bills in play this legislative session.

"The bill signing completes a years-long effort by the profession to update our state's scope of practice laws so that they more closely align with the standard of optometric care and the laws in other states," says the president of the Optometric Physicians of Washington (OPW), Michael Sirott, OD, in a press release. "Optometrists are frontline health care workers who often serve as primary care providers, especially in rural areas of our state. This bill will allow me and my colleagues to more fully treat our patients and ensure they receive access to safe, high-quality care without incurring additional delays, travel costs or expenses to address their eyecare needs," Dr. Sirott adds.

Prior to the governor's signature, the legislation received strong bipartisan support in both the Senate and House, which passed SSB 5389 with votes of 46-2 and 81-15, respectively. However, the legislative process did have bumps along the way. While the original bill had proposed that optometrists be allowed to perform certain laser procedures and suturing, the final document removed such language due to amendments that were introduced in both the Senate and House. Nonetheless, the signing of SSB 5389 is a huge victory for optometrists in and out of the state, and it will serve as a precedent for Washington's future legal fight to add laser privileges to the practice scope.

In the OPW press release, Dr. Sirott accredits the win to the advocacy efforts of OPW members, as well as the efforts and leadership of several members of the Senate—Senators Annette Cleveland and Ann Rivers—and the House—Representatives Marcus Riccelli and Joe Schmick. "We also want to thank Governor Inslee, whose signature is the last step in the process to ensure patients in Washington will have more choices in the delivery of their eye care," says Dr. Sirott.

Governor Jay Inslee signed SSB 5389 into law, effectively expanding Washington's optometric scope of practice for the first time since 2003.

In order for Washington ODs to take advantage of the new privileges, the state's Board of Optometry must first complete its rulemaking process to decide on the training and certification requirements and implementation strategy, a process that could take 18 months or longer, according to the OPW.

Alabama Laser Bill Killed Before Senate Vote

While optometrists in Washington celebrate the win, those in Alabama are facing frustration after a recent Senate motion stuck a fork in their efforts to

add lasers and other procedures to the state's optometric practice scope.

First introduced back in April, HB 349 was the result of years of negotiation, advocacy and legislative efforts, largely led by the Alabama Optometric Association (ALOA). The bill had proposed a long-overdue update to Alabama's optometric practice scope to include YAG capsulotomy, trabeculoplasty, skin lesion removal, fluorescein angiography, intense pulsed light treatment and vaccines during health crises.

The scope bill received strong bipartisan support from the Alabama House, which voted 78-6 in its favor during the May 9 hearing. However, the following week, hours before a scheduled hearing on the Senate floor, the state's Healthcare Committee motioned to terminate the legislation.

The decision came after HB 349's sponsor, Representative Danny Garrett, presented an overview of the bill to the Committee just prior to the scheduled hearing, which proceeded to vote 7-6 against a favorable report. Despite the attendance of about a dozen ODs from the ALOA at the meeting, the entire affair lasted just 10 minutes with minimal discussion. To make matters even more frustrating, Committee members who voted against the bill did not even offer the rationale behind their decisions.

If the bill had been brought to the Senate floor, the ALOA feels confident that it would have received strong bipartisan support, as it did in the House. But, due to the Committee's motion, the ALOA, Alabama optometrists and other supporters of the legislation were denied the opportunity to have their voices heard.

An enduring battle. The first scope bill the ALOA filed was back in 2019, which was ultimately shot down due to concerns regarding OD training. More recently, in March 2022, another scope bill introduced by the ALOA passed the Senate with a vote of 17 to 12. However, the bill did not pass the House Health Committee, ending its run that session.

Alabama's Senate Healthcare Committee voted 7-6 against the state's laser bill prior to a public hearing. This month, the ALOA will discuss plans to reintroduce the legislation.

At the time, Caleb Gardner, OD, former president of the ALOA, commented that, "The session ended, and we didn't get a vote in the House because we ran out of time, which ultimately is because of the power of our opposition."

This year, prior to the bill's official introduction, the ALOA took part in a six-week-long negotiation process overseen by leadership in the House of Representatives to come to an agreement on the provisions. The association reports that in the end, political ophthalmology walked away unwilling to compromise at all on the procedures outlined in the legislation.

Because of this, Alabama's House Health chairman, Representative Paul Lee, decided to write his own bill—HB 349—which optometry accepted, but MD groups aggressively fought back against, which evidently played a hand in the Senate Healthcare Committee's decision.

In it for the long haul. Despite the setback, Howard Day, OD, president of the ALOA, commends the members of the ALOA scope expansion team—referred to as "Hornets"—for fighting hard to advocate for the bill. "I'm super proud of the Hornets Nest," says Dr. Day. "We've never had such passion to update our scope in years. Our esprit de corps is at an all-time high. We will prevail," he assures. "Lasers in '24!"

Efforts to expand the practice scope for Alabama ODs certainly aren't ceasing. This month, the ALOA and other advocates plan to regroup and determine how to reintroduce the legislation in a future session. In the meantime, the association and ODs in the state will remain tenacious in their advocacy efforts and continue to strengthen their relationships with legislators.

"Success boils down to trusted relationships between legislators and key person optometrists," says Dr. Day. "So, if your state is contemplating scope expansion, start now," he advises.

California Bill Hearings Postponed

Like in Alabama, California's scope bill has also taken a backseat—but only temporarily.

Coming back from last year's loss, scope expansion advocates in California introduced an identical laser bill again this past March. The bill proposes that under the clinical circumstances outlined in the bill, ODs in the state should be allowed to perform SLT, peripheral iridotomy, posterior capsulotomy, lesion removal, corneal crosslinking and eyelid injections.

Again sponsored by Assemblymember Evan Low, AB 1570 was scheduled to be heard by the Assembly Business and Professions Committee in April. However, due to Governor Newsom's concerns regarding training requirements—the same reason he vetoed the legislation last session—the author pulled the bill prior to the hearing to allow for more time to work on the amendments necessary to strengthen the governor's support.

California's ongoing fight for optometric laser authority is certainly not over; since AB 1570 is a two-year bill, it will be eligible for a hearing again in January. Until then, the California Optometric Association and other proponents of the bill will be reworking the bill's terms to address the concerns while still retaining the language necessary to adequately expand the practice scope for California optometrists.

B+L, Novaliq Eye Drop Approved For Treatment of DED

Miebo, previously known as NOV03, aims to ease signs and symptoms in as few as 15 days by reducing tear evaporation, the companies say.

After years of clinical trials and widespread anticipation, Bausch + Lomb and Novaliq announced the FDA approval of a new eye drop for evaporative dry eye. Named Miebo (perfluorohexyloctane ophthalmic solution; formerly known as NOV03), the drop is indicated to treat the signs and symptoms of dry eye disease (DED). The solution is also the first on the market to directly target tear evaporation—a leading cause of DED.

Miebo achieved primary endpoints and showed good tolerability in all three trial phases. The two Phase III clinical trials—GOBI and MOJAVE—enrolled more than 1,200 patients with a history of DED and clinical signs of meibomian gland dysfunction (MGD). "The GOBI study showed a 30% improvement in total corneal staining and a 46% greater improvement in eye dryness scores from baseline at the primary endpoint day 57," says Paul Karpecki, OD, medical director for Keplr Vision and the Dry Eye Institutes of Kentucky and Indiana and the chief clinical editor for *Review of Optometry*.

Dr. Karpecki notes that what he finds even more impressive about the Phase III trial results is the speed of the drop's effect. "Miebo demonstrated clinically significant improvement in total corneal staining and symptoms of DED in as early as two weeks," he cites from the company's data. In both Phase III trials, on day 15 (as well as day 57), a significant reduction in total corneal staining was observed in participants using Miebo vs. saline (placebo), B+L and Novaliq reported in a press release.

Adverse reactions from Miebo were scarce in clinical trials. The most common adverse reactions observed in users were blurred vision (1.3% to 3%) and eye redness (1% to 3%).

Photo: Bausch + Lomb

Miebo may benefit those with DED related to MGD, as it targets tear evaporation.

With MGD contributing to most dry eye, Miebo may offer these patients an alternative therapeutic option to other types of drops and in-office procedures.

Age of Menopause Affects Glaucoma Development

Women make up the majority of glaucoma patients; it's been hypothesized that estrogen exposure may provide a protective effect to the optic nerve, while decreased estrogen levels increase the optic nerve's vulnerability to glaucomatous damage. Researchers at an Atlanta VA Center conducted a retrospective study of female veterans to investigate the previously suggested link between menopause and glaucoma and presented their findings at ARVO 2023 in New Orleans.

The study included female patients with negative ophthalmological and menopausal screenings prior to glaucoma and menopause diagnoses.

To estimate the impact of age of menopause on glaucoma onset, the researchers created two separately matched populations. The first group included those with early menopause (n=221, ages 35 to 45) matched against a control menopause group (n=663, 45 to 55). The second group consisted of those with late menopause (n=488, 55 to 65) matched against a control menopause group (n=488, 45 to 55).

The data showed that patients in the early menopause group developed glaucoma an average of 5.5 years before matched controls. Those in the late menopause group developed glaucoma an average of 5.3 years later than matched controls. "These models predicted that for each year later a woman went into menopause, there was a 0.67-year and 0.68-year delay in developing glaucoma for the early-to-control and late-to-control populations, respectively," they wrote in their abstract.

"Our study is the first to demonstrate a direct association between the age of menopause and the onset of glaucoma," they wrote, adding that patients who reach menopause early are likely to develop glaucoma at an earlier age, impacting long-term vision outcomes. "Future work will look at the impact of menopause on glaucoma incidence and role of hormone replacement therapy on glaucoma onset," they concluded.

Abstract content © Association for Research in Vision and Ophthalmology 2023.

Hogan K, Cui X, Giangiacomo A, et al. Menopause is associated with age of developing glaucoma: a retrospective study of female veterans. ARVO 2023 annual meeting.

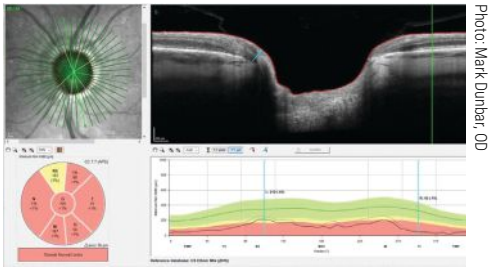


Photo: Mark Dunbar, OD

Those experiencing early menopause developed glaucoma about 5.5 years earlier than average, and those with late menopause developed it about 5.3 years later than average.

NOW APPROVED: the first and only FDA-approved treatment for GA secondary to AMD[1]

GA unravels so much
SAVE RETINAL TISSUE BY SLOWING PROGRESSION[1-3]

SYFOVRE achieved continuous reductions in mean lesion growth rate* vs sham pooled from baseline to Month 24[1]

Monthly	Every Other Month (EOM)
OAKS trial (mm^2): (3.11 vs 3.98) **22%**	OAKS trial (mm^2): (3.26 vs 3.98) **18%**
DERBY trial (mm^2): (3.28 vs 4.00) **18%**	DERBY trial (mm^2): (3.31 vs 4.00) **17%**

SE in trials (monthly, EOM, sham pooled): OAKS: 0.15, 0.13, 0.14; DERBY: 0.13, 0.13, 0.17.

*Slope for baseline to Month 24 is an average of slope of baseline to Month 6, Month 6 to Month 12, Month 12 to Month 18, and Month 18 to Month 24.[1]
Based on a mixed effects model for repeated measures assuming a piecewise linear trend in time with knots at Month 6, Month 12, and Month 18.[1]

AMD=age-related macular degeneration; GA=geographic atrophy; SE=standard error.

Learn more about the SYFOVRE clinical data at SyfovreECP.com/efficacy

INDICATION

SYFOVRE™ (pegcetacoplan injection) is indicated for the treatment of geographic atrophy (GA) secondary to age-related macular degeneration (AMD).

IMPORTANT SAFETY INFORMATION

CONTRAINDICATIONS

- SYFOVRE is contraindicated in patients with ocular or periocular infections, and in patients with active intraocular inflammation

WARNINGS AND PRECAUTIONS

- **Endophthalmitis and Retinal Detachments**
  - Intravitreal injections, including those with SYFOVRE, may be associated with endophthalmitis and retinal detachments. Proper aseptic injection technique must always be used when administering SYFOVRE to minimize the risk of endophthalmitis. Patients should be instructed to report any symptoms suggestive of endophthalmitis or retinal detachment without delay and should be managed appropriately.
- **Neovascular AMD**
  - In clinical trials, use of SYFOVRE was associated with increased rates of neovascular (wet) AMD or choroidal neovascularization (12% when administered monthly, 7% when administered every other month and 3% in the control group) by Month 24. Patients receiving SYFOVRE should be monitored for signs of neovascular AMD. In case anti-Vascular Endothelial Growth Factor (anti-VEGF) is required, it should be given separately from SYFOVRE administration.
- **Intraocular Inflammation**
  - In clinical trials, use of SYFOVRE was associated with episodes of intraocular inflammation including: vitritis, vitreal cells, iridocyclitis, uveitis, anterior chamber cells, iritis, and anterior chamber flare. After inflammation resolves, patients may resume treatment with SYFOVRE.
- **Increased Intraocular Pressure**
  - Acute increase in IOP may occur within minutes of any intravitreal injection, including with SYFOVRE. Perfusion of the optic nerve head should be monitored following the injection and managed as needed.

ADVERSE REACTIONS

- Most common adverse reactions (incidence ≥5%) are ocular discomfort, neovascular age-related macular degeneration, vitreous floaters, conjunctival hemorrhage.

Please see Brief Summary of Prescribing Information for SYFOVRE on the adjacent page.

Trial Design: SYFOVRE safety and efficacy were assessed in OAKS (N=637) and DERBY (N=621), multi-center, 24-month, Phase 3, randomized, double-masked trials. Patients with GA (atrophic nonexudative age-related macular degeneration), with or without subfoveal involvement, secondary to AMD were randomly assigned (2:2:1:1) to receive 15 mg/0.1 mL intravitreal SYFOVRE monthly, SYFOVRE EOM, sham monthly, or sham EOM for 24 months. Change from baseline in the total area of GA lesions in the study eye (mm^2) was measured by fundus autofluorescence (FAF).[1,4]

References: 1. SYFOVRE (pegcetacoplan injection) [package insert]. Waltham, MA: Apellis Pharmaceuticals, Inc.; 2023. **2.** Pfau M, von der Emde L, de Sisternes L, et al. Progression of photoreceptor degeneration in geographic atrophy secondary to age-related macular degeneration. *JAMA Ophthalmol*. 2020;138(10):1026-1034. **3.** Bird AC, Phillips RL, Hageman GS. Geographic atrophy: a histopathological assessment. *JAMA Ophthalmol*. 2014;132(3):338-345. **4.** Data on file. Apellis Pharmaceuticals, Inc.

SYFOVRE™ (pegcetacoplan injection) 15 mg / 0.1 mL

Apellis

SYFOVRE ™ (pegcetacoplan injection), for intravitreal use
BRIEF SUMMARY OF PRESCRIBING INFORMATION
Please see SYFOVRE full Prescribing Information for details.

INDICATIONS AND USAGE

SYFOVRE is indicated for the treatment of geographic atrophy (GA) secondary to age-related macular degeneration (AMD).

CONTRAINDICATIONS

Ocular or Periocular Infections

SYFOVRE is contraindicated in patients with ocular or periocular infections.

Active Intraocular Inflammation

SYFOVRE is contraindicated in patients with active intraocular inflammation.

WARNINGS AND PRECAUTIONS

Endophthalmitis and Retinal Detachments

Intravitreal injections, including those with SYFOVRE, may be associated with endophthalmitis and retinal detachments. Proper aseptic injection technique must always be used when administering SYFOVRE in order to minimize the risk of endophthalmitis. Patients should be instructed to report any symptoms suggestive of endophthalmitis or retinal detachment without delay and should be managed appropriately.

Neovascular AMD

In clinical trials, use of SYFOVRE was associated with increased rates of neovascular (wet) AMD or choroidal neovascularization (12% when administered monthly, 7% when administered every other month and 3% in the control group) by Month 24. Patients receiving SYFOVRE should be monitored for signs of neovascular AMD. In case anti-Vascular Endothelial Growth Factor (anti-VEGF) is required, it should be given separately from SYFOVRE administration.

Intraocular Inflammation

In clinical trials, use of SYFOVRE was associated with episodes of intraocular inflammation including: vitritis, vitreal cells, iridocyclitis, uveitis, anterior chamber cells, iritis, and anterior chamber flare. After inflammation resolves patients may resume treatment with SYFOVRE.

Increased Intraocular Pressure

Acute increase in IOP may occur within minutes of any intravitreal injection, including with SYFOVRE. Perfusion of the optic nerve head should be monitored following the injection and managed as needed.

ADVERSE REACTIONS

Clinical Trials Experience

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

A total of 839 patients with GA in two Phase 3 studies (OAKS and DERBY) were treated with intravitreal SYFOVRE, 15 mg (0.1 mL of 150 mg/mL solution). Four hundred nineteen (419) of these patients were treated in the affected eye monthly and 420 were treated in the affected eye every other month. Four hundred seventeen (417) patients were assigned to sham.

The most common adverse reactions (≥5%) reported in patients receiving SYFOVRE were ocular discomfort, neovascular age-related macular degeneration, vitreous floaters, and conjunctival hemorrhage.

Table 1: Adverse Reactions in Study Eye Reported in ≥2% of Patients Treated with SYFOVRE Through Month 24 in Studies OAKS and DERBY

Adverse Reactions	PM (N = 419) %	PEOM (N = 420) %	Sham Pooled (N = 417) %
Ocular discomfort*	13	10	11
Neovascular age-related macular degeneration*	12	7	3
Vitreous floaters	10	7	1
Conjunctival hemorrhage	8	8	4
Vitreous detachment	4	6	3
Retinal hemorrhage	4	5	3
Punctate keratitis*	5	3	<1
Posterior capsule opacification	4	4	3
Intraocular inflammation*	4	2	<1
Intraocular pressure increased	2	3	<1

PM: SYFOVRE monthly; PEOM: SYFOVRE every other month

*The following reported terms were combined:

Ocular discomfort included: eye pain, eye irritation, foreign body sensation in eyes, ocular discomfort, abnormal sensation in eye

Neovascular age-related macular degeneration included: exudative age-related macular degeneration, choroidal neovascularization

Punctate keratitis included: punctate keratitis, keratitis

Intraocular inflammation included: vitritis, vitreal cells, iridocyclitis, uveitis, anterior chamber cells, iritis, anterior chamber flare

Endophthalmitis, retinal detachment, hyphema and retinal tears were reported in less than 1% of patients. Optic ischemic neuropathy was reported in 1.7% of patients treated monthly, 0.2% of patients treated every other month and 0.0% of patients assigned to sham. Deaths were reported in 6.7% of patients treated monthly, 3.6% of patients treated every other month and 3.8% of patients assigned to sham. The rates and causes of death were consistent with the elderly study population.

USE IN SPECIFIC POPULATIONS

Pregnancy

Risk Summary

There are no adequate and well-controlled studies of SYFOVRE administration in pregnant women to inform a drug-associated risk. The use of SYFOVRE may be considered following an assessment of the risks and benefits.

Systemic exposure of SYFOVRE following ocular administration is low. Subcutaneous administration of pegcetacoplan to pregnant monkeys from the mid gestation period through birth resulted in increased incidences of abortions and stillbirths at systemic exposures 1040-fold higher than that observed in humans at the maximum recommended human ophthalmic dose (MRHOD) of SYFOVRE (based on the area under the curve (AUC) systemically measured levels). No adverse maternal or fetal effects were observed in monkeys at systemic exposures approximately 470-fold higher than that observed in humans at the MRHOD.

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

Lactation

Risk Summary

It is not known whether intravitreal administered pegcetacoplan is secreted in human milk or whether there is potential for absorption and harm to the infant. Animal data suggest that the risk of clinically relevant exposure to the infant following maternal intravitreal treatment is minimal. Because many drugs are excreted in human milk, and because the potential for absorption and harm to infant growth and development exists, caution should be exercised when SYFOVRE is administered to a nursing woman.

Females and Males of Reproductive Potential

Contraception

Females: It is recommended that women of childbearing potential use effective contraception methods to prevent pregnancy during treatment with intravitreal pegcetacoplan. Advise female patients of reproductive potential to use effective contraception during treatment with SYFOVRE and for 40 days after the last dose. For women planning to become pregnant, the use of SYFOVRE may be considered following an assessment of the risks and benefits.

Pediatric Use

The safety and effectiveness of SYFOVRE in pediatric patients have not been established.

Geriatric Use

In clinical studies, approximately 97% (813/839) of patients randomized to treatment with SYFOVRE were ≥ 65 years of age and approximately 72% (607/839) were ≥ 75 years of age. No significant differences in efficacy or safety were seen with increasing age in these studies. No dosage regimen adjustment is recommended based on age.

PATIENT COUNSELING INFORMATION

Advise patients that following SYFOVRE administration, patients are at risk of developing neovascular AMD, endophthalmitis, and retinal detachments. If the eye becomes red, sensitive to light, painful, or if a patient develops any change in vision such as flashing lights, blurred vision or metamorphopsia, instruct the patient to seek immediate care from an ophthalmologist.

Patients may experience temporary visual disturbances associated either with the intravitreal injection with SYFOVRE or the eye examination. Advise patients not to drive or use machinery until visual function has recovered sufficiently.

Manufactured for:
Apellis Pharmaceuticals, Inc.
100 Fifth Avenue
Waltham, MA 02451

SYF-PI-17Feb2023-1.0

2/23 US-PEGGA-2200163 v2.0

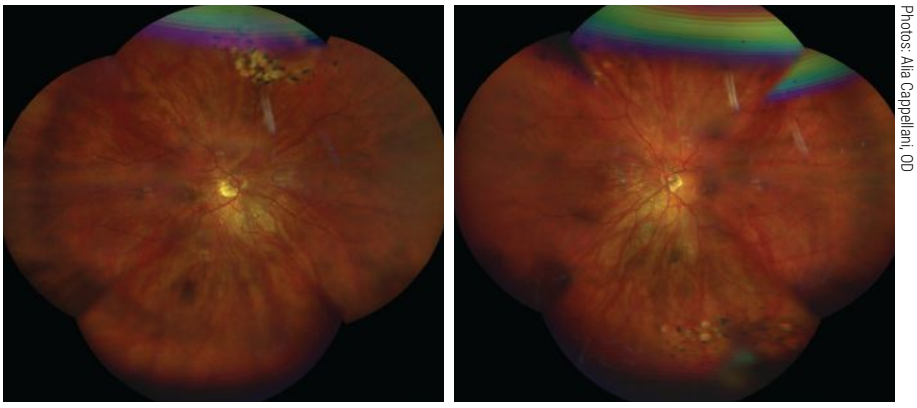
2023 ORS Resident Case Report Contest Winner

This paper, by an NSUOCO resident, highlights the complications of pathologic myopia.

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

The case described below was selected by the ORS Awards Committee as the winner of the seventh annual Larry Alexander Resident Case Report Contest.

"Every year, I am so excited to have the opportunity to review the resident manuscript submissions," says Julie Rodman, OD, professor and chief of the Fort Lauderdale (Broward) Eye Care Institute at Nova Southeastern University in Florida, and ORS treasurer. "I am always so impressed by the outstanding cases and the breadth of knowledge displayed by the residents. This year's winning manuscript provided outstanding visuals including the use of multimodal imaging."



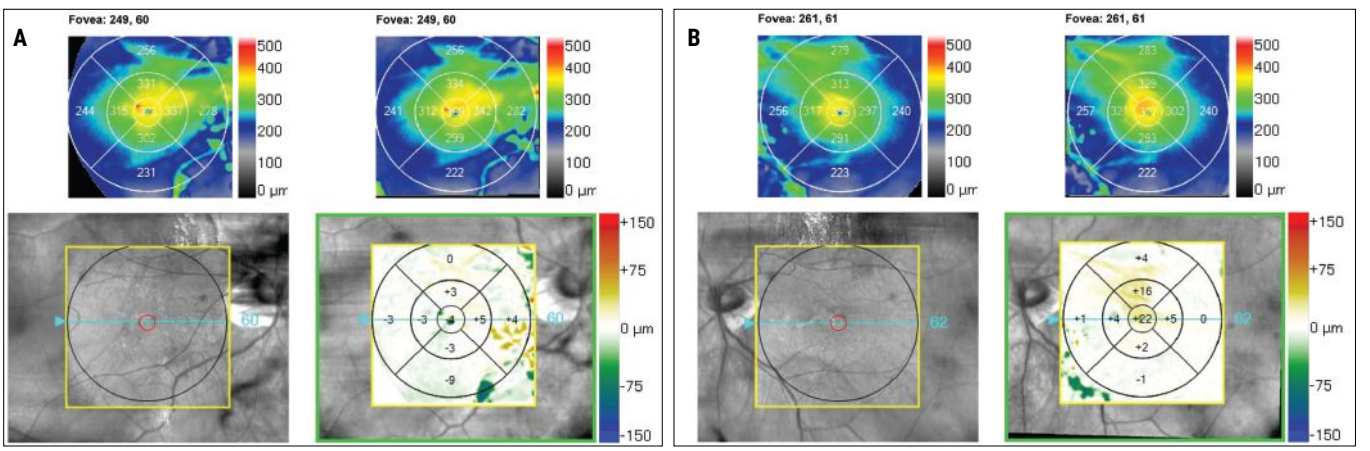
Photos: Alia Cappellani, OD

Ultra-widefield retinal photos of the right (A) and left (B) eyes in a 54-year-old patient with pathologic myopia showing a tessellated fundus, tilted nerve with temporal crescent. Laser scars surrounding a laser tears superior OD/OS and lattice degeneration inferior OS.

The full text and images of the report are available on our website, reviewofoptometry.com.

Alia Cappellani, OD, a cornea and contact lens resident at Northeastern State University Oklahoma College of Optometry, presented a case involving a 54-year-old Native American female with pathologic myopia, which represents degenerative ocular structural complications that arise secondary to high myopia and can lead to severe visual impairment. The report discussed various diagnostic entities to ensure a proper diagnosis, the various manifestations and management options.

Among Dr. Cappellani's conclusions from the case is that pathologic myopia has a significant socioeconomic impact worldwide, and the rising prevalence rates of myopia imply that more ocular diseases will follow suit, shedding light on evidence-based preventative measures, which could ultimately lead to a reduction of patients with pathologic myopia. She also emphasized the importance of multimodal imaging for the diagnosis and management of posterior segment structural changes that occur secondary to excessive axial elongation, as well as educating patients on the necessity of consistent monitoring to avoid visual loss.



OCT change analyses of the macula, six months apart. The right eye (A) is stable; the left (B) shows evidence of +22µm central subfield thickness.

Parkinson's Drug Has Protective Effect Against Conversion to Wet AMD

This was determined by retrospective analyses of patients exposed to levodopa during the course of the disease.

While dry age-related macular degeneration (AMD) is generally less visually disabling to patients, especially in its earliest stages, the spectre of conversion to wet AMD looms large in the minds of patients. Neovascularization may not be totally unavoidable, but new research shows that exposure to the dopamine precursor levodopa (L-dopa), commonly used to treat Parkinson's disease, may offer a protective effect in patients who need that medication. Perhaps in time the potent L-dopa, which carries a significant side effect profile, will be studied as a potential therapy in AMD.

Two retrospective analyses and a case-control study considered the implications of levodopa exposure in: (1) wet AMD patients followed for two years, (2) dry AMD patients followed for one to five years and (3) newly diagnosed neovascular AMD cases that were matched with controls without neovascular AMD. Eyes were divided into two groups: those exposed to levodopa before or on AMD diagnosis date and eyes not exposed to the drug.

In the first study, the number of intravitreal injections used over a course of two years was one less on average for wet AMD eyes exposed to levodopa (11 rather than 12). When looking at studies two and three, L-dopa exposure in dry AMD eyes was associated with a 21% reduced risk of conversion at year two, 35% at years three and four and 28% at year five. Cumulative levodopa dose 100mg to 300mg per day or greater than 300mg per day over two years was associated with decreased risk of wet AMD development by 14% and 23%, respectively.

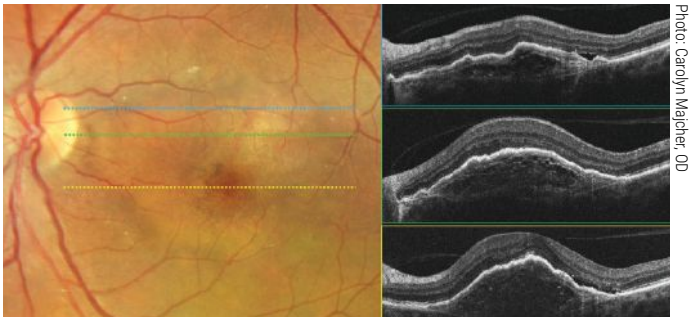


Photo: Carolyn Majcher, OD

Dopamine precursor levodopa may provide protective effects against neovascular AMD conversion and diagnosis.

Based on the findings, the researchers believe "these data from two non-overlapping independent databases support our hypothesis that L-dopa may be protective against conversion to neovascular AMD," they wrote in their paper.

Furthering this conclusion is the finding that eyes exposed to levodopa were in patients older than the control subjects, suggesting AMD might develop slower or later from a protective levodopa effect. In this study, non-neovascular control eyes were 76 years old on average, while levodopa-treated wet AMD eyes were 79 years old. Adding to this association's credibility: older age was found to increase risk of wet AMD conversion by 4% to 5%.

Sex was another factor found to influence conversion risk, with male sex reducing risk to wet AMD conversion by 11% to 16%. Conversely, female sex was associated with 10% increased odds of developing wet AMD.

The researchers noted that levodopa can cross the blood-retinal and blood-RPE barriers and that 100mg to 300mg of the dopamine precursor would be "biologically justifiable" due to very high flow within the choroid.

However, they do caution that "there is a possibility of unexpected side effects in people without Parkinson's disease, and we should refrain from easy use of L-dopa. Thus, a randomized, controlled clinical trial to test the ability of low-dose L-dopa at 100mg to 300mg per day to reduce the risk of conversion to neovascular AMD in high-risk eyes should be considered."

Hyman MJ, Skondra D, Aggarwal N, et al. Levadopa is associated with reduced development of neovascular age-related macular degeneration. Ophthalmol Retina. May 3, 2023. [Epub ahead of print].

IN BRIEF

Delayed Glaucoma Treatment Doesn't Always Put Patients Behind. Upon comparing the long-term visual outcomes in the two arms of the Early Manifest Glaucoma Trial to determine if delayed treatment was associated with decreased visual function, researchers found that **vision impairment occurred at similar rates in both treatment arms with a slight preponderance in the treatment group, while visual field (VF) damage was slightly higher in the control group.**

A total of 255 subjects with newly detected, untreated glaucoma were randomized to immediate treatment with topical betaxolol and argon laser trabeculoplasty or to no initial treatment as long as no progression was detected. Subjects were followed with standard automated perimetry, VA measurements and tonometry for up to 21 years.

The percentages of eyes with vision impairment or blindness were slightly higher in the treated group than in the untreated control group (12.1% vs. 11.0% and 9.4.% vs. 6.1%, respectively), as were subjects with vision impairment ≥one eye (19.5% vs. 18.7%). The differences were not significant, nor were the cumulative incidences of vision impairment in at least one eye. Controls had more VF loss than treated patients, with median mean deviation in the worse eye of -14.73dB vs. -12.85dB and rate of progression of -0.74dB/year vs. -0.60dB/year, not statistically significant. VA difference was minimal.

"These findings indicate that the cost of delaying glaucoma diagnosis, and thus treatment, a few years in patients with early glaucoma should not be associated with a large visual function penalty but probably with some smaller penalty," the team concluded.

Daniel Lee D, Yu Y, Bunya VY, et al. Two-year progression of dry eye disease in the dry eye assessment and management (DREAM) study. ARVO 2023 annual meeting.

The Paradigm Shift in Keratoconus Treatment

Daniel G. Fuller, OD, FAAO Dipl, FSLS
Memphis, TN

KEY TAKEAWAYS

- Only iLink® cross-linking can slow or halt the progression of keratoconus.
- Referring progressing patients to a cornea specialist prior to vision loss is ideal.
- Slowing or halting keratoconus progression may allow patients to continue to tolerate contact lenses.

Ten years ago, there was little reason to refer a patient with keratoconus to a cornea specialist early in the course of their disease. All we could do was manage patients' vision as long as possible, hoping they didn't progress to needing a corneal transplant.

The approval of iLink® cross-linking marked a major paradigm shift in keratoconus management. Professional societies have adjusted treatment guidelines to reflect the ability of cross-linking treatment to slow or halt progression of the underlying disease. The American Academy of Ophthalmology, for example, now states in its Preferred Practice Pattern (PPP) that referral prior to vision loss is ideal, and that when keratoconus is suspected, more frequent follow-up to look for progression is warranted.[1] Any signs of progression or onset of keratoconus at a young age should lead to a prompt referral.[1]

Optometry is very good at helping patients with keratoconus see better with gas permeable (GP), hybrid, and scleral lenses. But as rewarding as it is to help the vision-impaired, we can have an even greater impact by catching this disease early and referring progressing patients for cross-linking before they lose vision, just as we refer glaucoma patients for treatment as soon as the disease is detected. For patients who are still in their peak earning and learning years, early treatment could mean 50+ years of functional vision.

Cost-effective and FDA approved

A discrete-event simulation model showed that, compared to conventional treatment, iLink cross-linking would reduce the rate of penetrating keratoplasty by 26%, and result in patients spending 28 fewer years in the advanced stages of keratoconus—all while saving money for patients, insurers, and society.[2]

The iLink procedure is an epithelium-off treatment that has undergone the scrutiny of randomized controlled clinical trials as part of the FDA approval process, demonstrating proven efficacy and safety. It is important to refer patients to doctors who use iLink, the only cross-linking procedure approved by the FDA. I believe that good science promotes good patient care and, in the case of iLink, also allows patients to use their insurance.

Vision correction post cross-linking

Slowing or halting keratoconus progression may allow patients to continue to tolerate contact lenses.[3,4] Typically, patients can resume contact lens wear within one to three months of the cross-linking procedure, although I find that corneal remodeling may continue for up to 12 months post-treatment. During this time, lens parameters may need to be adjusted. About one-third of eyes are able to continue in habitual contact lenses after cross-linking, while two-thirds require a new contact lens fit.[5]

With iLink cross-linking and modern specialty contact lenses, we have the best keratoconus management options now that I've ever seen. This represents not just a business opportunity, but the chance to have a life-changing impact on our patients. ▪

Contact Lens Fitting Post Cross-Linking[5]

100% ACCEPTABLE FIT | **65%** IMPROVED SUBJECTIVE COMFORT | **20%** INCREASE IN NEAR-IDEAL FIT

REFERENCES:
1. Garcia-Ferrer FJ, Akpek EK, Amescua G, et al, for the AAO PPP Corneal/External Disease Committee. Corneal ectasia PPP 2018. **2.** Lindstrom RL, Berdahl JP, Donnenfeld ED, et al. Corneal cross-linking versus conventional management for keratoconus: a lifetime economic model. J Med Econ 2021;24(1):410-20. **3.** Singh K, Bhattacharyya M, Arora R, et al. Alterations in contact lens fitting parameters following cross-linking in keratoconus patients of Indian ethnicity. Int Ophthalmol. 2018;38(4):1521-30. **4.** Isik P, Harbiyeli II, Erdem E, Yagmur M. Improved contact lens fitting after corneal cross-linking in eyes with progressive keratoconus. Cont Lens Anterior Eye. 2021;3:101488. **5.** Mandathara PS, Kalaiselvan P, Rathi VM, et al. Contact lens fitting after corneal collagen cross-linking. Oman J Ophthalmol. 2019;12(3):177-80.

SCAN WITH PHONE
Learn more about iLink corneal cross-linking here

iLink® CROSS-LINKING PROCEDURE

Sponsored by Glaukos
GLAUKOS TRANSFORMING VISION

Florida Not-a-doctor Bill Vetoed by Gov. DeSantis

Persistent advocacy from optometry preserves ODs' right to use professional titles.

For months, optometry has been vigorously fighting to push back against SB 230, a Florida bill threatening to ban ODs from using the titles "doctor" and "physician" and subjecting them to a felony prosecution for doing so. On June 2, optometrists and advocates finally saw the fruits of their labor when Governor Ron DeSantis vetoed the harmful legislation.

"It is with sincere gratitude that the governor was able to see that SB 230 discriminated against the honorable profession of optometry and that the role optometrists play in the delivery of quality eye care for all Floridians will not be diminished or tarnished in any way," says Mark Marciano, OD, president of the Florida Optometric Association (FOA).

Ronald Benner, OD, president of the American Optometric Association (AOA), also praises the governor's decision. "Governor DeSantis's decisive action is a powerful message of support for and recognition of optometry's essential and expanding role in health care," he says.

The legislation, introduced in February, received strong bipartisan support from both the Florida Senate (37-0) and House (111-3). The original version of the bill proposed that while other medical specialists with similar four-year postgraduate degrees, such as dentists, chiropractors and podiatrists,

RON DESANTIS
GOVERNOR

June 2, 2023

Secretary Cord Byrd
Secretary of State
R.A. Gray Building
500 South Bronough Street
Tallahassee, Florida 32399

Dear Secretary Byrd:

By the authority vested in me as Governor of the State of Florida, under the provisions of Article III, Section 8, of the Constitution of Florida, I do hereby veto and transmit my objections to Committee Substitute for Committee Substitute for Senate Bill 230 (CS/CS/SB 230), enacted during the 125th Session of the Legislature of Florida, during the Regular Session of 2023 and entitled:

An act relating to Health Care Practitioner Titles and Designations

Sincerely,

Ron DeSantis
Governor

THE CAPITOL
TALLAHASSEE, FLORIDA 32399 • (850) 717-9249

On June 2, Governor Ron DeSantis vetoed Florida's anti-optometry bill SB 230.

would still be able to identify themselves as "doctor" and "physician," optometrists would not, due to the exclusion of the terms from Florida's optometric practice act.

In an attempt to remove this stipulation from the bill's language, after the third Senate reading last month, optometry advocates including the FOA and AOA introduced this amendment:

"*An optometrist licensed under chapter 463 [Florida's practice act for optometrists] may use the following titles and abbreviations as applicable to his or her license, specialty and certification: 'doctor of optometry,' 'optometric physician' and other titles or abbreviations authorized under his or her practice act.*"

Both the Florida Senate and House refused to accept the amendment, sending the final bill to the Governor's desk with its anti-optometry language intact. But, in the end, optometry went home with the win, thanks to Gov. DeSantis's commendable decision to veto and the tireless advocacy efforts of individual ODs and their associations.

"The FOA board of trustees wants to thank everyone across the state of Florida and around the country who took the time to write or call the governor's office asking for a veto," praises Dr. Marciano. "We also wish to thank the AOA and its staff, every state association and the many individuals, entities and organizations for their ongoing support of Florida optometry."

Dr. Benner also commends the state society. "The FOA fought and prevailed in a challenging battle and did so by remaining confident in the knowledge that truth, fairness and the needs of patients across the Sunshine State would prevail," he says. "I'm proud to say thank you to Governor DeSantis, the FOA and colleagues from across the country who helped ensure that our profession was heard loud and clear."

While we celebrate the triumphant win in Florida, it's important to recognize that at least six other states still have not-a-doctor bills in play this year, highlighting the need for the optometry community to remain tenacious in advocating for ODs' rights.

IN BRIEF

iStent Improves Post-Cataract IOP Reduction. The iStent (Glaukos) is a popular minimally invasive glaucoma device inserted either at the time of phacoemulsification or as a standalone procedure. A new study looked at the iStent's IOP-reducing ability when combined with cataract surgery. Endpoints included reduction in IOP and number of glaucoma drops.

A total of 1,453 eyes from 10 studies were included. Of those, 853 received combined phaco/iStent and 600 underwent just conventional cataract surgery. **A 22% IOP reduction was observed in the combined surgery group, while phaco alone resulted in a 14% IOP decrease. IOP reduction in the combined surgery group averaged 4.7±2mm Hg. Phaco alone resulted in a mean IOP reduction of 2.8±1.9mm Hg.**

Similarly, use of post-op eye drops was also reduced more in the combined group. These patients experienced an average decrease of 1.2±0.3 drops vs. phaco alone, which saw a decrease of 0.6±0.6 drops. Based on these results, the study authors noted that "**the iStent has a synergistic effect with phacoemulsification.**" This echoes the results of two prior meta-analyses, adding to this study's validity.

The authors added that in their subgroup analysis excluding the iStent inject, IOP reduction was less following combined surgery. However, the change in glaucoma eye drops remained the same. The authors concluded, "Although the results were statistically significant with iStent showing a good safety profile and offering the benefit of decreased dependence on glaucoma eye drops, the clinical implications are debatable. Studies still need to assess the long-term IOP-lowering effect of iStent, the protective effect on the optic nerve and how long it delays the need for future, more invasive glaucoma surgeries."

Kahale F, Chanbour W, El Zein L, et al. Phacoemulsification with and without iStent: a systematic review and meta-analysis of comparative studies. Ophthalmic Res. May 26, 2023. [Epub ahead of print].

Less Frequent DR Screenings May Suffice After Age 80

Study found disease progression and need for treatment quite uncommon in this population.

Due to the heightened risk of retinopathy in patients with diabetes, it's recommended that those with the disease receive eye examinations annually. More recent research, however, suggests that screening may only be necessary once every two years in patients with the lowest risk of diabetes-related sight loss. In 2020, Public Health England introduced these extended intervals.

To assess whether patients aged 80 to 90 fall into this low-risk category and can safely receive exams less frequently than every 12 months, researchers performed a five-year study on patients being monitored for diabetic retinopathy (DR) in digital surveillance clinics. Included were 1,880 patients aged 80 and 1,105 patients aged 85 at baseline. In follow-up, 21% and 49% of the 80- and 85-year-old cohorts died, respectively.

Between 0.7% and 1.4% of the 80-year-old cohort was referred to a hospital eye service for DR each year. Over five years, a total of 4% of the

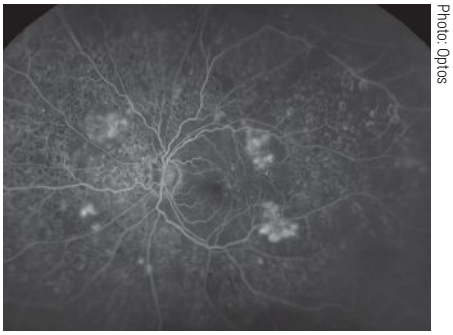


Photo: Optos

Over five years, only 4% of 80-year-olds and 2.4% of 85-year-olds in this study were referred to a hospital DR, and less than 1% received treatment.

80-year-old patients were referred to the hospital for DR, and a mere 0.6% of those received treatment. Similar but slightly lower numbers were reported in the 85-year-old cohort. Between 0.1% and 1.3% of these patients were referred each year to a hospital eye service for DR. A total of 2.4% of 85-year-olds were referred throughout the course of follow-up, and of those, only 0.4% were treated.

All patients in both cohorts who received treatment did so for maculopathy. No cases of proliferative DR requiring treatment were reported.

"This study showed that the risk of progression of retinopathy over five years is quite low in this elderly age group (80 to 90), and only a small proportion of patients developed referable retinopathy requiring any treatment," the researchers summarized in their paper on the study, published in the journal *Eye*. "This raises the question of the need for annual diabetic eye screening in this age group."

The authors bring up the point that screening attendance rates tend to decline as age increases, something that extended screening intervals may help address. The cost-effectiveness of less frequent exams is another potential benefit to patients.

The researchers concluded that this new data calls for a review of current guidance on screening recommendations for elderly patients with diabetes and no signs of retinopathy.

Thomas K, Albutt N, Hamid A, et al. Five-year outcomes of digital diabetic eye screening in individuals aged 80 and 85 years. Eye. May 20, 2023. [Epub ahead of print].

Macrolides Safer, More Effective, than Tetracyclines for MGD

With growing bacterial resistance to antibiotics on one hand and the need for effective affordable treatment modalities for meibomian gland dysfunction (MGD) on the other, it is crucial to determine the most effective and safe antibiotic treatment for MGD. A recent systematic review and meta-analysis addressed this issue by comparing oral macrolide with tetracycline antibiotic treatment for the condition. While both were effective, macrolides exhibited better efficacy and safety.

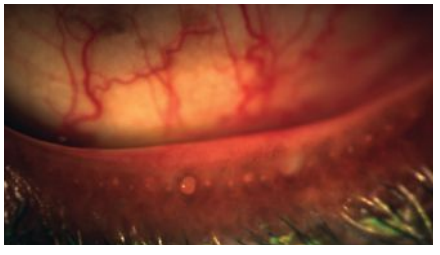


Photo: Doan Huynh Kwak, OD

Macrolides were superior to tetracyclines in both efficacy and safety when used for MGD management.

Six prospective studies were ultimately included for analysis, which reported on 563 cases from three countries. Affected patients with moderate to severe MGD were between ages 12 and 90.

In pooled analysis, macrolides were significantly superior in the total signs score (pooled standardized mean difference [SMD]: -0.51), meibomian gland secretion score (pooled SMD: -0.25), TBUT (SMD: -0.31) and fluorescein staining score (SMD: -1.01). While no severe complications were reported for both treatments, the macrolide group exhibited significantly fewer adverse events (pooled odds ratio: 0.24).

"The analysis demonstrated a significant superiority of the macrolides in both efficacy and safety when used for MGD management," the researchers wrote. "We believe that these results may aid clinicians in choosing their first-line treatment strategy for MGD patients in the future."

Ben Ephraim Noyman D, Chan CC, Mimouni M, Safir M. Systemic antibiotic treatment for meibomian gland dysfunction-A systematic review and meta-analysis. Acta Ophthalmol. May 4, 2023. [Epub ahead of print].

When Selecting a Prescription Dry Eye Treatment

DON'T MAKE HER WAIT.

Not an actual patient.

Indication

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

Important Safety Information

- Xiidra is contraindicated in patients with known hypersensitivity to lifitegrast or to any of the other ingredients.
- In clinical trials, the most common adverse reactions reported in 5-25% of patients were instillation site irritation, dysgeusia and reduced visual acuity. Other adverse reactions reported in 1% to 5% of the patients were blurred vision, conjunctival hyperemia, eye irritation, headache, increased lacrimation, eye discharge, eye discomfort, eye pruritus and sinusitis.
- To avoid the potential for eye injury or contamination of the solution, patients should not touch the tip of the single-use container to their eye or to any surface.

NOVARTIS

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East Hanover, New Jersey 07936-1080

xiidra® (lifitegrast ophthalmic solution)5%

CHOOSE XIIDRA

Because lasting symptom relief can start as early as 2 WEEKS[1]*

Access to Xiidra is better than ever[2]

*Xiidra reduced symptoms of eye dryness at 2 weeks (based on Eye Dryness Score compared to vehicle) in 2 out of 4 studies, with improvements observed at 6 and 12 weeks in all 4 studies.[1†]

Important Safety Information (cont)

- Contact lenses should be removed prior to the administration of Xiidra and may be reinserted 15 minutes following administration.
- Safety and efficacy in pediatric patients below the age of 17 years have not been established.

Please see Brief Summary of Important Product Information on adjacent page.

†Pivotal trial data
The safety and efficacy of Xiidra were assessed in four 12-week, randomized, multicenter, double-masked, vehicle-controlled studies (N=2133). Patients were dosed twice daily. **Use of artificial tears was not allowed during the studies.** The study end points included assessment of signs (based on Inferior fluorescein Corneal Staining Score [ICSS] on a scale of 0-4) and symptoms (based on patient-reported Eye Dryness Score [EDS] on a visual analogue scale of 0-100).[1]

Effects on symptoms of dry eye disease: A larger reduction in EDS favoring Xiidra was observed in all studies at day 42 and day 84. Xiidra reduced symptoms of eye dryness at 2 weeks (based on EDS) compared to vehicle in 2 out of 4 clinical trials.[1]

Effects on signs of dry eye disease: At day 84, a larger reduction in ICSS favoring Xiidra was observed in 3 of the 4 studies.[1]

References: 1. Xiidra [package insert]. East Hanover, NJ: Novartis Pharmaceuticals Corp. **2.** Data on file. DRF Fingertip Formulary® Novartis Pharmaceuticals Corp; July 2022.

XIIDRA® (lifitegrast ophthalmic solution), for topical ophthalmic use
Initial U.S. Approval: 2016

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

1 INDICATIONS AND USAGE

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

4 CONTRAINDICATIONS

Xiidra is contraindicated in patients with known hypersensitivity to lifitegrast or to any of the other ingredients in the formulation *[see Adverse Reactions (6.2)]*.

6 ADVERSE REACTIONS

The following serious adverse reactions are described elsewhere in the labeling:

- Hypersensitivity *[see Contraindications (4)]*

6.1 Clinical Trials Experience

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

In five clinical trials of DED conducted with lifitegrast ophthalmic solution, 1401 patients received at least one dose of lifitegrast (1287 of which received lifitegrast 5%). The majority of patients (84%) had less than or equal to 3 months of treatment exposure. One hundred-seventy patients were exposed to lifitegrast for approximately 12 months. The majority of the treated patients were female (77%). The most common adverse reactions reported in 5%-25% of patients were instillation-site irritation, dysgeusia, and reduced visual acuity.

Other adverse reactions reported in 1%-5% of the patients were blurred vision, conjunctival hyperemia, eye irritation, headache, increased lacrimation, eye discharge, eye discomfort, eye pruritus, and sinusitis.

6.2 Postmarketing Experience

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

Rare serious cases of hypersensitivity, including anaphylactic reaction, bronchospasm, respiratory distress, pharyngeal edema, swollen tongue, urticaria, allergic conjunctivitis, dyspnea, angioedema, and allergic dermatitis have been reported. Eye swelling and rash have also been reported *[see Contraindications (4)]*.

8 USE IN SPECIFIC POPULATIONS

8.1 Pregnancy

Risk Summary

There are no available data on Xiidra use in pregnant women to inform any drug-associated risks. Intravenous (IV) administration of lifitegrast to pregnant rats, from premating through gestation day 17, did not produce teratogenicity at clinically relevant systemic exposures. Intravenous administration of lifitegrast to pregnant rabbits during organogenesis produced an increased incidence of omphalocele at the lowest dose tested, 3 mg/kg/day (400-fold the human plasma exposure at the recommended human ophthalmic dose [RHOD], based on the area under the curve [AUC] level). Since human systemic exposure to lifitegrast following ocular administration of Xiidra at the RHOD is low, the applicability of animal findings to the risk of Xiidra use in humans during pregnancy is unclear *[see Clinical Pharmacology (12.3) in the full prescribing information]*.

Data

Animal Data

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

8.2 Lactation

Risk Summary

There are no data on the presence of lifitegrast in human milk, the effects on the breastfed infant, or the effects on milk production. However, systemic exposure to lifitegrast from ocular administration is low *[see Clinical Pharmacology (12.3) in the full prescribing information]*. The developmental and health benefits of breastfeeding should be considered, along with the mother's clinical need for Xiidra and any potential adverse effects on the breastfed child from Xiidra.

8.4 Pediatric Use

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

8.5 Geriatric Use

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

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REVIEW of OPTOMETRY
Leadership in clinical care

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FEATURES

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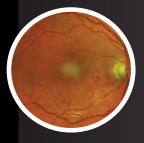

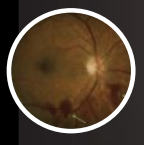

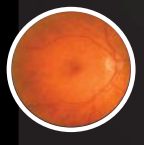

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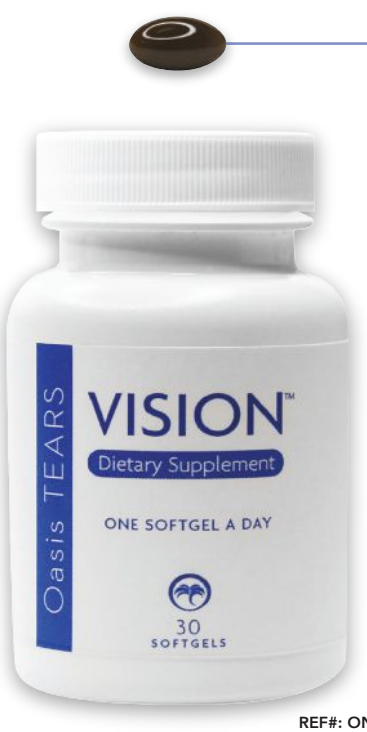

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ZEAXANTHIN (5mg) & LUTEIN (25mg)

Forms the macular pigment in the retina with the chemical structure that absorbs and filters blue light, protecting underlying cell layers from oxidative damage.[4]

ASTAXANTHIN (6mg)

Found in red algae and is a carotenoid, similar in structure to Lutein and Zeaxanthin while demonstrating stronger antioxidant activity in restoring cells after UVA light damage.[5]

DHA (Docosahexaenoic Acid) (200mg)

Required for the process of transforming light into an electrophysiological signal and for the regeneration of the light sensitive pigment in the retina – rhodopsin.[6]

1. Nakamura S, Tanaka J, Imada T, Shimoda H, Tsubota K. Delphinidin 3, 5-O-diglucoside, a constituent of the maqui berry (Aristotelia chilensis) anthocyanin, restores tear secretion in a rat dry eye model. Journal of functional foods. 2014;10:346-354.
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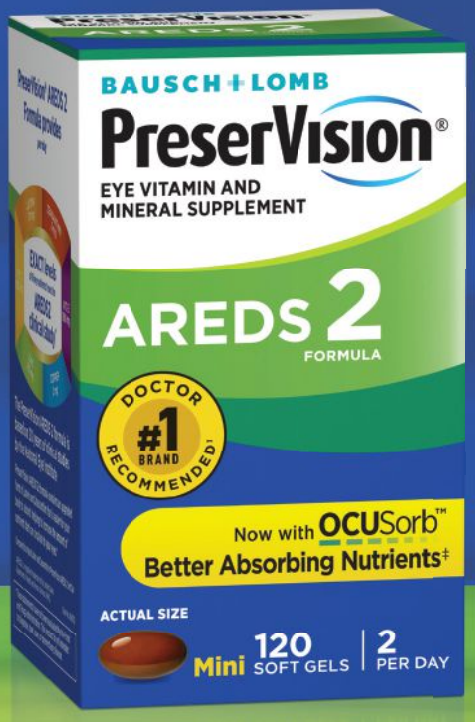

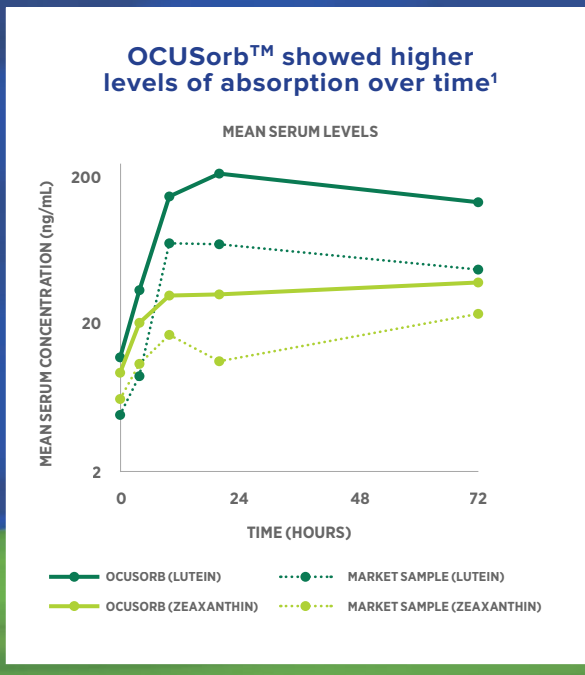

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NEI = National Eye Institute

1. Compared to the market sample. Kotagiri SR, Morde A, Rai D, et al. Ophthalmol Ther. 2022;11(4):1463-1477

‡ Compared to original lutein and zeaxanthin in PreserVision AREDS 2 Formula Soft Gels

*This statement has not been evaluated by the Food and Drug Administration. This product is not intended to diagnose, treat, cure or prevent any disease.

REVIEW of OPTOMETRY

Founded 1891

BY JACK PERSICO
EDITOR-IN-CHIEF
OUTLOOK

Retina on the Rise

New meds, methods and motivations are bringing it to the forefront of optometric care.

When I started writing about optometry in the early 1990s, the bread and butter of the field was refractions, dispensing and anterior segment care. There was also a growing recognition that glaucoma was a good fit for optometry and would become integral to practice once the state laws finally came around. Retina, however, was still kind of a niche. Beyond performing basic posterior segment exams with a binocular indirect, there wasn't much an OD could do.

Optometric retina luminaries of the 1990s like Jerry Sherman, Lou Catania and Larry Alexander worked diligently to elevate the profession's capabilities and interest in retinal disease, but it was never quite an equal partner to anterior segment care back then. OCT was brand new and only made sense for researchers and subspecialists. Anti-VEGF was still over a decade away. The AREDS study had begin recruiting but wouldn't finish until 2001.

Fast forward three decades and various trends are moving retinal disease assessment from niche pursuit to mainstream optometry.

Start with the elephant in the room: optometry's two-to-one advantage over ophthalmology. A growing and aging population needs ever more health care, and stagnation in the population of ophthalmologists does nothing to quell demand. More patients need primary eye care every year, and that can only happen in optometry offices.

In 2023, OCT is now an essential device in optometry as well as ophthalmology, giving ODs access to the single most significant tool for retinal disease assessment. The much-vaunted coming wave of AI-powered diagnostic devices will only help demystify retinal conditions even more and give optometrists more reliable diagnostic resources for disease identification and long-term follow-up.

Probably the biggest catalyst right now for optometry's more prominent role in retina is the emergence of therapies for geographic atrophy. With one drug on the market and another expected later this year, GA is suddenly a hot topic. There's finally something to recommend—but the strike zone for what constitutes a viable candidate is rather small. Patients have to be well selected and properly educated on expectations and outcomes. That's not just an invitation to optometry, it's practically an imperative to act. ODs see the lion's share of primary eye care patients and will be the first-responders who screen, identify, triage and prep GA patients for what's to come.

This parallels a similar impetus in diabetic retinopathy screening, as the 2021 Panorama study found value in prophylactic treatment of severe NPDR as a means of preventing vision loss. Again, it's the ODs who are out there in the trenches finding these patients day in and day out.

Lastly, today's new optometry grads come out of school fully read in on retina. It's as much a part of their world as anything else.

Add it all up and it's clear that optometric retina care is rife with opportunity. I wish there were stronger financial incentives—or, to put it more equitably, I wish insurers valued your role commensurate with the societal gain it represents. You may never make a killing in retina, but you can really make a difference. ■

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References: 1. Walkden A. Amniotic membrane transplantation in ophthalmology: an updated perspective. *Clin Ophthalmol*. 2020;14:2057-2072. **2.** Craig JP, Nichols KK, Akpek EK, et al. TFOS DEWS II definition and classification report. *Ocul Surf.* 2017;15(3):276-283. **3.** Jones L, Downie LE, Korb D, et al. TFOS DEWS II management and therapy report. *Ocul Surf.* 2017;15(3):575-628.

*There are no specific FDA indications for the product.

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Théa
let's open our eyes

BY PAUL M. KARPECKI, OD
CHIEF CLINICAL EDITOR

AMD from A to Z

Get familiar with each stage and the available treatments.

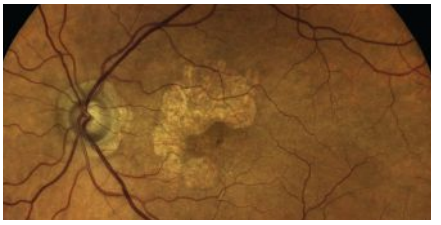
With the recent approval of Syfovre (pegcetacoplan injection, Apellis Pharmaceuticals) for dry age-related macular degeneration (AMD)/geographic atrophy (GA), we have a great opportunity to lead and individualize patient management. Each stage has various treatment options and knowing them may prevent permanent vision loss. Let's review the stages and options, as well as the symptoms to look for and the most effective instruments to use.

Early AMD

This can be a difficult stage to identify, as few visual cues may be present. For many patients, the only symptoms might include difficulty with driving at night (contrast sensitivity loss) or difficulty recovering vision after entering a darkened room such as a theater (dark adaptation). Testing may consist of contrast sensitivity testing (M&S Technologies) or dark adaptation (AdaptDx Pro, Maculogix). Management focuses on lifestyle changes—smoking cessation, improving nutrition and taking formulations such as vitamins (*e.g.*, Ocuvite, MacuHealth) or whole body nutrition supplements with antioxidants (*e.g.*, OcularProtect, ProMacular Defense).

Intermediate AMD

Symptoms sometimes increase at this stage, affecting reading and even visual acuity. Research has shown that AREDS II formulations (*e.g.*, PreserVision) slow progression. Patients should be monitored regularly for wet AMD changes (*e.g.*, ForeseeHome, Notal Vision) or GA development through more frequent OCT and high-resolution color fundus photography with blue-light fundus autofluorescence (FAF) imaging (*e.g.*, iCare, Eidon). Higher risk of conversion to neovascular AMD may be indicated by drusenoid material that are large, soft or subretinal.



GA, easily identified with the iCare Eidon Widefield TrueColor Confocal scanner.

Neovascular/Wet AMD

Fluid under the macular is best observed via OCT as either intraretinal or subretinal fluid, with pigment epithelial detachments. *En face* imaging may help visualize the neovascular net. OCT is critical at this stage and referral to a retina specialist for anti-VEGF injections is required.

GA

The approval of Syfovre and a pending approval later this year for Zimura (Iveric Bio) for a second complement system inhibitor gives hope to patients with dry AMD. These therapeutics were shown to slow progression of GA, but the hope is that after slowing progression of the devitalized cells/photoreceptors/retinal pigment epithelium (RPE), healthy cells in adjacent areas will avoid atrophy altogether. It is up to us to determine when to refer patients to a retina specialist for these injections.

While most GA occurs in patients with dry AMD, candidates also include patients with wet AMD who have been treated with anti-VEGF and experience significant drying. Starting with a 90D or 78D condensing lens or fundus photography helps determine if FAF or OCT should be ordered. FAF can help identify GA lesions through hyperfluorescence. Certain GA patterns, such as a patchy pattern (lesions greater than 200µm) or a diffuse pattern, are more prone to faster progression.

OCT is yet another effective way to determine ideal GA candidates, which show hypertransmission—like a bar code pattern—indicating atrophic areas of absent RPE. Patients with fovea-sparing lesions are excellent candidates, although they may not be identified due to maintaining good vision. Patients with central GA may still make good candidates if it's not late-stage AMD.

Late-stage AMD

If the patient has passed the point of treatment options such as Syfovre, there's still hope. From a surgical referral standpoint, there is the implantable miniature telescope (Samsara Vision), which is an IOL for cataract lens replacement that magnifies the image 2.7x, obtaining three to four lines of vision on average. A spectacle option includes AR technology that moves the object off the central macular scotoma (Eyedaptic). Finally, if none of these are possible, such as a patient who's already had cataract surgery, refer them to a low vision specialist. ■

About Dr. Karpecki

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

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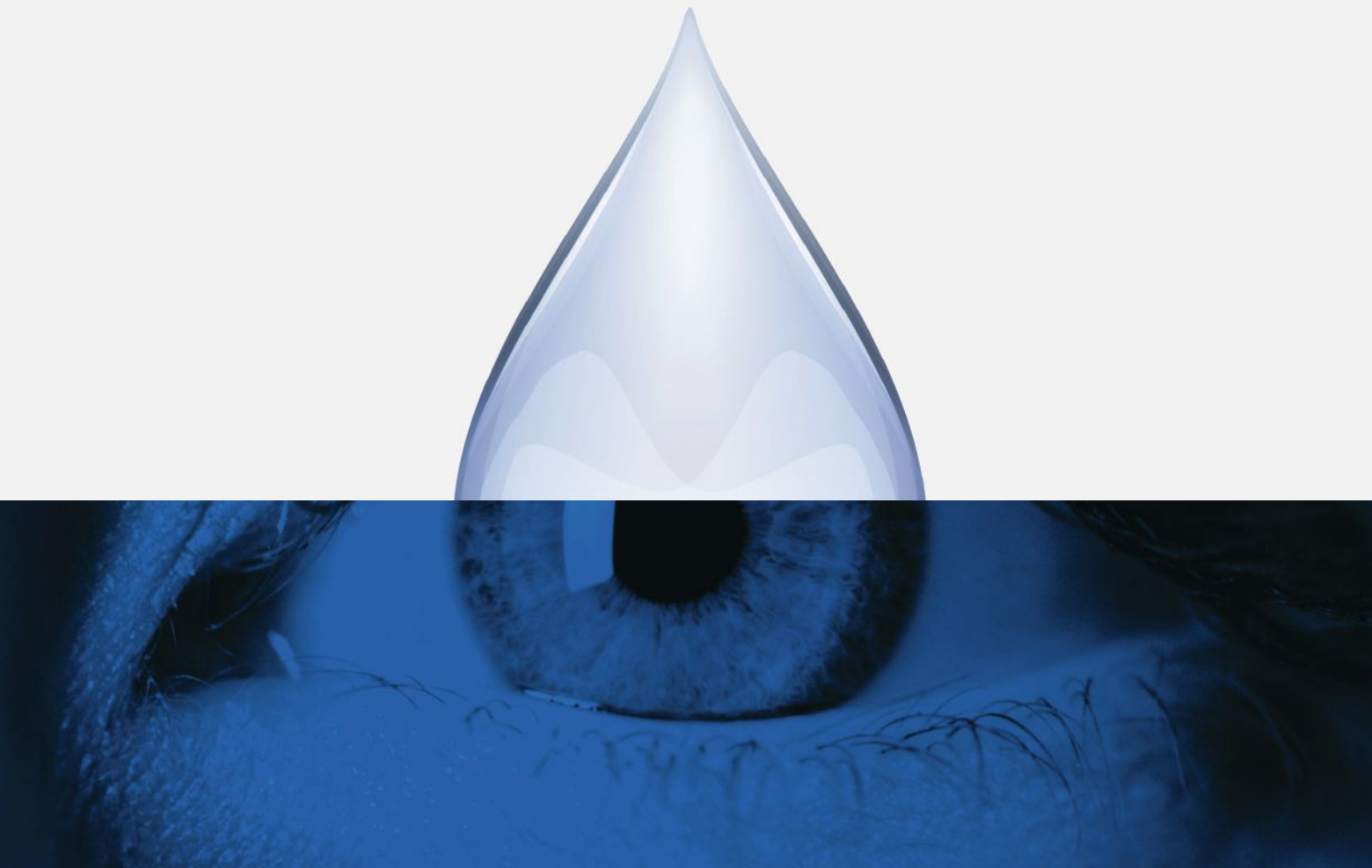

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let's open our eyes

PRC-PSaF-1092-v1 05.2023

BY MONTGOMERY VICKERS, OD

CHAIRSIDE

You Say Weakness, I Say Strength

The two might just be one and the same.

I guess it must be obvious that the Greek philosopher Plato and I have a lot in common. Those of you with a liberal arts undergrad education may recall that "Plato" was actually a nickname that referred to his physical form, which was considered kind of broad. FYI, it is a little known fact that Plato's given name was Steve. He was just built, well, broad. My own broadness is a product of fast-food America.

> **Spend your time on your greatest strength. That's the best way to succeed. Your greatest weakness will not need your time and effort.**

So, Plato and I are certain that an optometrist's greatest strength is also his or her greatest weakness… two sides of the same coin, you might say. There are many examples of this in our profession. Here are a few:

1. We are almost always nice. This is why we give lots of stuff and our precious time away like crazy. We want to be liked. The problem is that people don't actually like us for this… they just innocently take advantage of us. I think 10 office visits in, they should pay us. That's as nice as I can be. I agree. That's too nice, too.

2. We continually want to learn new things. The problem is that it is impossible to constantly learn everything new without forgetting that the patient just wants to see their computer a little better. Maybe we should spend more time on that instead of memorizing the chemical composition of every eye drop ever produced.

3. We are obsessive about cleanliness. This is how superbugs were invented, you know. The worst place in the world to be sick is in a hospital thanks to the massive colonies of killer germs. No wonder all the kids these days are weird… too much bleach inhalation. Want them healthy? Let them get dirty sometimes.

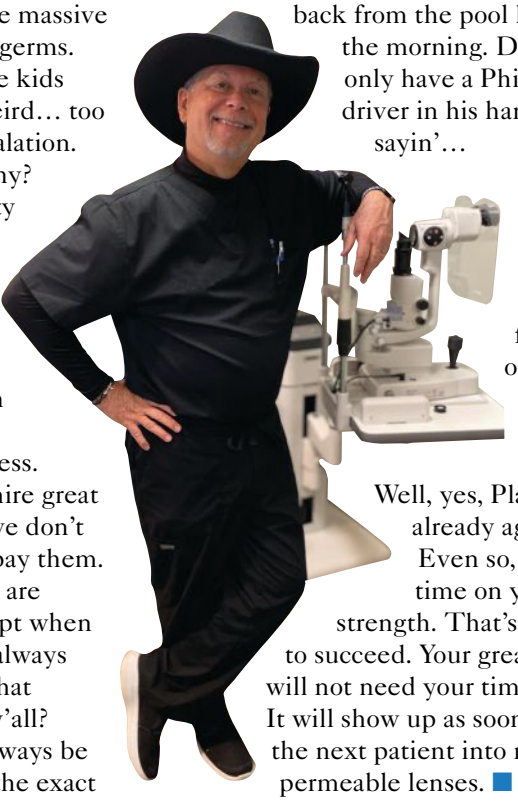
4. We love CE meetings. We hate CE meetings. I'll let you decide which is a strength and which is a weakness.

5. We want to hire great employees, but we don't actually want to pay them.

6. We think we are always right except when we think we are always wrong. Where's that happy medium, y'all? Think you can always be perfect? You are the exact wrong species. Please refer to Ted Lasso's goldfish speech in season one.

7. We think we completely understand all the ins and outs of each and every vision insurance plan. Okay, if you completely understand that these programs are designed to, first and foremost, benefit the vision plan and then, maybe somehow, a patient can benefit a little bit, then maybe you are wise indeed. How do they feel about you, the doctor? You are their greatest strength and their greatest weakness.

8. We think it's always good to make patients see as well as you can. This is not only a strength but also a weakness because we simultaneously assume a single pair of glasses or contact lenses can be just as good, if not better, staring at a screen as they are when the patient is driving back from the pool hall at two in the morning. Does a carpenter only have a Phillips screwdriver in his hand all day? Just sayin'…



What is your greatest strength? I truly believe it's good to figure that out if you can. Could it also be your greatest weakness? Well, yes, Plato and I have already agreed on that. Even so, spend your time on your greatest strength. That's the best way to succeed. Your greatest weakness will not need your time and effort. It will show up as soon as you fit the next patient into multifocal gas permeable lenses. ■

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

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‡To limit blurriness when using contact lenses, remove contacts, apply drops, then insert contacts.

Reference: 1. Data on file.

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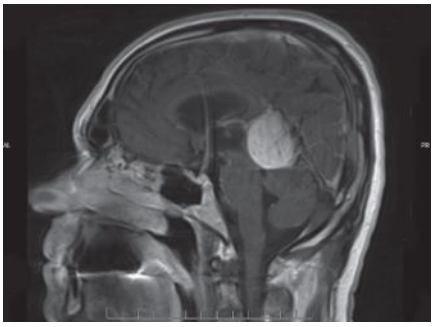
BY MARC B. TAUB, OD, MS, EdD, AND PAMELA H. SCHNELL, OD

Different Fields for Different Folks

Kinetic testing may prove more beneficial than static for brain-injured patients.

The occurrence of an acquired brain injury (ABI) can be life-altering. The damage can be mental, emotional, cognitive and, of course, physical. The visual system can be impacted in a number of ways, including difficulty with eye teaming, focusing and tracking, double vision, visual information processing and visual field loss. According to a meta-analysis of 22 eligible publications, three visual conditions were identified as being commonly associated with traumatic brain injury (TBI).[1] Random-effects models yielded the following combined prevalence estimates: accommodative dysfunction (42.8%), convergence insufficiency (36.3%) and visual field loss (18.2%). Another study reported many other visual dysfunctions associated with ABI, including loss of color discrimination, brightness detection and contrast sensitivity, visuospatial attention deficits with a slower response to visual cues, nystagmus, reading problems, saccadic and pursuit disorders and photosensitivity.[2]

In other columns, we've covered the topics of photosensitivity and double vision, but it is time to address the issue of visual field testing in patients suffering from ABI or TBI. As we all learned in optometry school, and perhaps in CE lectures, there are numerous methods for assessing visual fields, including confrontation fields, tangent screening, static perimetry (*e.g.*, the Humphrey visual field by Zeiss) and kinetic perimetry (*e.g.*, the Goldmann visual field).



Falcotentorial meningioma near the pineal region.

The Goldmann is the gold-standard kinetic perimeter, since it establishes peripheral and central defects and correlates better than other options with activities of daily living.[3] Unfortunately, in addition to the fact that the Goldmann equipment takes up significant real estate in the office due to its size, it requires a skilled technician to perform. It doesn't help that the devices are no longer being manufactured. The Octopus 900 by Haag-Streit, an alternative kinetic perimeter, also provides a full 90° field, but it is automated. The advantage here is an improvement in test-retest reliability and a decrease in testing time. A study of 26 eyes from a neuro-ophthalmology clinic comparing the Goldmann with the Octopus found that 88.5% of subjects were correctly matched for normal or abnormal visual fields, and 89% of quadrants matched between the two tests.[4]

The same study found that 88.5% of subjects were correctly matched for normal or abnormal visual fields between the Octopus and the Humphrey static perimeter. A total of 80% of quadrants matched between these two tests. Despite the seemingly positive correlation in the results, we have not seen the same findings personally. We include one of our own cases highlighting the use of two systems in a brain-injured patient to demonstrate why we prefer the kinetic (Octopus) over the static (Humphrey) visual field.

Case Report

A 63-year-old white male presented to the Advanced Care Ocular Disease Service at SCO post-surgery for a left occipital craniotomy due to a falcotentorial meningioma near the pineal region two months prior. Immediately following the surgery, the patient complained of right-sided visual field loss, which had mostly returned by the time of the examination. He was concerned about driving again and was instructed to get an evaluation by his neurosurgeon. He was taking only two medications (allopurinol and bupropion HCl) and was otherwise healthy. His visual acuity was 20/20 OD, OS and OU, and his pupils were equal, round and reactive to light with no APD. Confrontation fields showed mild constriction on the right side OU. Out of caution, OCTs of the macula and optic nerve were completed and found to be normal. A 24-2 SITA Fast visual field showed a right homonymous hemianopsia that was denser superiorly, with macular sparing inferiorly, but not absolute. One thing to note was that

About Drs. Taub and Schnell

Dr. Taub is a professor, chief of the Vision Therapy and Rehabilitation service and co-supervisor of the Vision Therapy and Pediatrics residency at Southern College of Optometry (SCO) in Memphis. He specializes in vision therapy, pediatrics and brain injury. **Dr. Schnell** is an associate professor at SCO and teaches courses on ocular motility and vision therapy. She works in the pediatric and vision therapy clinics and is co-supervisor of the Vision Therapy and Pediatrics residency. Her clinical interests include infant and toddler eye care, vision therapy, visual development and the treatment and management of special populations. They have no financial interests to disclose.

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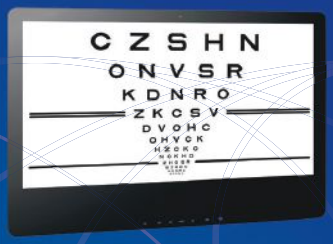

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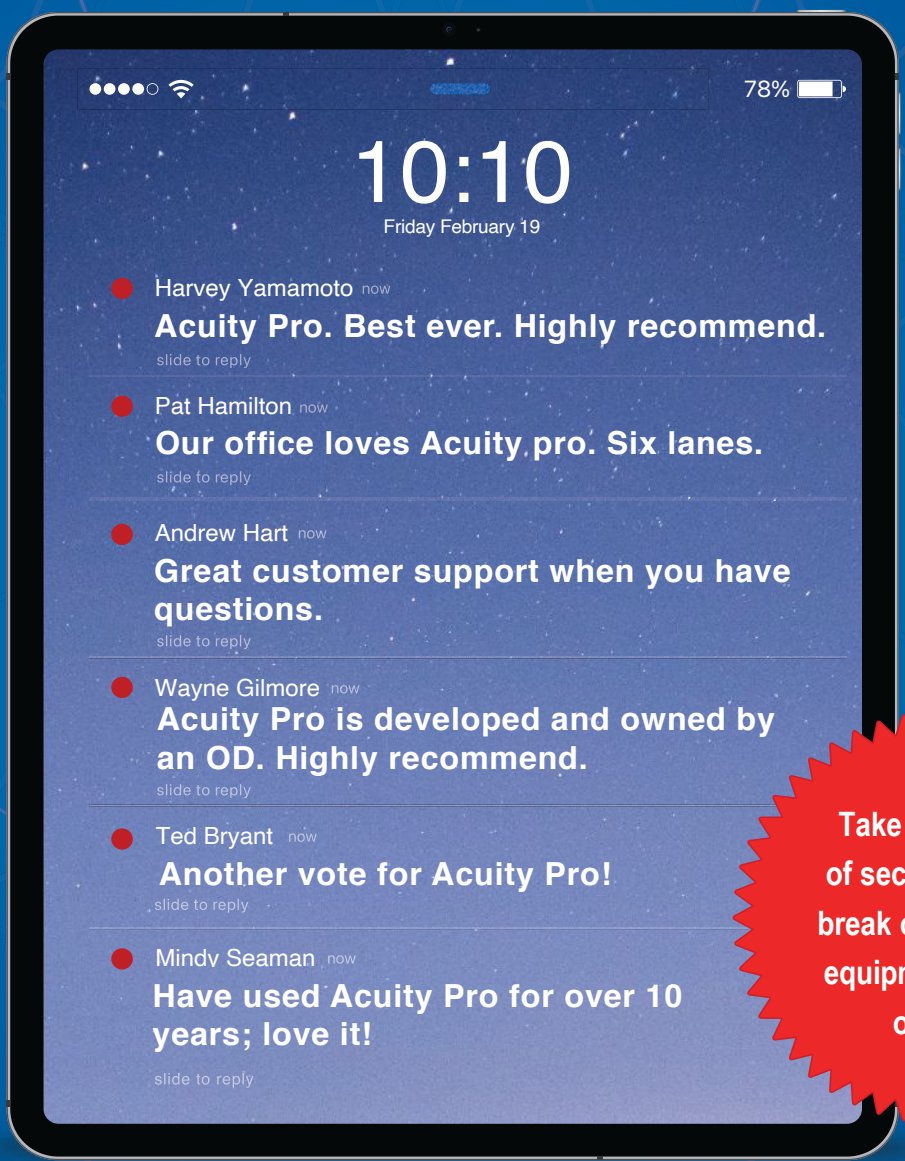

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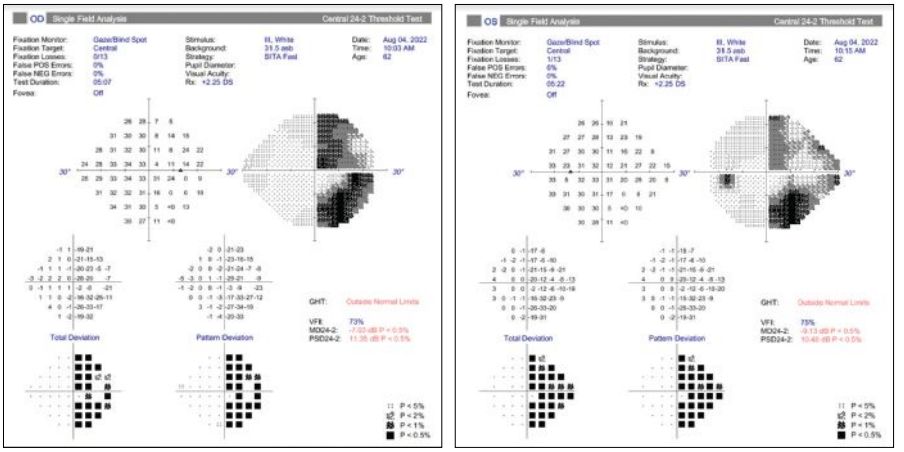


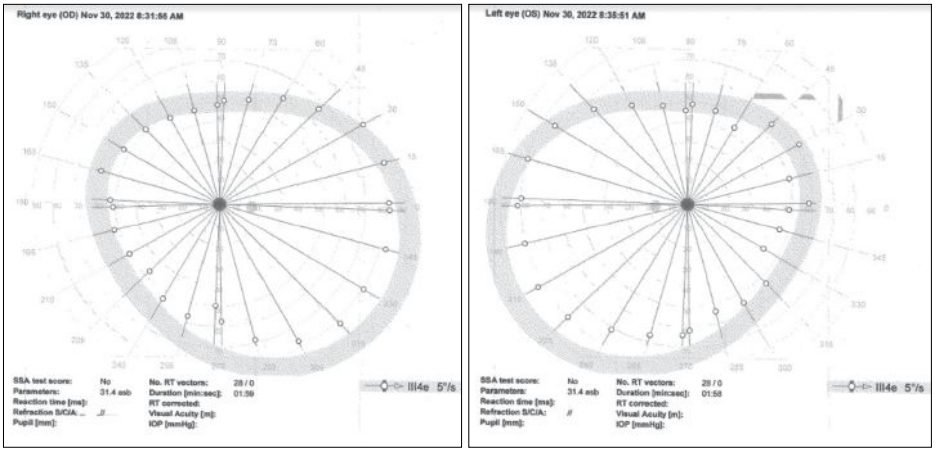
24-2 Humphrey visual fields showing non-absolute, right homonymous hemianopsia, denser superiorly with macular sparing inferiorly.

the fixation losses OD and OS were quite high. The patient was counseled on the field loss and educated on and referred for vision therapy.

He reported several weeks later to the Vision Therapy and Rehabilitation Service with the same complaints as in the previous exam, with no improvement. The patient did not complain of diplopia, trouble with bumping into things on the right side or issues with balance. The chair skills were the same, including acuity and pupils. The fields by confrontation showed only right-sided constriction. A kinetic field on the Octopus using a III 4e target at 5°/sec showed slight constriction on the right side in both eyes: approximately 10% and 15% constricted in the right and left eyes, respectively.

Based on the kinetic visual field and the patient's minimal complaints, it was determined that vision therapy would be the best treatment option to assist him in learning some basic scanning concepts into far-right gaze, as well as to improve his visual attention to that side. Keep in mind that his ultimate goal was to get back behind the wheel. The plan was for the patient to complete 10 vision therapy sessions and then reassess his readiness for a driving evaluation by a specialized occupational therapist.

The therapy focused on peripheral attention and awareness and incorporated both free-space and computer-based activities. As with any therapy activity, feedback is crucial. The patient must know how they are performing so they can make adjustments and learn. Any opportunity to employ the other senses, particularly auditory and tactile, will assist the patient in creating new neurological pathways.



Octopus kinetic visual field using a III 4e target at 5°/sec showing only slight constriction.

After 10 sessions, the patient's kinetic field showed slight improvement, and he was asymptomatic. He was referred for driving evaluation and was thrilled when he passed with flying colors.

Discussion

In this case, the two types of visual fields, kinetic and static, produced very different results. Even though there were several weeks between the tests, it is unlikely that the homonymous hemianopsia detected on the static visual field test dissipated that quickly. The two tests are quite different. The kinetic field measures 180° (technically 150° due to physical/physiological limitations), while the static field measures only 24°. The kinetic field has a constant target size and illumination, while the static field presents the illumination at different intensities to find the level of detection. The static field flashes the target for 0.2 seconds, while the kinetic target is constant. For patients who have trouble with attention and perhaps poor ocular motility, the quick flash of the target can be problematic and can potentially lead to poorer results.

Over our careers, we have seen countless patients who perform quite differently on the two visual field types. When evaluating brain injury patients, including those with neurologic disease, we have found that the kinetic field is more faithful to the true defect and provides a better understanding of the defect, especially in the periphery. For this reason, we recommend that practitioners who see a significant number of ABI or TBI patients in their offices consider using this valuable tool for a better visual field assessment. ■

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The Benefit of Biologics

These treatment options can be useful against diabetic peripheral corneal neuropathy.

Q I have a patient with persistent epithelial defects consistent with neurotrophic keratitis (NK). She has failed on traditional treatments such as steroids and punctal plugs. What else can I do?

A "There are many safe and effective biologic treatments for NK including amniotic membrane, Oxervate (cenegermin 0.002%, Dompe), autologous serum eye drops (ASEDs) and platelet-rich plasma (PRP)," says Hardeep Kataria, OD, of Avant Eyes Optometry & Advanced Dry Eye Center in Porter Ranch, CA. "Early NK is often misdiagnosed as moderate to severe dry eye disease due to its similarity in presentation with persistent superficial punctate keratitis (SPK)," she notes. "However, one test can differentiate dysfunctional corneal nerves: corneal sensitivity."

A Sensitive Topic

A 66-year-old woman presented to Dr. Kataria with a longstanding history of systemic hypertension and uncontrolled type 2 diabetes with moderate nonproliferative diabetic retinopathy and diabetic macular edema (DME) in both eyes. Her medication list included insulin and antihypertensives. She had a history of intravitreal Avastin (bevacizumab, Genentech) injections and focal laser for DME.

She presented with a chief complaint of blurred vision and SPEED score of 3/28, with infrequent and mild irritation. There was no active macular edema. She admitted to having seen multiple providers who had prescribed low-dose topical steroids, cyclosporine, lifitegrast and a history of punctal plugs without improvement. She had also undergone two thermal evacuation (Lipiflow, Tear Sciences) and microblepharoexfoliation (ZEST, Zocular) procedures for severe atrophy of her meibomian glands and presence of blepharitis. Her best-corrected vision was 20/30 in each eye.

> **Amniotic membranes work well as induction therapy until a patient is approved for Oxervate.**
>
> **—Hardeep Kataria, OD**

Anterior segment examination revealed a persistent inferior epithelial defect OD and confluent SPK inferior and central OS (*Figure 1*). Corneal sensitivity testing with a cotton tip applicator revealed corneal anesthesia OD and hypoesthesia OS. "This corneal sensitivity points us in the direction of NK," Dr. Kataria says.

One cryopreserved amniotic membrane was placed in each eye for three days with a temporary tape tarsorrhaphy (*Figure 2*). Both corneas appeared healed immediately after treatment. "Due to the progressive nature of NK, we knew that long-term treatment would be required," Dr. Kataria notes.

Oxervate was ordered, and the patient was compliant for the typical eight weeks of treatment. The epithelial defects completely resolved, and the patient is being monitored on artificial tears.

Striking a Nerve

NK is a progressive degenerative disease of the corneal epithelium and stroma caused by damage to the corneal nerves. Maintenance of ocular surface health highly depends on a complex neuroanatomic pathway that innervates the corneal epithelium and corneal nerves.

"This mutual support promotes cellular proliferation, migration and

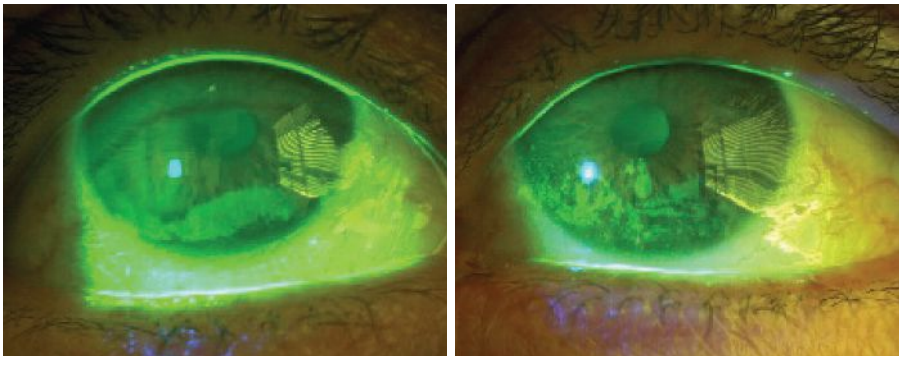

Fig. 1. OD persistent inferior corneal epithelial defect, OS persistent confluent inferior and central SPK.

About Dr. Ajamian **Dr. Ajamian** is board certified by the American Board of Optometry and serves as Center Director of Omni Eye Services of Atlanta. He is vice president of the Georgia State Board of Optometry and general CE chairman of SECO International. He has no financial interests to disclose.

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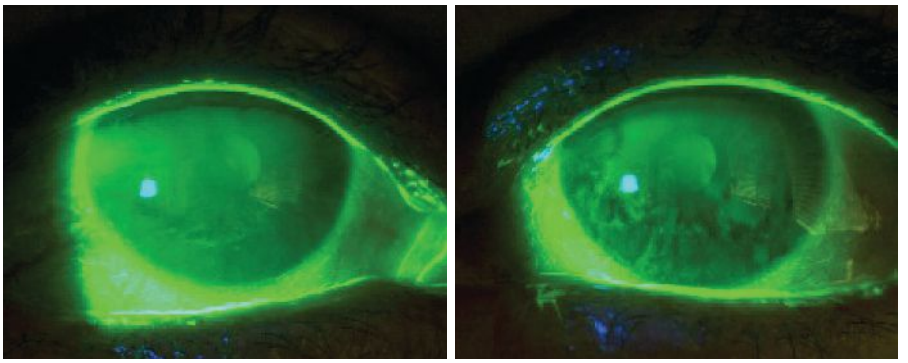

Fig. 2. At left, OD improvement of epithelial defect after amniotic membrane. At right, OS improvement of confluent SPK after amniotic membrane.

differentiation and corneal nerve repair," Dr. Kataria emphasizes.

Diabetic peripheral neuropathy is often seen with hyperglycemia that causes neurodegeneration and apoptosis of neuronal cells. It affects 50% of patients with diabetes, and the trigeminal nerve is no exception.[1] Patients with diabetes can also present with reduced corneal nerve density as imaged on *in vivo* confocal microscopy, further increasing suspicions that NK can occur frequently in patients with diabetes.[2]

Need for Growth

Neurotrophic keratitis occurs when the corneal epithelium is persistently irregular and cannot support the corneal nerves, or vice-versa. Treatments restore the supply of neurotrophic factors to the corneal nerves and epithelium, specifically nerve growth factors. Therefore, treatments that are rich in nerve growth factors may work best, such as amniotic membrane, ASEDs, PRP and recombinant human nerve growth factor, Oxervate.

Treatment goals for early NK include promoting eyelid closure, preventing tear evaporation and improving the health of the corneal epithelium and corneal nerves. Dr. Kataria recommends using steroids and NSAIDs with caution so as to not promote superinfection and corneal melt, respectively. Punctal plugs can increase the tear lake and prevent evaporation. "Amniotic membranes, rich in nerve growth factors, are considered a first-line treatment," she states.

Oftentimes, Oxervate requires an insurance pre-authorization that can take two weeks to obtain. Amniotic membranes work well as induction therapy until Oxervate is approved.

ASEDs and PRP are rich in neurotrophic factors and can be used as primary treatment for a persistent corneal epithelial defect.[3] Sometimes, defects that are refractory to ASEDs can improve with PRP therapy.[3]

Oxervate has been FDA-approved for all stages of NK and is a safe and effective option to encourage corneal re-epithelialization and increase in corneal nerve density.[4,5] One drop of Oxervate is instilled six times a day at two-hour intervals for eight weeks.

"Progression of NK can be sight-threatening, as deeper layers of the cornea become involved and the risk of superinfection and tissue lysis increases," Dr. Kataria notes. "Early detection and intervention is key to preventing progression and maintaining vision." ■

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Dr. Michele Andrews

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Dr. Sahil Dosaj

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1 Vision Council 2019. The Vision Council shines light on protecting sight and health in a multiscreen era. https://thevisioncouncil.org/blog/vision-council-shines-light-protecting-sight-anhealth-multi-screen-era. Accessed 19 July 2022. **2** CVI data on file 2018. Prospective, multi-center (5 US sites), open label, bilateral wear, one week dispensing study with MyDay Energys. N=77 habitual soft CL wearers. 75% of patients slightly agree/agree/strongly agree. **3** CVI data on file 2018. Prospective, multi-center (5 US sites), open label, bilateral wear, one week dispensing study with MyDay Energys. N=77 habitual soft CL wearers. 80% of patients agreed when asked "CLs make my eyes feel less tired" (slightly agree/agree/strongly agree). **4** CVI data on file 2018. Prospective, multi-center (5 US sites), open label, bilateral wear, one week dispensing study with MyDay Energys. N=77 habitual soft CL wearers. 74% of patients agreed when asked "CLs make my vision less blurry end of day, even after a long day using digital devices" (slightly agree/agree/strongly agree). **5** CVI data on file 2022. **6** CVI data on file Q4 2022. US Industry reports and internal estimates. **7** CooperVision data on file 2020. Rx coverage database n=120,406 eyes for Rx with <0.75DC; 14 to 70 years.

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BY MOHAMMAD RAFIEETARY, OD, AND ROYA ATTAR, OD
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Age-related macular degeneration (AMD) is a common condition that can cause significant central loss of vision. According to the CDC, in 2019, an estimated 19.8 million Americans aged 40 years and older were affected by some degree of AMD.[1] Its prevalence also increases with age, from 2% among people aged 40 to 44 to 46.6% among people aged 85 and older.[1]

AMD is a specific retinal degenerative disease with distinct clinical features primarily associated with alteration of Bruch's membrane/choroidal complex, retinal pigment epithelium (RPE) and photoreceptor cells in the macula which is defined as the central 5mm of the retina.[2] Aside from aging, genetic susceptibility, cigarette smoking, obesity and higher BMI, female gender, white race, cardiovascular disease, hypertension, hypercholesteremia and poor dietary habits are also among the risk factors for AMD.[3-6]

Several challenges remain in the detection and management of AMD. Approximately 25% of cases go undetected during routine eye examinations.[7] In addition, there are several macular degenerative and non-degenerative conditions with phenotypic and genotypic similarities to AMD that can be mistaken for and misdiagnosed as AMD. Two common ones include adult vitelliform macular dystrophy and Stargardt's disease.[8]

Here, we'll discuss how to interpret clinical findings and use multimodal imaging to more accurately diagnose and classify AMD. We will also walk through three case examples of patients misdiagnosed with AMD presenting with classic signs of other forms of macular degenerative disease. To wrap up, we'll review the current and future treatment and management options for patients with varying levels of AMD.

Case 1

A 70-year-old Caucasian female presents for second opinion. She has had reduced vision for the last four years that has worsened in the past two years, particularly in her left eye. She has never smoked. Her medical history was remarkable for systemic hypertension for six years. Her ocular history was remarkable for cataract surgery and YAG capsulotomy nearly 10 years ago. She was diagnosed with AMD by more than one eyecare provider and never received an explanation for the severe vision loss in her left eye.

The patient's best-corrected visual acuity (BCVA) was 20/40 OD and 20/200 OS. All other findings were unremarkable except for posterior

AMD Risk Factors[3-6]

In no particular order, the following factors have been linked to an increased risk of AMD:

- Advancing age
- Genetic susceptibility
- Cigarette smoking
- Obesity
- Higher BMI
- Female gender
- White race
- Cardiovascular disease
- Hypertension
- Hypercholesteremia
- Poor dietary habits

About the authors

Dr. Rafieetary is a consultative optometrist at the Charles Retina Institute in Germantown, TN. He is a fellow of the American Academy of Optometry and the Optometric Retina Society. Dr. Rafieetary is on advisory boards for Heidelberg Engineering, Apellis, Iveric Bio, Notal Vision and OcuTerra. He is also a paid speaker for Optos, Heidelberg Engineering and Notal Vision. **Dr. Attar** is an assistant professor in the Department of Ophthalmology at the University of Mississippi Medical Center. She is on advisory boards for Heidelberg Engineering, Apellis and OcuTerra Therapeutics.

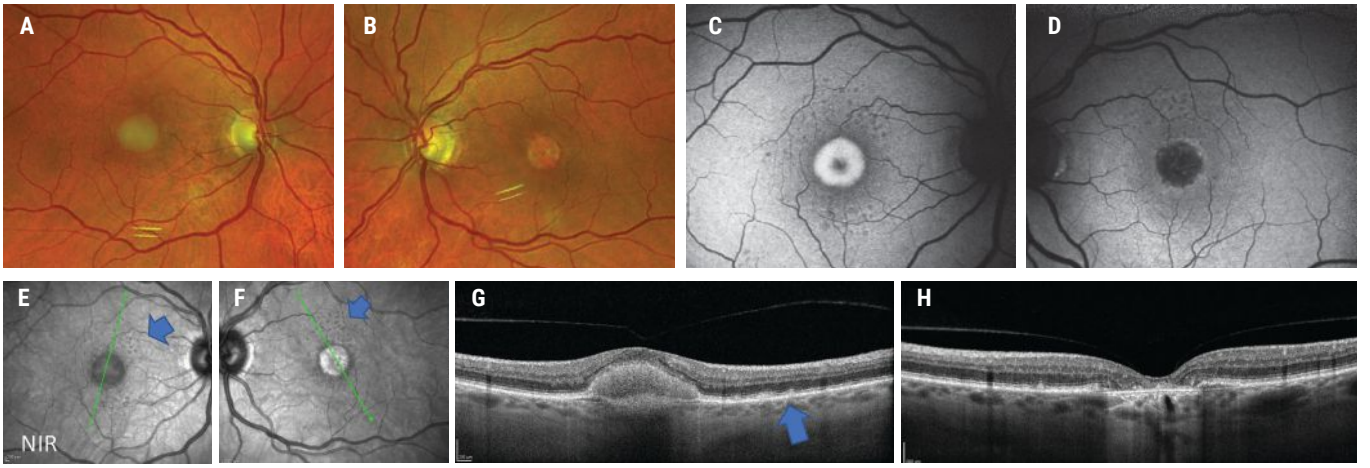


Fig. 1. Fundus photos show a classic foveal vitelliform lesion OD (A) and an atrophic foveal lesion OS (B). FAF of the right eye (C) shows hyperautofluorescence associated with increased lipofuscin, and the left eye (D) shows hypoautofluorescence associated with loss of RPE. The near-infrared reflectance *en face* images (E, F) show drusen and central lesions (blue arrows). OCT of the right eye (G) shows the central hyperreflective PED lesion and few drusen (blue arrow), and the left eye OCT (H) shows the central RPE and outer retinal atrophy.

chamber intraocular lens and YAG OU. Her fundus examination was remarkable for a large central vitelliform lesion OD and a circular foveal atrophic area OS (*Figure 1A/B*). Her optical coherence tomography (OCT) was remarkable for a subretinal hyperreflective lesion in the foveal area, as well as a few drusen and drusenoid pigment epithelial detachments (PEDs) in the right eye and a central area of outer retinal atrophy with extrafoveal drusen in the left (*Figure 1C/D*).

Drusen surrounding the central lesions is also appreciable on *en face* infrared reflectance of the patient's OCT (*Figure 1E/F*). Fundus autofluorescence (FAF) shows a central hyperautofluorescent lesion OD with a central hypoautofluorescent lesion OS and multiple small hypoautofluorescent lesions OU surrounding the central lesions (*Figure 1G/H*).

These findings are the dominant reason for the patient's visual loss due to adult-onset vitelliform dystrophy, an autosomal dominant macular degenerative disease often resulting in central vision loss. While the right eye shows a classic vitelliform or "egg yolk" lesion, the left eye shows the sequela following the dissolution of the vitelliform lesion it results in atrophy.[9] The presence of drusen could suggest a concurrence of AMD.

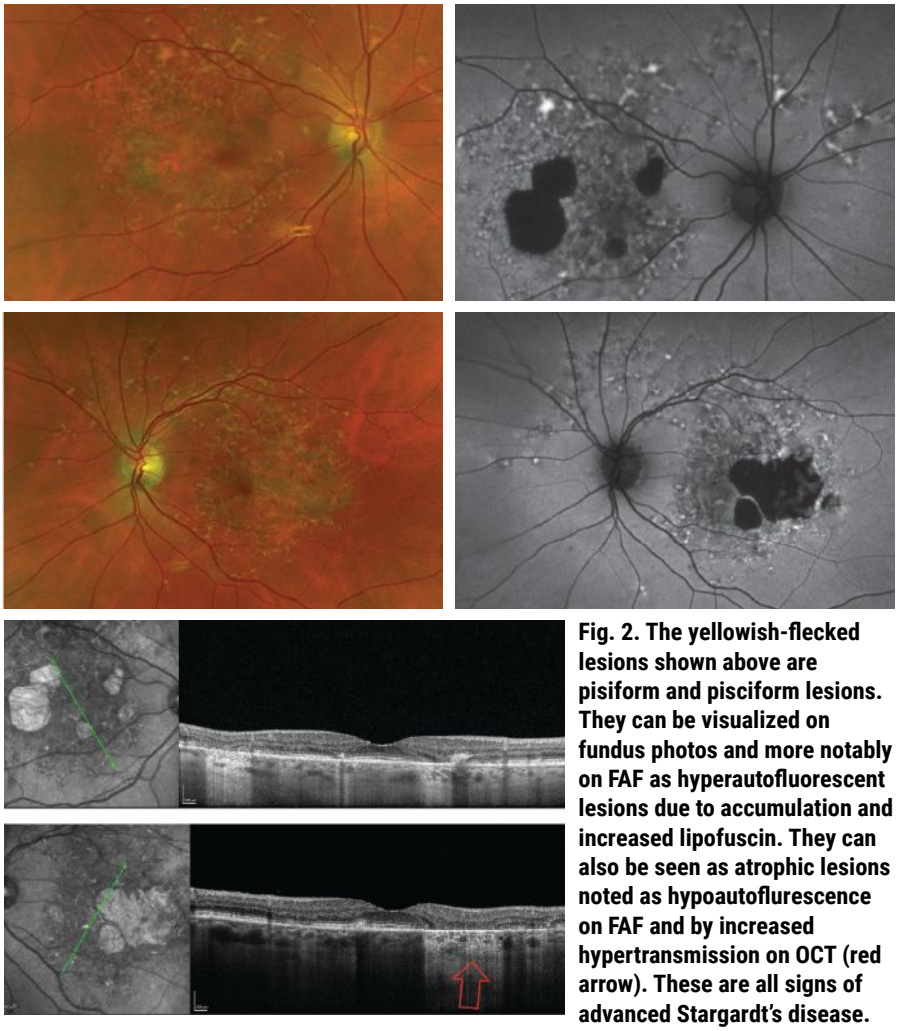


Fig. 2. The yellowish-flecked lesions shown above are pisiform and pisciform lesions. They can be visualized on fundus photos and more notably on FAF as hyperautofluorescent lesions due to accumulation and increased lipofuscin. They can also be seen as atrophic lesions noted as hypoautoflurescence on FAF and by increased hypertransmission on OCT (red arrow). These are all signs of advanced Stargardt's disease.

With no effective treatment for adult vitelliform currently available, this patient will require periodic monitoring—the same as those with dry AMD—due to possibility of choroidal neovascularization.[10]

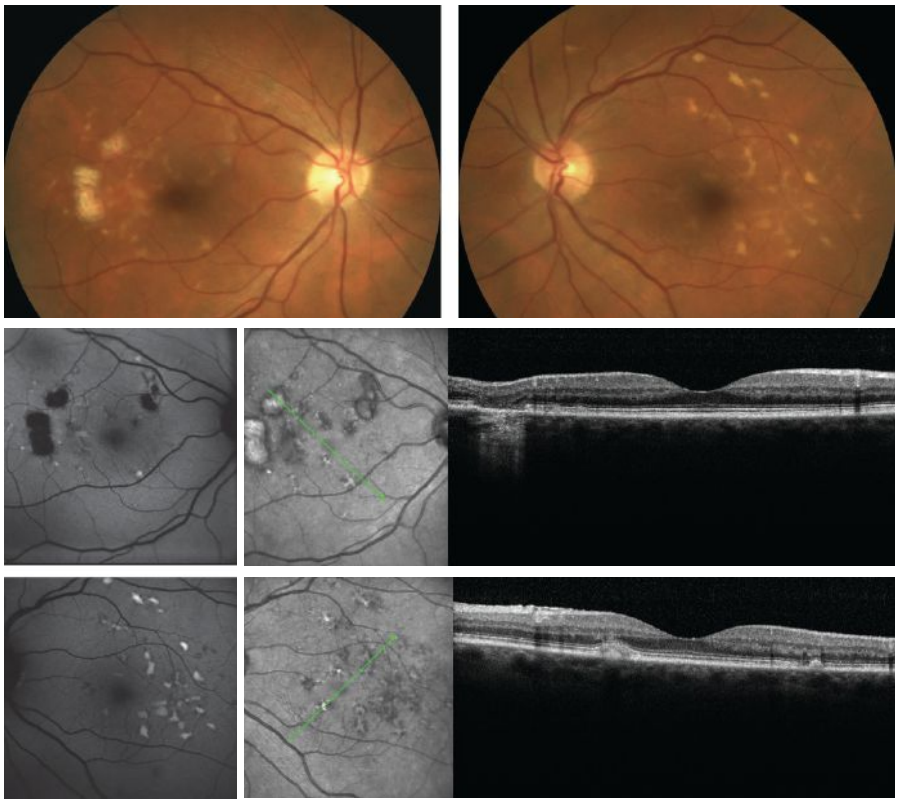


Fig. 3. Same patient as in Figure 2, 14 years younger showing an earlier stage of Stargardt disease. Classic pisciform lesions are seen on fundus photos and FAF. At this stage, the right eye of the patient had already begun with RPE atrophy, while the left eye only demonstrated areas of increased localized deposits of lipofuscin.

Case 2

A 78-year-old Caucasian female presents for evaluation of AMD with possible geographic atrophy (GA). The patient complains of some degree of "blurred vision" but reports that it has not caused significant difficulties in her daily life. She is a nonsmoker and has had systemic hypertension for more than 10 years. She had cataract surgery several years earlier and is aware of her diagnosis of AMD. Her BCVA is 20/20 in both eyes. Anterior segment examination is remarkable for posterior chamber intraocular lens OU.

Fundus examination revealed several yellowish flecked lesions (pisiform and pisciform) throughout the posterior pole with additional large areas of outer retinal atrophy were present temporal to the fovea (*Figure 2*). The view of the lesions was enhanced by FAF imaging. OCT showed areas of incomplete and complete RPE and outer retinal atrophy as well as thickening and increased lipofuscin within the RPE. These findings are consistent with advanced Stargardt's disease, an autosomal recessive disease caused by the alteration of the ABCA4 gene, which is also associated with other macular degenerative diseases including AMD.[11]

This patient had been seen at our clinic 14 years earlier and was noted to have yellowish flecked lesions (pisciform lesions) on clinical examination and FAF, as well as alteration of the RPE and outer retina (*Figure 3*). At that time, she was diagnosed with Stargardt disease based on the presence of classic findings. This case, just like the first one, warrants periodic surveillance. Novel therapies such as use of complement inhibitors for Stargardt's patients are currently under investigation and will eventually allow us to intervene in the disease more aggressively.[12]

Classifying the Disease

Proper staging of AMD relies on a thorough review of the clinical data

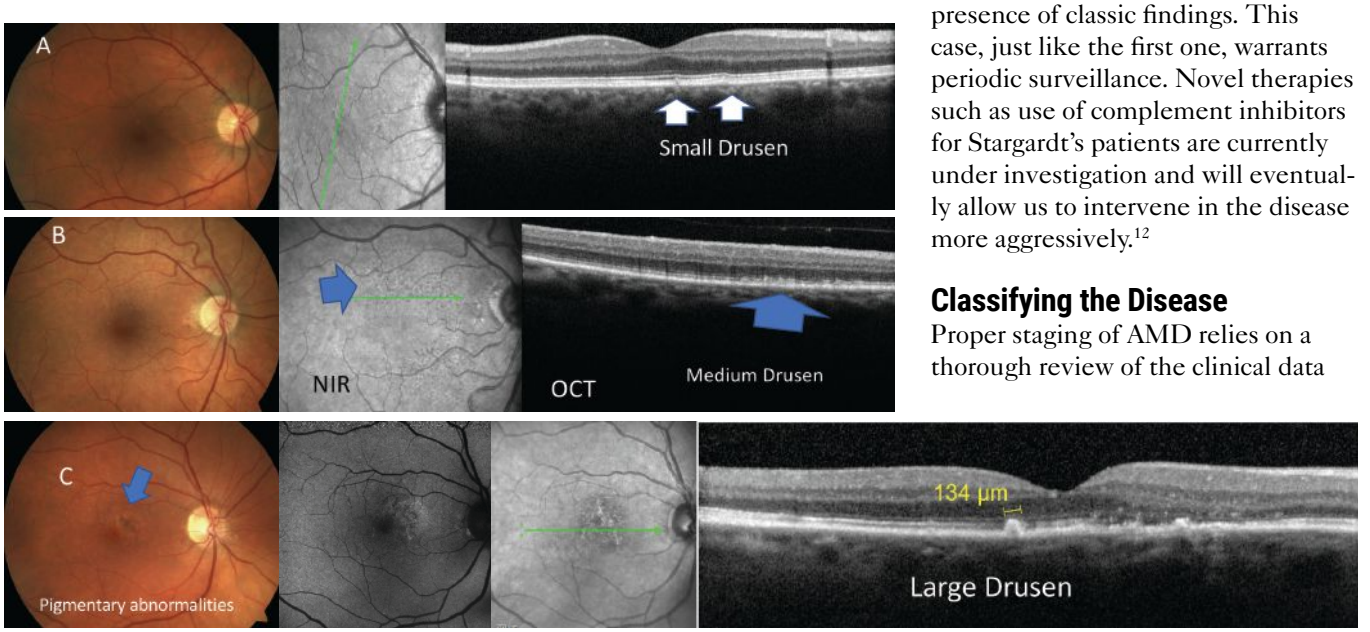


Fig. 4. Examples of the stages of dry or atrophic macular degeneration shown by common imaging modalities.

and images. The presence of small yellowish deposits known as drupelets and drusen between the RPE and Bruch's membrane is the earliest sign of AMD. Although these deposits can present due to natural aging, detection during clinical examination should raise the eyecare provider's suspicion (*Figure 4*).[13] Additionally, risk factors previously mentioned and subjective and/or objective findings such as genetic testing, reduced dark adaptation and other clinical findings such as reduced choroidal thickness detectable on SD-OCT are other clues in the differential diagnosis of AMD vs. normal aging.[14-15]

The basic classification of AMD is considered in a person aged 55 or older, with clinical findings within a two-disc diameter of the fovea.[13] According the Beckman classification (*Table 1*), early AMD is defined as presence of medium size drusen (>63µm to <125µm) without pigmentary changes.[13] Intermediate AMD is in the presence of pigmentary abnormalities and medium or large size drusen (>125µm). The presence of GA or choroidal neovascularization is considered as late or advanced AMD (*Figures 4 and 5*).[11] A simplified severity scale for AMD estimates the risk of conversion from dry to wet AMD to be 0.5% in the absence of large drusen and pigmentary changes.[16] The risk of conversion from dry to wet AMD increases to 50% when both eyes demonstrate the presence of large drusen and pigmentary abnormalities.[16]

TABLE 1. BECKMAN AMD CLASSIFICATION[11]

Classification of AMD	Clinical Findings
Normal Aging	Drupelets or small drusen ≤63µm
Early AMD	No pigmentary abnormalities Medium drusen >63µm to <125µm
Intermediate AMD	Pigmentary abnormalities and/or large drusen

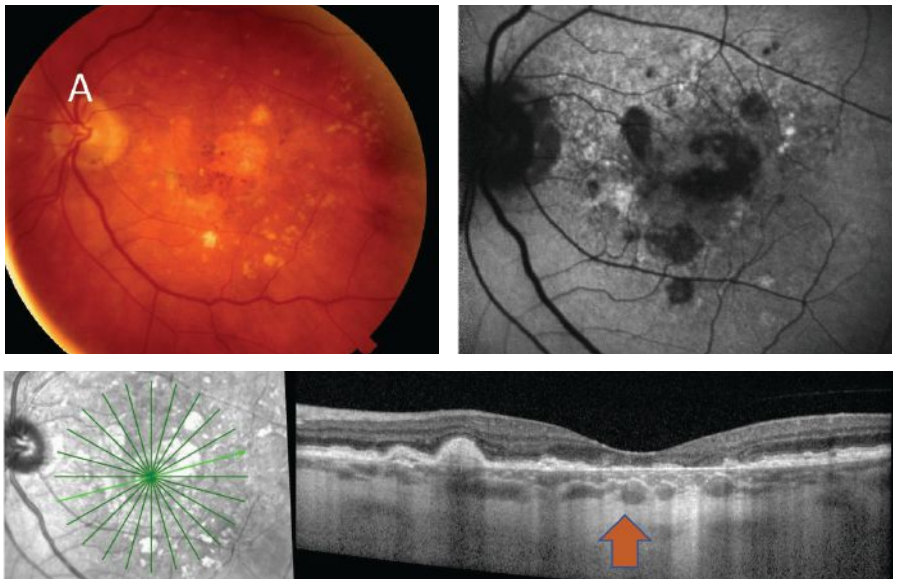
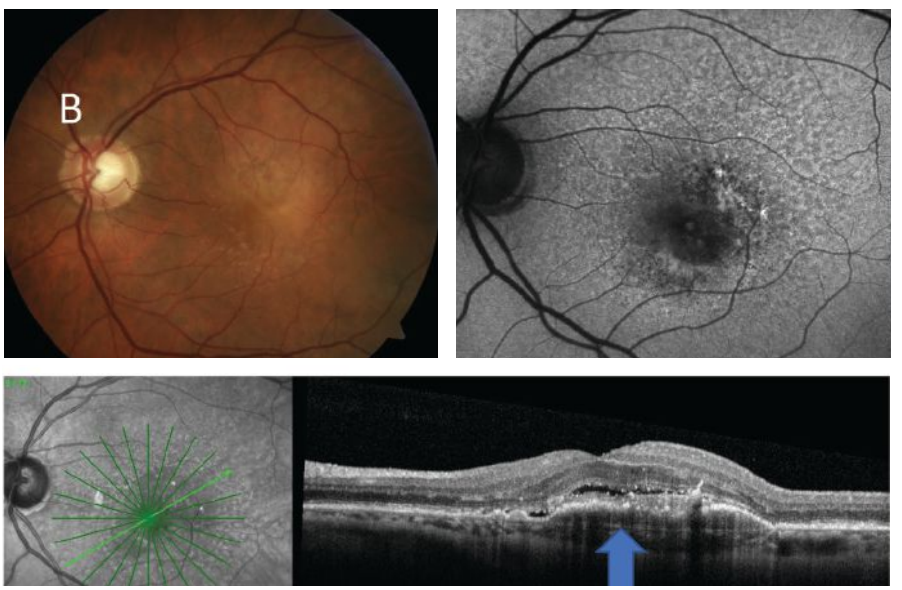


Fig. 5. The clinical images above and below depict the two types of late or advanced macular degeneration. (A) Late or advanced atrophic AMD in the presence of GA. (B) Late or advanced wet AMD.



Case 3

A 66-year-old Caucasian female complains of blurred vision. She has had cataract surgery five years earlier. She is a past smoker and has been on medication for systemic hypertension for several years. Her BCVA is 20/20 OD and 20/20 OS. At first glance, her fundus examination may appear to have several small drusen, as she had previously been given a diagnosis of early AMD. However, a closer look at her fundus photograph reveals subtle pigmentary abnormalities and presence of medium and large size drusen that can be confirmed on OCT. These findings allude to a diagnosis of intermediate AMD, the category with a higher risk for conversion to wet AMD (*Figure 6*).

Diagnostic Tools

AMD diagnosis involves a thorough ocular examination and includes a battery of specialized tests such as visual acuity assessment, dilated fundus examination and ancillary imaging modalities. Multimodal imaging plays a crucial role in timely diagnosis and

appropriate staging of AMD. These include color fundus photography in either conventional white flash or the use of scanning laser ophthalmoscopy, FAF, multispectral imaging including infrared or near-infrared reflectance, OCT and OCT angiography. All of these imaging modalities are instrumental in the diagnosis of AMD.

In cases where AMD is suspected, supplementary diagnostic procedures such as fluorescein angiography or indocyanine green angiography may be employed to determine disease extent and guide appropriate management strategy (*Figures 4 and 5*).[17] The earlier you detect the disease, make an accurate diagnosis based on clinical evidence and start the patient on treatment, the better their chances of avoiding the development of vision loss.

Management Options

In the early stage, patient education is the most important aspect of management. Communication regarding smoking cessation, improved dietary habits, monocular vision self-assessment, management of comorbidities, physical activity and exercise is essential. Additionally, patient understanding of potential consequences of disease progression and the need for regular annual eye examinations is a crucial component of patient education. In intermediate AMD, in addition to the above, vitamin and antioxidant supplementation as recommended by the AREDS and AREDS 2 studies should be discussed with the patient.[18] Implementation of home monitoring devices such as Foreseehome (Notal Vision) has led to early detection at the time of conversion from dry to wet AMD, leading to better visual outcomes.[19]

For those patients who experience difficulties with daily living activities, low vision examination and rehabilitation are best considered earlier than later in the disease cycle.[20]

Management of late or advanced AMD is based on the neovascular or non-neovascular findings. The progression of early and intermediate AMD to GA or wet AMD varies based on different studies; however, its incidence is frequent enough to require patient surveillance for timely diagnosis and management.[13,21,22] GA and wet AMD can also concurrently be present in the same eye and need to be independently treated.[23]

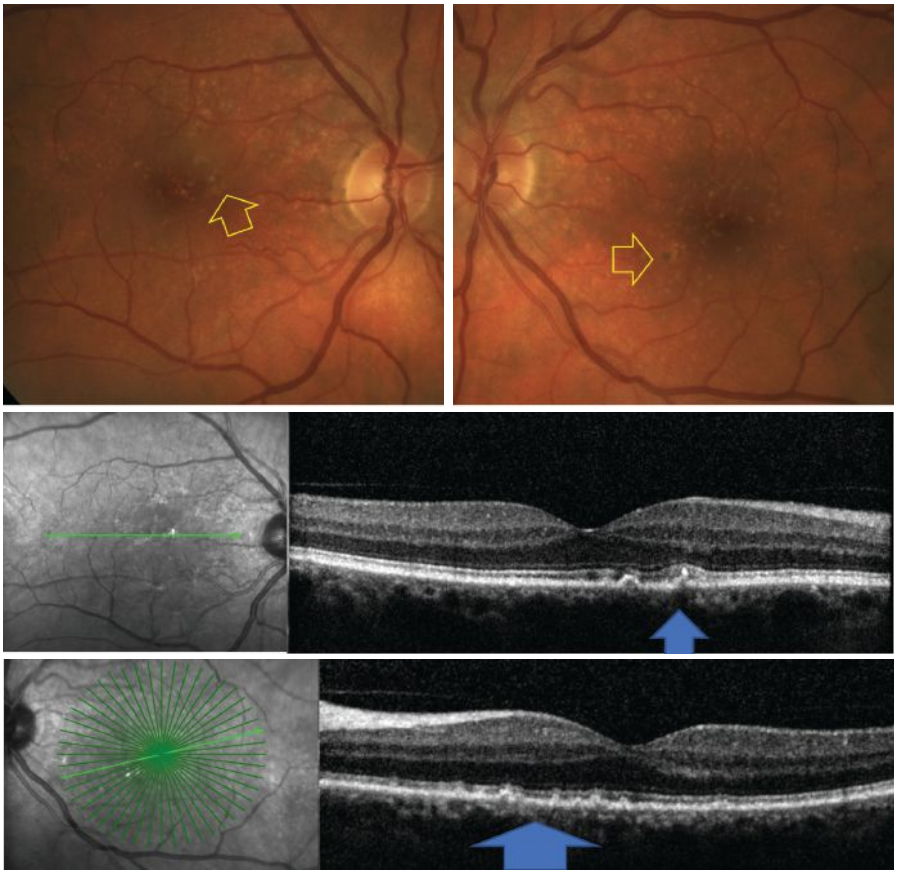


Fig. 6. Fundus photos (top) show multiple drusen and pigmentary abnormalities (yellow arrow). OCT (bottom) shows medium and large size drusen (blue arrows).

Medical Interventions

For the last two decades, the standard therapy for wet AMD has been intravitreal anti-VEGF injections. This began with the FDA approval of pegaptanib (Macugen) in 2004. Since then, a number of FDA-approved and one non-FDA-approved anti-VEGF have been used with good safety and efficacy for treatment of choroidal neovascularization caused by AMD and other conditions such as myopic degeneration.

Several other biosimilar agents either compatible with the current FDA-approved agents have either been approved or are in clinical trials.[24] One downside of these agents is short durability, which results in requiring frequent injections for wet AMD, in some cases as often as monthly. Lapses in treatment have been shown to lead to poor outcomes.[25] As a result, several strategies have been considered to reduce the injection burden that many patients experience. One of these is a recently FDA-approved biphasic anti-angiopoietin 2/anti-VEGF faricimab, Vabysmo (Genentech), that has increased durability and requires fewer injections.[26]

Another concept is the port delivery system (Susvimo; 100mg/mL ranibizumab injection, Genentech/Roche), which requires and implantation of a reservoir and can then be refilled every six months or longer. Following its 2021 FDA approval for wet AMD and during clinical trials for use of port delivery systems in the treatment of diabetic macular edema, the device had a voluntary recall by its developers with safety concerns occurring during refill

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procedures involving the device seal.[27] Genentech/Roche have estimated that the product will return to the market by next year once the production issues are resolved.

Other alternatives to monthly injections in the pipeline include genetic modification by subretinal, suprachoroidal or intravitreal single injection of genetically modified viral vectors. A number of trials are ongoing to evaluate the viability of this modality as an alternative or augmentation to the monthly need for intravitreal injections.[28]

Another group of patients with advanced AMD are those suffering from GA, a progressive irreversible cause of central vision loss. On average, in 2.5 years, a small noncentral GA lesion can reach the foveal center resulting in significant loss of visual function, including decline in reading speed, ability to recognize faces and even inability to drive a car.[29,30]

Despite GA's severity and imposed threat to visual function, until recently there were not any medical treatments for the condition. A number of novel and investigative agents had either failed to show efficacy or are involved in ongoing clinical trials. There is ample evidence of the role of genetic alteration in development of GA and choroidal neovascularization in AMD.[31,32] Genetic variation in the complement genes have shown to be partly responsible in development of GA. Overreaction of the complement system, which is a part of human innate immune system, results in the development of the membrane attack complex, causing destruction of the RPE and photoreceptors.[33]

Based on the evidence, the inhibition of complements C5 and C3 have proven in clinical trials to result in slowing the progression of GA. Based on the results of clinical trials DERBY and OAKS, pegcetacoplan (Syfovre, Apellis Pharmaceutical), a C3 inhibitor, was recently FDA approved as an intravitreal injection for GA to be administered once every 25 to 60 days.[34] Iveric Bio's avacincaptad pegol, a C5 inhibitor, has also been shown to reduce growth of GA lesions in clinical trials GATHER 1 and 2 and is currently under review for FDA approval.[35]

With one therapy for GA on the market and others working their way through the pipeline, eyecare providers should be especially vigilant in identifying the condition to ensure appropriate management or referral of these patients.

Takeaways

AMD is a common condition seen by optometrists in the United States and worldwide, and its patient population is only growing larger with time. A significant number of patients can develop functional vision loss caused by various consequences of AMD. Various conditions can also mimic AMD, making it more difficult to properly diagnose and manage these patients.

To improve clinical and visual outcomes, optometrists should be well-versed in detecting and staging AMD, as well as know when to intervene and refer patients to retina subspecialty facilities for further assessment for treatment. ■

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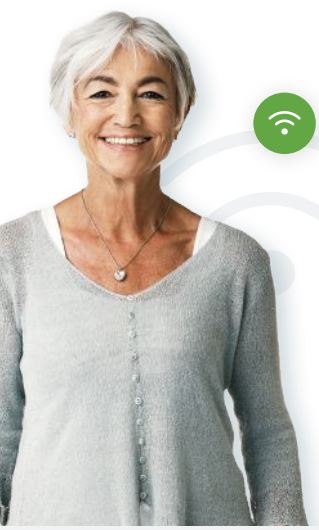

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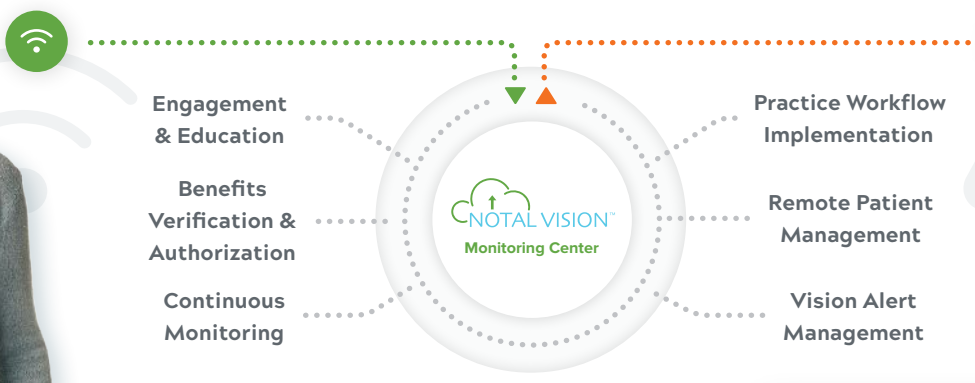

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TIMING THE RETINAL REFERRAL: TIPS FOR SUCCESS

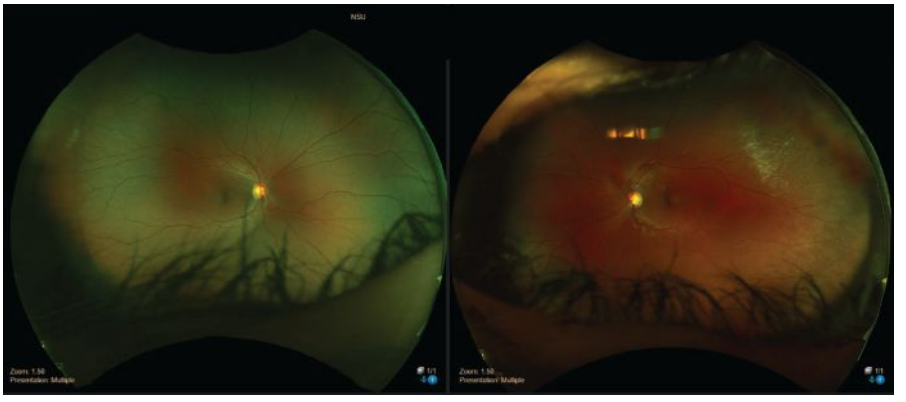
No one wants to hold a patient too long—or pull the trigger too soon. Consider how you would handle these real-world cases in deciding whether a subspecialty consult is necessary.

JULIE RODMAN, OD, AND BRIANNA HERRING, OD
FORT LAUDERDALE, FL

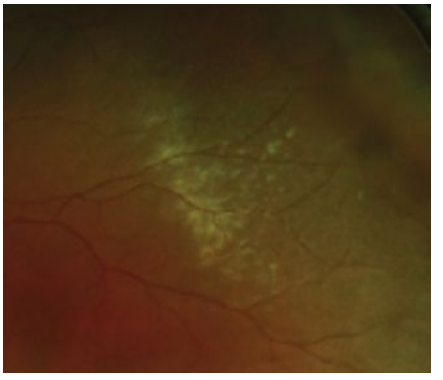
As optometrists, we are confronted daily with retinal pathologies that necessitate proper diagnosis and management. Delaying a referral to a retina specialist may ultimately result in negative visual sequelae for the patient, including loss of vision. It is our responsibility to not only be familiar with myriad retinal pathologies, but also understand the management guidelines that may be specific for each patient. Here, we will address a variety of retinal conditions that may present and provide clinical pearls on the proper protocol for when to refer or when not to refer to a retina specialist.

Case #1

A 17-year-old female presented to the clinic with a history of blunt trauma to her right eye one day prior. She went to the ER the day of the injury, but no fractures were found on imaging. She reported photophobia, eye pain, eyelid tenderness and blurry vision in the right eye since the incident occurred. She denied flashes, floaters, diplopia or headaches. Base-corrected visual acuity (BCVA) measured 20/40 OD with no improvement with pinhole and 20/20 OS. External evaluation revealed swelling and ecchymosis of the upper and lower right eyelid. She reported pain on extraocular movements in all positions of gaze. Pupils were round and reactive without an afferent pupillary defect. There was a trace anterior chamber reaction OD. Fundus examination was unremarkable OD



Commotio retinae superior temporal of the left eye.



Magnified view of commotio retinae highlighting a confluent area of retinal whitening in the mid-periphery.

About the authors

Dr. Rodman is a professor and chief of the Fort Lauderdale (Broward) Eye Care Institute at Nova Southeastern University in Florida. She is a consultant and speaker for Visionix (Optovue), iCare, LKC Technologies, Iveric Bio, Apellis and OcuTerra. **Dr. Herring** graduated from Nova Southeastern Univeristy College of Optometry (NSUCO) last year and is currently completing a residency in primary care with an emphasis in pediatric and binocular vision at NSUCO. She has no financial interests to disclose.

with a discrete area of retinal whitening superior temporal OS. The retinal vasculature was normal in appearance and the macula was flat and intact. The patient was diagnosed with commotio retinae OS and a mild traumatic uveitis OD. *Refer or maintain?*

Commotio retinae. This condition occurs as a result of damage to the retina due to blunt trauma to the globe. The trauma causes shock waves resulting in disruption of the photoreceptor outer segments and swelling of the retinal pigment epithelium (RPE). The retinal blood vessels in the affected area are not affected. Commotio retinae presents clinically as confluent areas of retinal whitening in the periphery or posterior pole either over the site of trauma or on the contralateral side of the globe (countercoup-180° opposite) secondary to traversing of the shock waves from the site of impact. When the macula is involved, it is referred to as Berlin's edema, which presents clinically with a pseudo cherry red spot. Other indications of retinal trauma, such as retinal hemorrhages, may be present as well.[1-3]

Patients will present with a history of recent ocular trauma with fogging of their vision for a few seconds to minutes after impact. The condition is painless, and symptoms are associated with the location and severity of retinal involvement. However, visual acuity does not always correlate with the degree of retinal whitening. If there is intraretinal fluid or cystoid macular edema, patients may experience decreased vision or metamorphopsia. Any patient presenting with ocular trauma needs a dilated fundus examination with or without scleral depression and ocular ultrasound (avoid scleral depression in cases of hyphema, ruptured globe or ocular inflammation).

There is no treatment for commotio retinae, with full recovery one to four weeks after presentation. Management should include addressing any comorbidities including orbital or facial fractures, corneal/conjunctival lacerations, uveitis and hyphema. Some patients may have residual RPE changes including atrophy or hyperpigmentation. In the majority of cases, vision will return to pre-injury levels, while those with macular involvement may have longstanding vision impairment.[4-6] Close follow-up including monitoring of VA, amsler grid and OCT can aid in assessing the staging of the condition. Proper protocol and follow-up are essential to monitor for the development of other sequelae of the trauma.[1-3]

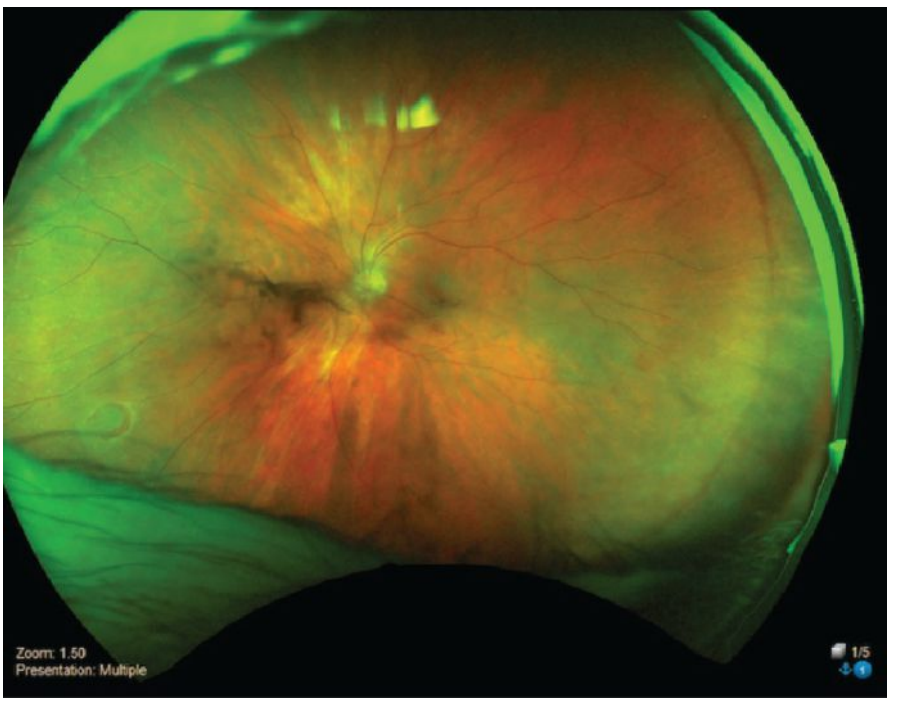
This patient was asked to return to the clinic in one week for a dilated fundus examination. She was advised to report to the clinic or ER immediately if she noted any new symptoms including changes in her vision.

Case #2

A 49-year-old Hispanic male complained of a recent-onset large floater in his left eye that started two days prior. He also reported two episodes of light flashes that had since been resolved. The patient denied any darkening or curtains over his vision. His ocular history was positive for myopia (-5.25D OU). BCVA measured 20/20 OD and OS. Biomicroscopy was unremarkable. Inspection of the anterior vitreous revealed pigmented cells OS. Fundus examination revealed a horseshoe retinal tear inferior nasal with pronounced vitreous condensation in the peripapillary region. *Refer or maintain?*

Horseshoe tear. These are full-thickness retinal breaks that appear as u-shaped or flap and occur due to significant localized vitreoretinal traction. Anomalous posterior vitreous detachment (PVD) is the most common etiology of retinal horseshoe tears. PVD is an insidious process that is a normal result of aging and occurs secondary to the liquefaction or synchesis of the vitreous and anterior contraction or syneresis of the vitreous body. Flap tears occur from spontaneous PVD, where the posterior hyaloid exerts traction on the retina, pulling it anteriorly towards the vitreous cavity, resulting in a retinal tear.

Tears can exist in any region of the peripheral retina, most often near the posterior margin of the vitreous base in areas of lattice degeneration, pigment clumps or retinal tufts. With



Horseshoe retinal tear inferior temporal OS.

chronicity, RPE hyperplasia may develop around the lesion. This reactive pigmentation acts as a retinal "seal" and may reduce the likelihood of retinal detachment development.

Certain risk factors predispose patients to the development of a horseshoe retinal tear. These include advancing age, myopia, lattice degeneration, trauma, retinoschisis, family or personal history of retinal detachment or previous ocular surgeries (aphakia/pseudophakia). There is no prophylactic treatment for the development of horseshoe retinal tears.

Patients with acute retinal breaks may present with flashes of light and/or floaters in their vision with or without vision changes. If the retinal break is longstanding, patients may be asymptomatic. Ocular examination should include dilated fundus examination with scleral depression. Careful inspection of the anterior vitreous is critical to evaluate for the presence of pigment (Shafer's sign), an indication of retinal break in patients without previous ocular surgeries. B-scan ultrasonography is useful if there are dense opacities obscuring accurate visualization of the retina.

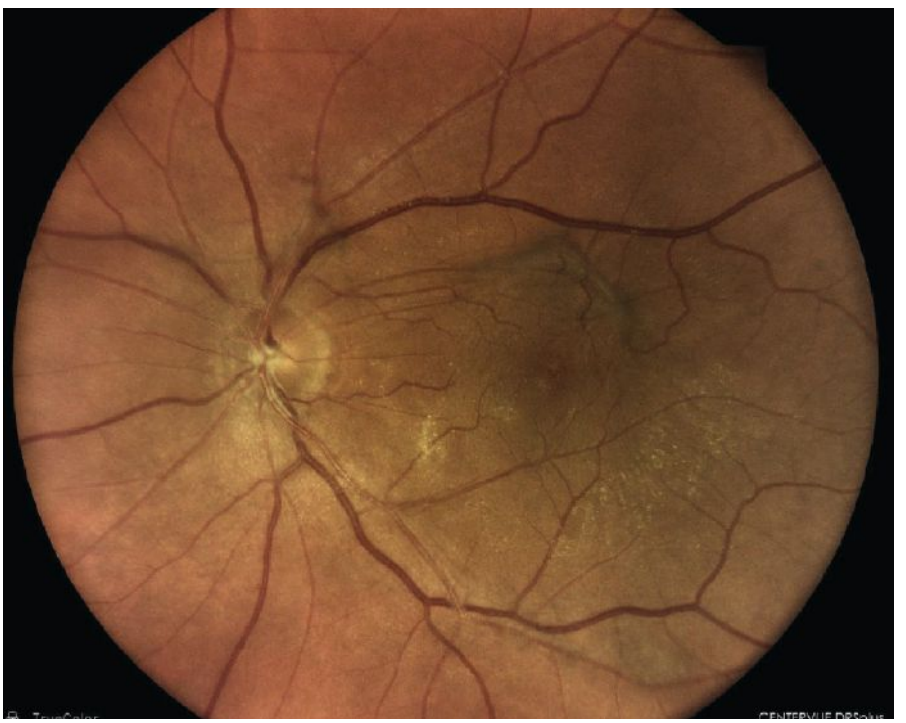
Horseshoe flap tears are the leading cause of rhegmatogenous retinal detachments, where a retinal break leads to the accumulation of subretinal fluid and separation of the neurosensory retina from the underlying RPE. This is secondary to the persistent traction of the vitreous on the apex of the flap. Symptomatic horseshoe tears have a 33% to 55% chance of progressing to retinal detachment. Asymptomatic flap tears with subretinal fluid have a 40% chance of progressing while those without subretinal fluid have a 5% to 10% chance of progressing.[7-12]

Acute symptomatic retinal breaks require immediate laser therapy or cryotherapy (freezing procedure) within 24 hours. The goal of treatment is to provide a chorioretinal seal around the lesion preventing vitreous from getting into and under the break resulting in a detachment. Treatment for asymptomatic horseshoe tears is case dependent. Observation without treatment may be appropriate for chronic tears, tears surrounded by pigmentation, tears without vitreoretinal traction or other asymptomatic patients. Following prophylactic laser treatment, the patient should be seen at one week and again at three to six weeks. Patients with risk factors or those with breaks that do not require treatment should be followed at three months and then every six to 12 months if stable.[7-12]

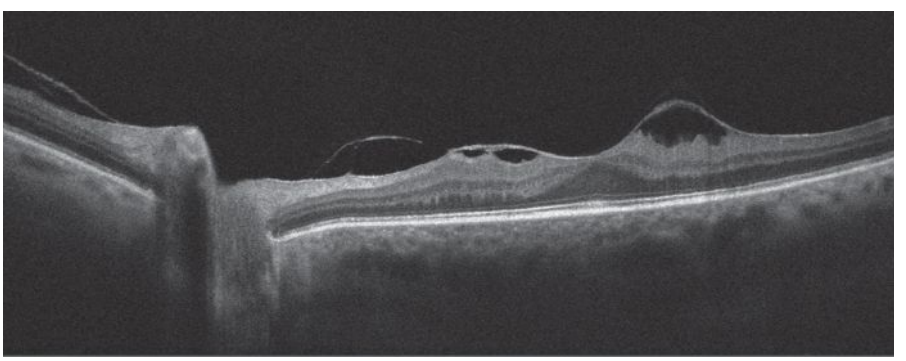
This patient was immediately referred to the retina specialist for intervention. Horseshoe tears should be sent to the retina specialist the same day or at the latest the following morning.

Case #3

A 60-year-old male presented with complaints of blurry vision and metamorphopsia OU. He stated that he started noticing the disturbance the previous year, but it got progressively worse. His ocular history was positive for successful cataract extraction two years prior. BCVA was 20/30 OD and 20/40 OS. His entrance testing was unremarkable; however, he did note squiggling of the lines on Amsler grid OS and a smudge in the center



Epiretinal membrane with overlying vitreomacular traction membrane OS.



SD-OCT image highlighting vitreomacular traction with epiretinal membrane OS.

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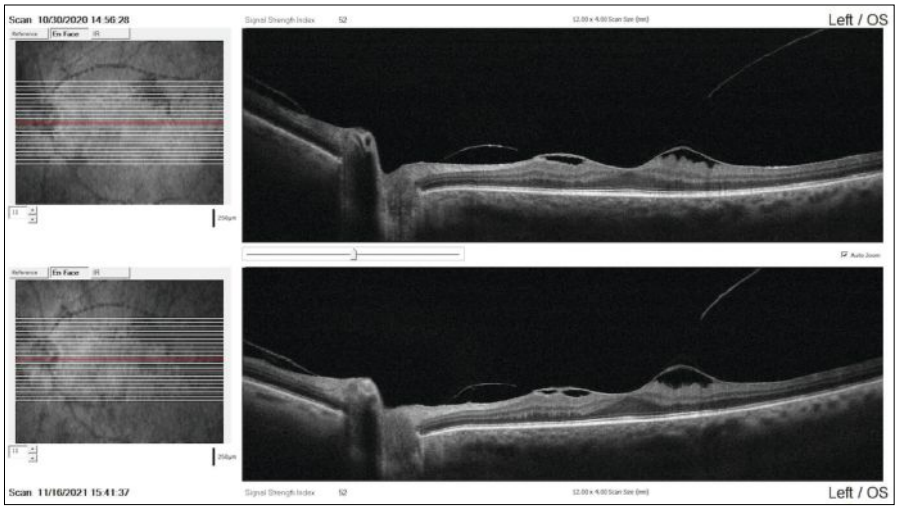


Comparison report with raster scans performed one year apart indicating the stability of the condition (top: baseline scan; bottom: recent scan).

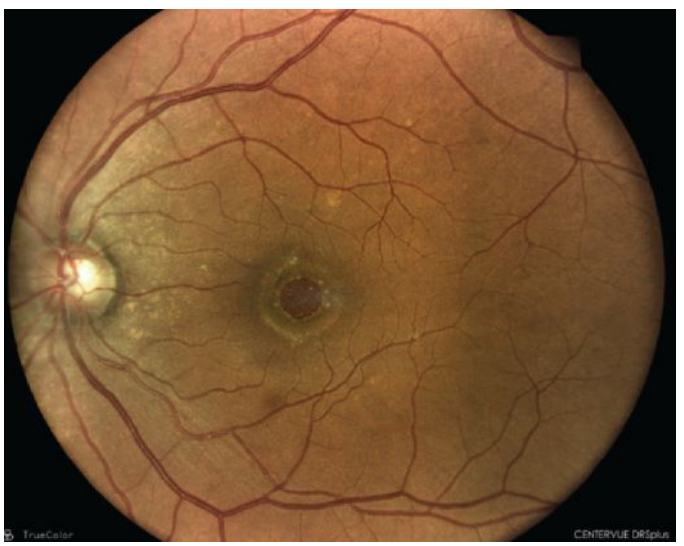
of his vision OD. Fundus examination revealed a maroon, well-defined circular lesion directly over the fovea OD and an epiretinal membrane with visible vitreous traction suspending from the optic nerve hypoplasia superior to the macula OS. Watzke Allen test was negative OD and OS. OCT confirmed the presence of a partial thickness hole (lamellar) OD and epiretinal membrane (ERM) with vitreomacular traction (VMT) OS. *Refer or maintain?*

ERM. This growth is defined as a fibrocellular proliferation of the superficial retina. The proliferation forms a scaffold that is composed of cellular contents, vitreous and fibrotic components and is associated with glial cell proliferation. A fine-to-thick glistening membrane will be seen on clinical examination. The incidence of ERM varies widely in the literature, from 2.2% to 11% in phakic patients without pre-existing ocular conditions.[13,14] Studies have shown that 2% of patients over the age of 50 and 20% over the age of 75 have evidence of ERMs, although most do not need treatment. Both sexes are equally affected. In about 10% to 20% of cases, both eyes have ERMs, but they can be of varying degrees of severity.[15] The most common etiology of ERM is an anomalous PVD with trauma, retinal breaks, retinal vascular diseases, retinal inflammation as well as others.

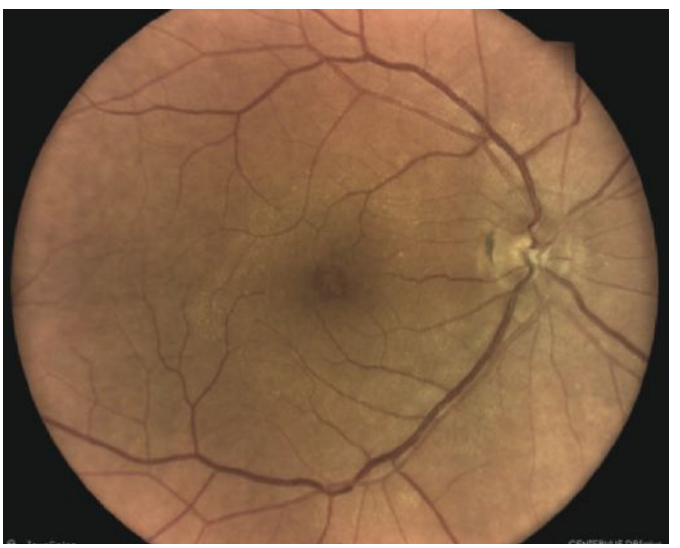
The pathophysiology behind ERM formation involves the presence of retinal microbreaks in the inner limiting membrane (ILM) allowing for microglial cells to gravitate and diffuse onto the retinal surface. The migration of these cells results in firm adherence of the resulting membrane to the underlying retina.[16-18] ERM can also lead to macular traction and symptomatology. If the residual vitreous exerts traction on the center 3mm radius of the fovea, resulting in a foveal contour change, VMT results. ERM and VMT can lead to vision loss, metamorphopsia and doubling of images.

OCT is a highly effective way of imaging both ERM and VMT and is used to make management/treatment decisions in affected patients. Three-dimensional analysis of the retinal architecture from inner limiting membrane through choroid allows for visualization of not only superficial retinal changes but also inspection of the photoreceptor integrity line and outer retina to assess for the extent of involvement.

Epiretinal membranes tend to remain stable and can be monitored when the patient is asymptomatic.



Full-thickness macular hole with surrounding fluid cuff.



Macular pseudohole.

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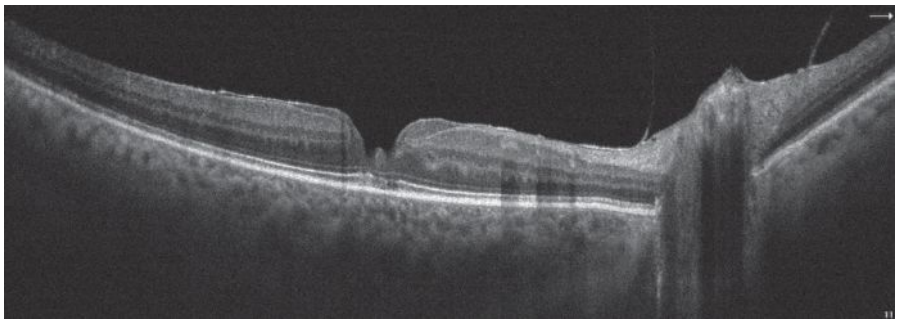
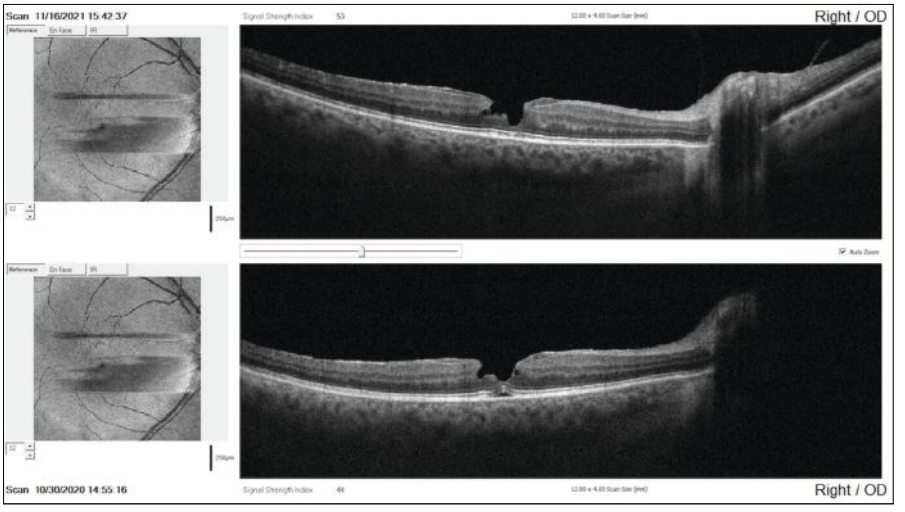


SD-OCT highlighting a partial thickness macular pseudohole with overlying epiretinal membrane.



SD-OCT comparison report taken one year apart showing minor evidence of change in the contour and irregularity of the pseudohole (top: recent scan; bottom: baseline scan).

However, if a patient's vision is 20/60 or worse, if they are symptomatic (metamorphopsia or doubling of vision) or if there is evidence of progression, they should be referred for possible surgical intervention. The most common surgery for ERM is pars plana vitrectomy with membrane peel with or without concomitant peeling of the ILM. Visual recovery can be slow ranging from three to 12 months postoperatively. Recurrence of membranes occurs in approximately 5% of patients.[19]

VMT. In these cases, if the patient is asymptomatic, regular monitoring with OCT (three-month intervals) and daily home amsler grid use is recommended. VMT spontaneously resolves in 11% to 53% of cases with mean time of release of 15 to 18 months. This statistic highlights the variability in the course of the condition.[20-24] If the patient becomes symptomatic, they should be referred to a retinal specialist, as VMT can lead to macular hole, ERM or cystoid macular edema. Pars plana vitrectomy is one treatment option that involves the manual release of the vitreous attachment thereby alleviating the traction. Pars plana vitrectomy can work well in cases of pseudophakia, concurrent ERM and broad vitreomacular adhesions.[25-27] Pneumatic vitreolysis is a procedure that involves injecting a small gas bubble into the eye which severs the adhesion between the vitreous and the macula inducing a PVD.[28,29] Due to the increased rate of retinal tears and detachments, this procedure is no longer being used.[30]

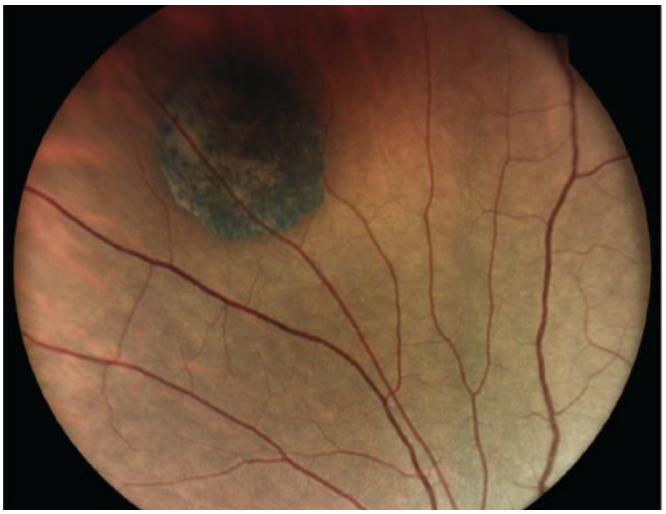
A partial thickness macular hole was found in the other eye. How do we differentiate it from a full-thickness macular hole?

Full-thickness macular hole is a localized, complete loss of tissue affecting the ILM layer through the RPE. During clinical exam, it will appear as a circular, reddish lesion over the fovea. The edge of the hole may contain diffuse areas of intraretinal pseudocysts that will appear as circular hyporeflective lesions.

Due to the wide variability in defining macular holes, The International Vitreomacular Traction Study Group provided a universal classification system using findings seen on OCT. The classification system evaluated two aspects of the hole. First, they evaluated the size of the hole using OCT metrics. If the hole measured <250µm at the narrowest point of separation, it was considered small. If the hole was 250µm to 400µm it was considered medium, and >400 µm was considered large. These measurements provide a means of determining efficacy and outcome of treatment. The second parameter that was assessed was the relationship of the vitreous to the hole. If there was vitreomacular traction or vitreous involvement, this would be considered a primary full-thickness hole and if the vitreous was not involved, then it would be considered a secondary hole. Examples of causative factors resulting in a secondary macular hole are trauma, high myopia and macular schisis.[31] Most full-thickness macular holes should be referred to a retina specialist; however, longstanding holes will not improve from intervention and thus may be monitored.

Lamellar macular hole is a partial-thickness macular hole with an incomplete loss of tissue from ILM to RPE. During clinical exam, it will appear as a circular or oval reddish lesion similar to a full-thickness macular hole. On OCT, lamellar holes may have: an irregular foveal contour, a defect in the inner fovea which does not have to be actual loss of retinal layers or schisis between the outer plexiform layer and outer nuclear layer in the presence of an intact photoreceptor layer.

Macular pseudoholes are also partial-thickness holes that resemble a "U" shape and can be related to ERM.



CHRPE; well-demarcated lesion with lacunae within.

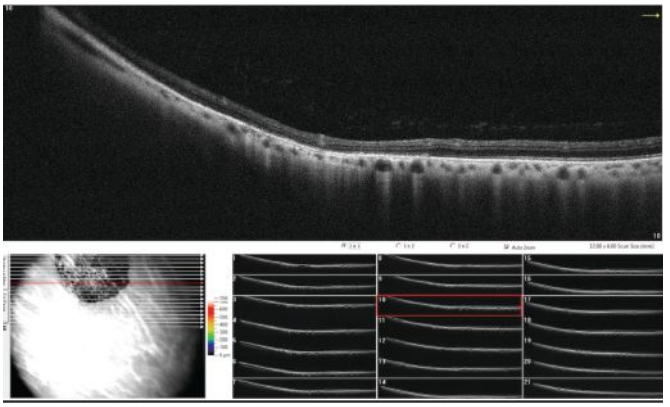
When should we refer partial-thickness holes? Surgical intervention tends to be controversial as results are variable. There may be reason to recommend surgery in patients with partial-thickness macular holes; particularly those with functional or anatomical deterioration, those experiencing metamorphopsia or those who have poor entering visual acuity resulting in inability to perform daily tasks. Other guidelines recommend surgical intervention whenever VA <20/40 and there is evidence of ERM.[32,33] Surgical options include phacoemulsification with vitrectomy and ERM and ILM peeling. Preoperative defects in the photoreceptor integrity line are associated with worse visual prognosis.[34]

Due to complaints of metamorphopsia and blurry vision, we opted to refer this patient to a retina specialist for consultation. Surgical intervention was not performed; however, the patient is being comanaged by our clinic and the retina specialist.

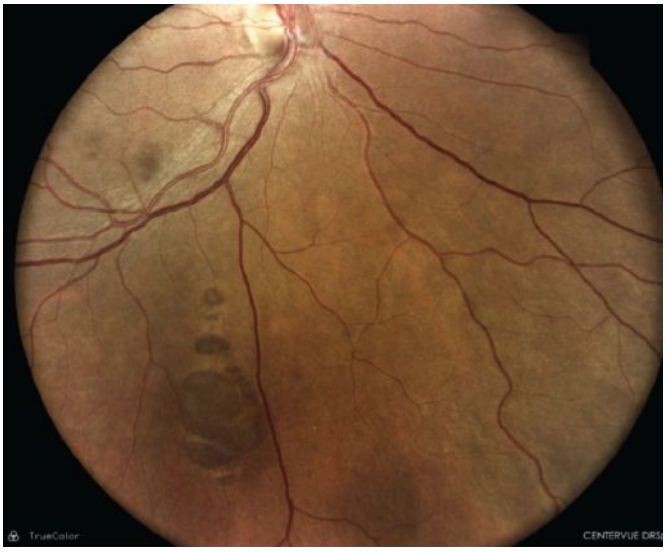
Case #4

A 45-year-old Hispanic female presented for a comprehensive eye examination. She reported mild blurry vision without her glasses but had no other visual complaints. Her ocular and medical history were unremarkable. Her family medical history was positive for skin cancer in her father and uterine cancer in her mother; both parents were still alive. Entrance testing including slit lamp biomicroscopy were unremarkable with BCVA 20/20 OD and OS. Funduscopy revealed a solitary, mid-peripheral 6-7DD darkly pigmented, well-defined lesion with lacunae within. The remainder of the fundus examination was unremarkable OU. Ancillary testing including OCT was obtained on the day of the visit. *Refer or maintain?*

Congenital hypertrophy of the retinal pigment epithelium (CHRPE). These are benign pigmented hamartomas of the retina that are routinely found on comprehensive eye



SD-OCT raster scan through the CHRPE illustrating retinal thinning above the lesions with photoreceptor loss and shadowing of underlying choroid.



Scattered CHRPE with depigmented tails and irregular borders.

examination. These lesions present clinically as well-defined, round to oval gray, brown to black lesions, while atypical amelanotic variants also exist. CHRPE are histologically composed of enlarged, taller RPE cells that are tightly packed with melanin granules. Defining characteristics of these lesions include hypopigmented borders or halo around the lesion with lacunae or pale colored areas within. Such lesions may present as unifocal or multifocal and may be present in the mid-peripheral or peripheral fundus.

Grouped CHRPE lesions are often referred to as "bear tracks." Atypical CHRPE is associated with familial adenomatous polyposis, including subtypes Gardner's syndrome, familial polyposis coli and Turcot syndrome; these are malignant conditions characterized by polyps of the colon and rectum.

The presence of multiple scattered, bilateral CHRPE with depigmented tails and irregular borders are considered a clinical disease marker and can be used to identify at-risk patients.[35] It has been proposed that four or more of these lesions is highly suggestive for familial adenomatous polyposis.[36-38]

Diagnostic imaging modalities include fluorescein angiography and indocyanine green angiography; both of which will show blockage of fluorescence of the pigmented lesions with transmission of choroidal fluorescence through the depigmented areas throughout the angiogram. Fundus autofluorescence can rule out lesions that simulate melanoma. Visual field findings of CHRPE are variable and include relative to absolute scotomas depending on the extent of the lesion. OCT will exhibit retinal thinning above the lesion with photoreceptor loss and shadowing of the underlying choroid.[39,40] Ultrasonography can be used to document thickness and confirm clinical findings; ultrasound of a melanotic lesion usually shows low-to-moderate reflectivity with choroidal excavation.

Management includes periodic observations. If evidence of subretinal fluid or exudation appears, or if the lesion has characteristics suspicious for malignancy, they should be referred to a retinal specialist for intervention. Lesions that are atypical in appearance should warrant colorectal screening with a gastroenterologist.

Back to our patient, the clinical appearance and ancillary testing was highly suggestive of typical unifocal CHRPE and thus we opted to monitor the patient annually. The patient was advised regarding the necessity and importance of regular dilated eye examinations.

Takeaways

As primary eyecare doctors, optometrists are exposed to an endless array of ocular conditions. It is our responsibility to be familiar with the proper diagnosis and management of these conditions, as timely referral may make a big difference in the final outcome. We are often the first point of contact for our patients, and with this comes an obligation to know when to hold and when to refer to our patient. ■

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VITREOUS OPACITIES: BENIGN OR SERIOUS?

From bothersome floaters to acute hemorrhage, we break down these findings and help you determine proper management.

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

The vitreous body marks the largest ocular structure, composing approximately 80% of the eye. But for its extremely large size, the vitreous receives modest attention in comparison to the more alluring retina, cornea and lens. This may be attributed to the fact that primary vitreal disease is uncommon, with many developing it early in infancy and less likely for the primary optometrist to run into. Nonetheless, the vitreous plays a significant role in retinal disease development.

Why else should we pay attention to the vitreous? Well, it is responsible for a frequently heard patient complaint—floaters. Symptoms of floaters could translate clinically into a number of vitreous opacities, most often benign, but sometimes indicating a serious condition. When encountering vitreous opacities, what are we looking at and when should we be concerned? This review is dedicated to answering those questions, starting with vitreous anatomy and imaging and delving deep into breaking down vitreous opacities and treatments.

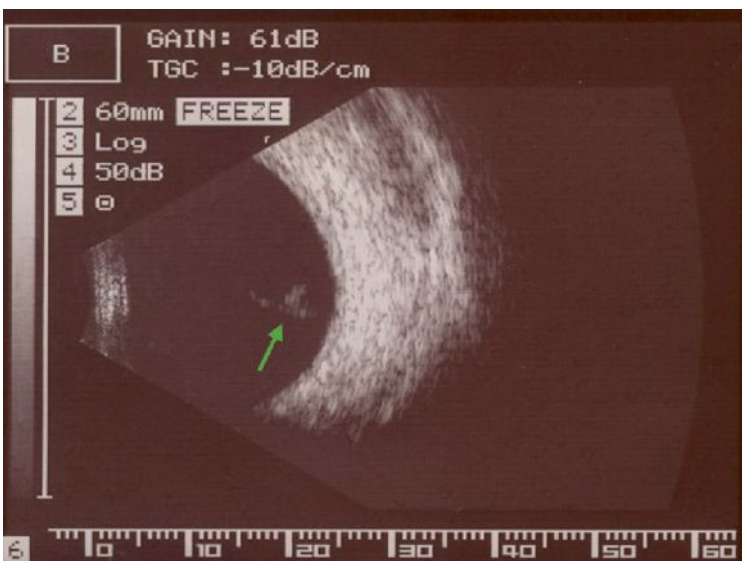

Fig. 1. B-scan ultrasonography imaging of a Weiss ring visible within the posterior vitreous after PVD.

Vitreous Composition

Understanding floaters and vitreous opacities starts with a clear understanding of the anatomy and the dynamic changes the vitreous undergoes with aging. The acellular vitreous is transparent by nature as it is mostly water, about 98%. Two main macromolecules, collagen and hyaluronan, provide structure and elasticity.

The majority of the vitreous is the core, where liquefaction occurs. Surrounding the core lies the vitreous cortex, a thin layer with an increased density of collagen fibrils. The thickness of the cortex is in the range of 100µm to 300µm, being thinnest in the central macula and absent over the optic nerve.[1,2] The posterior hyaloid membrane is adherent to the

About the authors

Dr. Bedwell is a clinical associate professor at Indiana University School of Optometry. She is a fellow of the American Academy of Optometry and the Optometric Retina Society, as well as a member of the American Optometric Association. She serves as editor of the Optometric Retina Society's e-newsletter. **Dr. Krenk** is an assistant clinical professor at Indiana University School of Optometry, where she teaches optometry students and see patients at the Indianapolis Eye Care Center. She is also a fellow of the American Academy of Optometry and a member of the American Optometric Association. They have no financial interests to disclose.

internal limiting membrane of the surface retina.[3] The posterior hyaloid contains type IV collagen (vs. type II in the core) as well as adhesive proteins: laminin and fibronectin.[3-5] Thus, it essentially functions as the glue between the vitreous and retina. The posterior hyaloid is only appreciable once posterior vitreous detachment (PVD) has occurred.

Vitreous gel liquefaction commences as young as four years old and then steadily increases with age.[6,7] Liquefaction occurs as collagen aggregates into bundles, causing development of empty pockets of liquid called lacunae. The vitreous transitions from a gelatinous composition at birth to a 50:50 ratio of gel to liquid by age 80.[1,7]

Examination

There are multiple ways to evaluate the vitreous body, the easiest of which is by simple slit lamp and ophthalmoscopy exam. However, this method has limitations in both the extent and fine detail of vitreous changes that can be assessed due in part to the vitreous's transparent nature.

B-scan ultrasound can image the entire vitreous body and can detect differences in density between liquefied and gel vitreous, as well as collagen aggregates and other opacities.[8] Although not often used clinically, objective, quantitative values of the vitreous can be measured from a B-scan and have been shown to correlate with contrast sensitivity and visual function.[9]

OCT provides a limited view of the vitreous, specifically giving information about the posterior vitreous and vitreoretinal interface. Unfortunately, it does not image the central or anterior vitreous very well and opacities in these areas may still be responsible for symptomatic floaters. Swept-source OCT, in particular, provides better vitreous visualization over SD-OCT and allows for a wider view to image the interface across the macula and optic nerve in a single scan. Normal anatomy can be appreciated with a higher resolution, wide angle OCT scan (*Figure 2*).

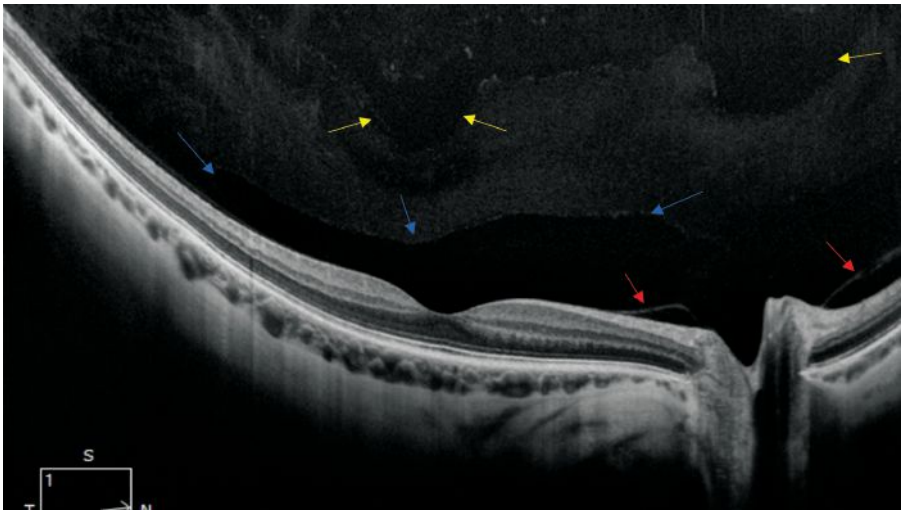

Fig. 2. A high-resolution single line scan OCT of a healthy, myopic 30-year-old (12mm HD 1-Line Raster 100x, Zeiss Cirrus OCT). The blue arrows point to the normal anatomic bursa premacularis. The red arrows reveal a non-pathologic release of the posterior hyaloid. In the more anterior aspect of the image, there is evidence of liquefaction noted by the optically empty voids noted by the yellow arrows.

Types of Vitreous Opacities

Whether physiologic (*e.g.*, floaters) or pathologic, these impact the inherent structural translucency of the vitreous. An opacity may be clinically subtle or apparent and likewise for patients either visually symptomatic or asymptomatic.

Floaters. The most encountered of all vitreous opacities are primary vitreous floaters, also known as by the Greek-derived term *myodesopsia*. Most floaters occur as a result of increasing liquefaction with age, myopic vitreopathy or in PVD. With age, collagen fibrils aggregate into fibers that can scatter light, resulting in symptomatic floaters.[10] Descriptions vary from patient to patient and include cobwebs, lines or hairs to dots, spots, gnats and flies. While floaters are a minor nuisance for most, some are chronically visually significant in terms of the visual disturbance and/or impact on contrast sensitivity.[11]

Treatment of chronically symptomatic floaters has increased due in part to development of smaller-gauge instruments in vitrectomy as well as recognition of the visual burden caused to some individuals. With proper patient selection, a standard three-port pars plana vitrectomy has proven successful in high rates of patient satisfaction, as well as low incidence of complication.[12-14] To reduce complication risk further, retina surgeons can employ a limited or core vitrectomy, removing just the central vitreous gel.

Sebag and colleagues performed a limited vitrectomy using 25-gauge instruments for symptomatic floaters. They found quantitative echodensity decreased by 94% as well as improvement in visual function questionnaire, visual acuity and contrast sensitivity function.[12]

An alternative procedure is YAG vitreolysis, which acts as a photodisruptor, breaking down a floater into much smaller opacities.[15] The targeted floater must be clinically appreciable, like a Weiss ring, and anterior enough from the retina to reduce risk of collateral damage.[16] More than one treatment session may be needed to reduce symptoms, and even then there are some patients that remain symptomatic from the smaller opacities.[8]

PVD. This process occurs as gel liquefaction increases and the adhesion at the vitreoretinal interface weakens. Understanding of the vitreous changes leading up to PVD have

changed dramatically, largely due to advances in imaging. PVD is now recognized as an insidious process occurring over years to decades until the development of complete PVD with vitreous release at the optic nerve.[17,18] Long prior to vitreoretinal separation, there is evidence of lacunae development and vitreoschisis, a lamellar separation in the cortex.[19] Vitreoschisis has been noted as early as the third decade at the mid-peripheral interface.[20]

Recent studies using montaged wide-angle OCT imaging demonstrated evidence of separation in the mid-peripheral retina first, contrary to previous thought that the vitreous releases first in the perimacula.[20,21] This is followed by release in the macula, maintaining adhesion at the fovea. On OCT, this finding is known as vitreomacular adhesion, evidenced in image *Figure 3a*.[22] Subsequently, the fovea will release, followed eventually by the optic nerve, considered a complete PVD, which may be evidenced clinically with a Weiss ring.

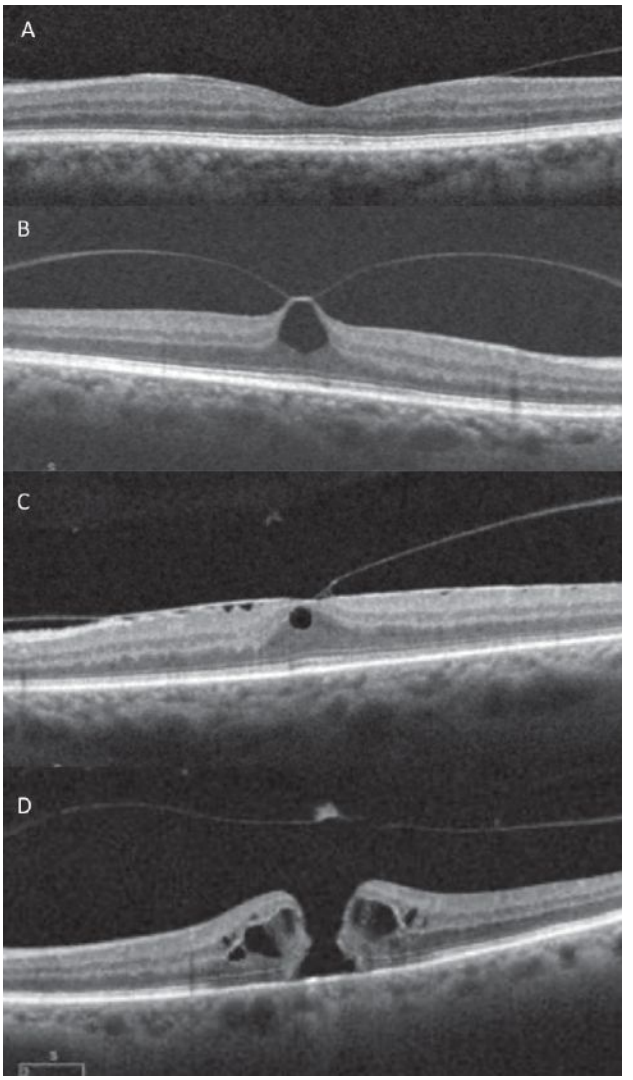

Fig. 3. SD-OCT images of the vitreomacular interface. (A) VMA, (B) focal VMT with pseudocyst, (C) broad VMT with ERM, (D) medium full thickness macular hole with operculum and posterior hyaloid release.

If gel liquefaction occurs faster than vitreoretinal dehiscence, termed *anomalous PVD*, multiple complications can occur. Across the retina, concomitant diseases such as proliferative retinopathy, diabetic macular edema and exudative AMD can be aggravated. In the periphery, the traction from anomalous PVD can result in retinal tear or detachment. The peak risk for rhegamatogenous retinal detachment is in the 50 to 70 age group as a complication of PVD.[23] For an acutely symptomatic PVD, there is a risk for a concomitant retinal break, with or without detachment, signifying need for a thorough retinal exam.

A recent study by Seider and colleagues found an incidence of retinal breaks or retinal detachment in 9.4% in a primary eye care setting.[24] Prior reports suggested higher but have been out of retinal specialty settings. Highest risk characteristics for a simultaneous or delayed retinal break include concomitant vitreous hemorrhage, greater than 3D of myopia, lattice degeneration and a history of break in the other eye.[24-26] Symptoms of photopsia during PVD are often a part of the process and not viewed as a risk factor.[24] Though uncommon, delayed retinal break has been reported in 1.8% to 2.6% of acute PVDs.[24-26] As such, follow-up is always recommended after an acute PVD, most suggesting four to six weeks. For those with risk factors, an additional follow-up may be considered.[25]

At the vitreomacular interface, the antero-posterior force of traction from anomalous PVD can lead to vitreomacular traction (VMT). Unlike VMA, VMT demonstrates a tractional anatomic change to the contour of the fovea without a full-thickness retinal defect.[22] With focal VMT, this may appear as a loss of foveal contour or pseudocyst formation.[22] Broad VMT, a wider area of attachment, more likely will show retinal thickening, cystoid macular edema or fovealschisis.[22]

While some VMT will spontaneously resolve, other patients may develop symptoms such as metamorphopsia or decreased vision, and some will progress on to macular hole. An estimated 5% to 12% of VMT will progress on to full thickness macular hole.[27-29]

Those asymptomatic or with minimal symptoms can be observed. However, in the development of visually significant symptoms or with progression to macular hole, the most successful treatment is vitrectomy. A small (<250µm) macular hole has a near 100% closure rate with vitrectomy and gas tamponade.[30,31] With vitrectomy, gas tamponade and internal limiting membrane peel are often needed to secure a return to proper foveal contour. An alternative treatment option was the intravitreal injection of Jetra (ocriplasmin, ThromboGenics), which produced proteolytic activity to the proteins at the interface to induce PVD. The drug's high cost, adverse effects and at best 50% success rate likely

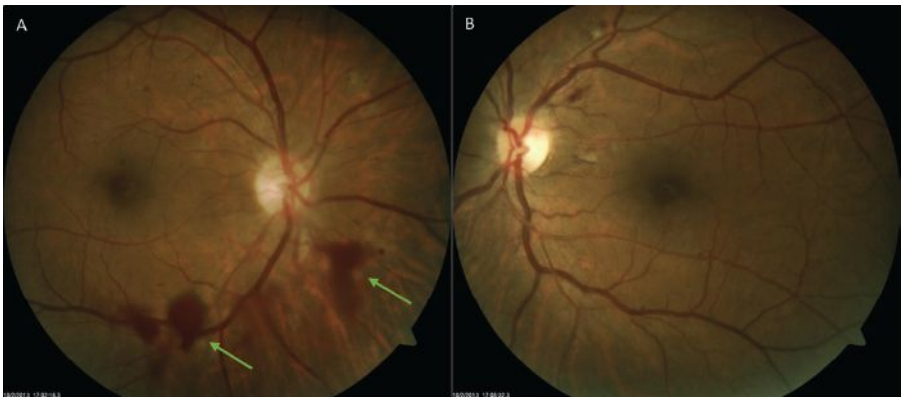

Fig. 4. An inferior localized vitreous hemorrhage (A) due to neovascularization elsewhere from diabetic retinopathy. Note the hazy quality from the hemorrhage when compared with OS (B) with moderate nonproliferative diabetic retinopathy.

contributed to its discontinuation in 2020.[32] The concept of pneumatic vitreolysis, injection of a small gas (C_3F_8 or SF_6) bubble, has gained interest showing highly successful release of VMT in small non-controlled trials.[33,34] This led to DRCR Retina Network clinical trials AG (VMT) and AH (macular hole), which were ultimately discontinued due to high rates of retinal tear and detachment.[35] To date, vitrectomy remains the only treatment option for VMT and macular hole.

Pigment. Sometimes during anomalous PVD, pigmented cells, known as Shafer's sign or "tobacco dust" sign, can be observed in the anterior vitreous. These cells are usually brown in color and are thought to represent released retinal pigment epithelial cells into the vitreous from a retinal break.[8] Multiple studies have shown that the presence of a Shafer's sign is almost pathognomonic for a retinal break or rhegmatogenous detachment and a clinician should be on high alert if noted on an examination.

One United Kingdom study found that approximately 95% of patients with an acute PVD and a positive Shafer's sign had a retinal break.[36] Additionally, out of 113 patients referred for a retinal detachment repair, 111 had vitreous pigment.[36] Because these cells are located within the anterior vitreous, they are best viewed at the slit lamp by using a narrow, bright beam and focusing just posterior to the lens. It may be helpful to have the patient look up or down in order to better observe the cells moving within the vitreous.

Hemorrhage. Acute-onset floaters may be the only presenting symptom with an acute vitreous hemorrhage, which makes it a challenge to discern from history alone. Some will notice a red hue to the new floaters. Others, particularly with a dense hemorrhage, may not notice floaters at all but rather experience a sudden loss of vision. Similarly, vitreous hemorrhage can vary clinically, appearing either diffuse or localized (*Figure 4*). A subtle vitreous hemorrhage may be most appreciable inferiorly with settling of red blood cells. Blood confined to the sub-hyaloid space will appear boat-shaped. In a dense vitreous hemorrhage, the media becomes hazy making visualization of the retina challenging. Thorough examination of the fellow eye is key to revealing clues toward the etiology. B-scan may also be necessitated in cases of poor retinal view. When blood is noted in the vitreous, it often boils down to one of two causes: abnormal vessels from proliferative retinopathy or a rupture of normal retinal blood vessels.[37,38]

Proliferative retinopathy. This is the most common cause of blood within the vitreous and refers to the growth of new blood vessels within the eye in response to capillary nonperfusion.[38] This neovascularization is most often seen in diabetic retinopathy, but also occurs in numerous other conditions such as retinal vascular occlusions, sickle cell disease and retinopathy of prematurity.[38] In an ischemic state, VEGF and other cytokines are released within the eye causing the formation of these abnormal blood vessels at the vitreoretinal interface. However, these new vessels are fragile and prone to shearing and leakage

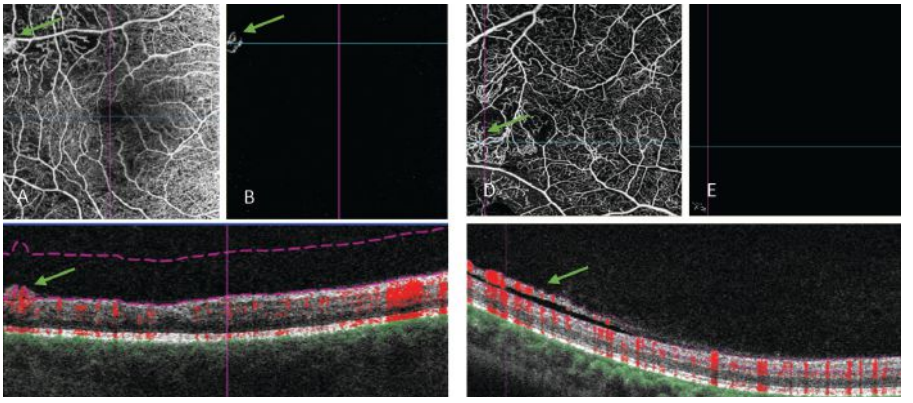

Fig. 5. OCT angiography of retinal neovascularization. (A) An angiogram of the retinal vessels. (B) VRI slab that segments out the vitreoretinal interface. This scan should normally be black, as this area is void of blood vessels in a healthy individual. (C) Corresponding B-scan, where you can see the abnormal vessels fully protruding past the posterior hyaloid and into the vitreous. The flow overlay tool shows red coloring within these vessels corresponding to active movement or flow within. (D, E and F) Another OCT-A of flat neovascularization that extends along the posterior vitreous surface. (F) Because of how it is improperly segmented in the B-scan (purple lines), the abnormal blood vessels do not show in the VRI slab.

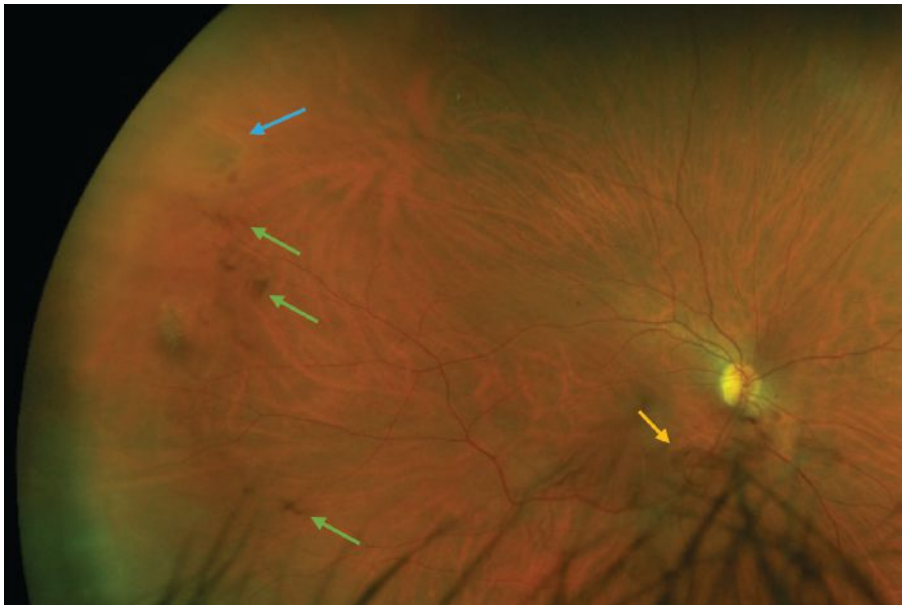

Fig. 6. A patient with an acutely symptomatic PVD and concomitant retinal horseshoe tear (blue arrow) and vitreous hemorrhage (green arrows). The Weiss ring can be seen inferior to the optic nerve head (yellow arrow).

when they experience traction from the vitreous, which can ultimately lead to a vitreous hemorrhage.[39]

Both OCT and OCT-A imaging can be helpful in diagnosing neovascularization and demonstrating its relationship with the vitreous. On an OCT B-scan, neovascularization will breach the internal limiting membrane, extend into the vitreous and can take on a number of appearances (*Figure 5*).[40] On OCT-A, extension of these vessels into the vitreous cavity can be seen on the vitreoretinal interface slab, one that should normally be black and devoid of blood flow. Flow within these vessels is also evidenced by using the flow overlay tool on the corresponding B-scan.

Hemorrhagic PVD. After proliferative retinopathy, the next most common cause of vitreous hemorrhage is from a disruption to normal blood vessels either via PVD, retinal break or trauma.[37,38]

When vitreous hemorrhage develops as a result of the traction that occurs during PVD, it signals a high risk of concomitant or delayed retinal break approximately 60% to 70% risk.[38,41] As such, either closer observation or a retina specialist referral is indicated depending on your comfort level and ability to visualize the retina. Also, be mindful that patients on anticoagulants like aspirin, coumadin and warfarin have a higher propensity for vitreous hemorrhage from PVD.[42]

In most cases, hemorrhage will clear on its own, typically at the rate of approximately 1% per day.[37] Younger eyes with a more gelatinous vitreous take longer to clear.[37] Vitrectomy indications are dependent on etiology. This procedure may be considered in the case of dense hemorrhage with poor retinal visualization or non-clearing hemorrhage (two to six months' duration) to reduce risk of complications.[38,41]

Asteroid hyalosis. This is an age-related condition in which small, yellow-white refractile bodies are suspended throughout an intact vitreous. These opacities are made up of calcium-phospholipid complexes and the exact etiology remains unknown.[43] It's fairly common, found in about 1% to 2% of the population, and its incidence increases with age.[44,45] It is usually unilateral but can be bilateral and in the past, it was thought to have an association with systemic conditions, including diabetes, hypertension and hyperlipidemia. It should be noted that recent epidemiologic studies have not confirmed a significant correlation.[44-46]

Unlike the other vitreous opacities described here, asteroid hyalosis has little effect on vision and affected patients are generally asymptomatic.[43] Sometimes, it can be difficult for a practitioner to accurately view and assess the fundus, and other techniques such as fluorescein angiography, B-scan, OCT and OCT-A should be used. For the few cases that are symptomatic or hinder a necessary view of the posterior pole, a vitrectomy may be indicated.

Similar in appearance to asteroid hyalosis, synchysis scintillans also presents as yellow-white highly refractile bodies within the vitreous. However, this entity is made up of cholesterol deposits and is a degenerative process found in severely diseased eyes.[47,48] Unlike asteroid

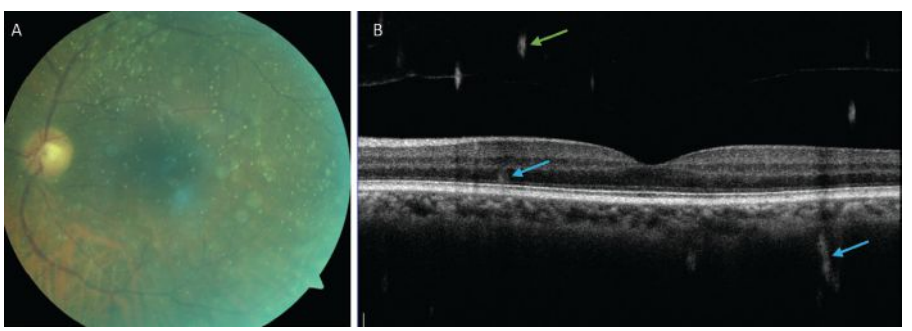

Fig. 7. (A) Fundus photo showing asteroid hyalosis and demonstrating the limited visibility of the retina. (B) An OCT B-scan of the retina with asteroid bodies visible in the vitreous (green arrows) and artifacts from the asteroid bodies within the retina and choroid (blue arrows).

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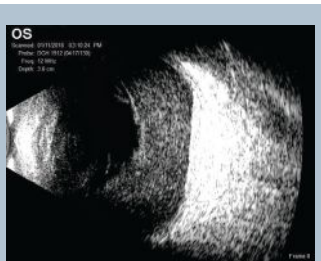
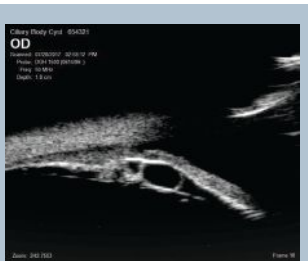
Scanmate Flex A-scan, showing the Flex user interface. The basic interface is the same for all three probe types.



Visualizing vitreous hemorrhaging with the Scanmate Flex B-Scan probe.



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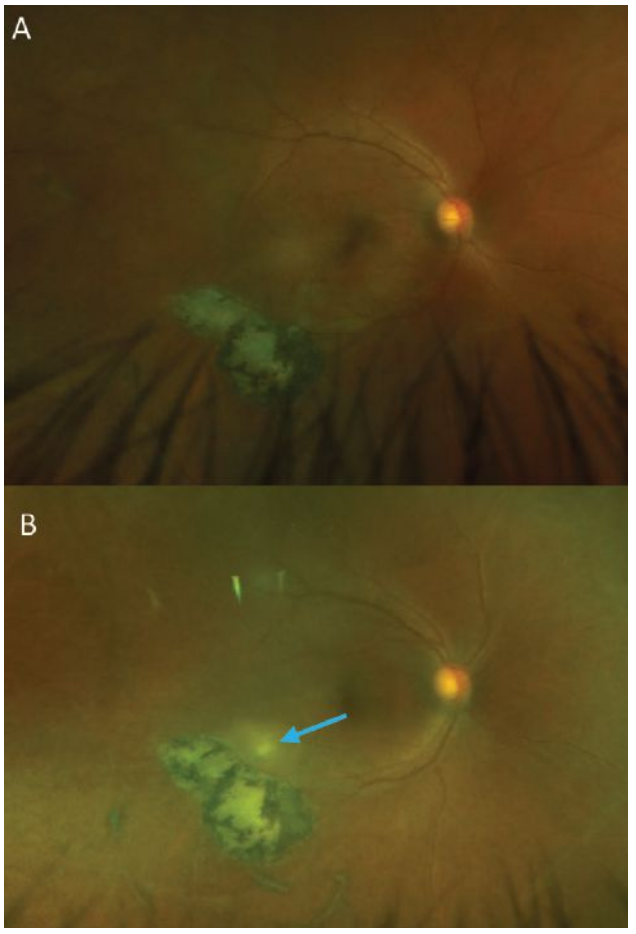

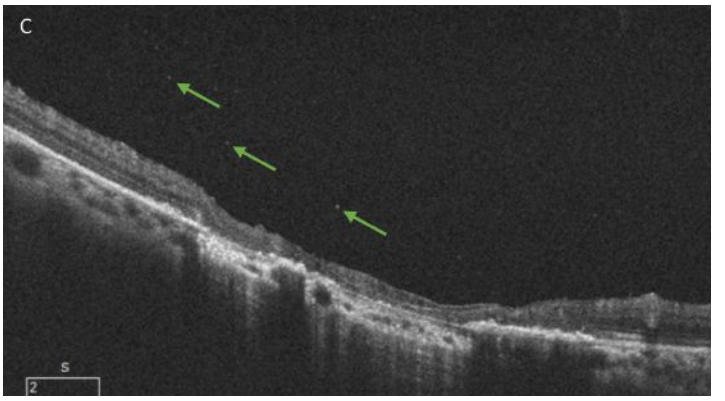

Fig. 8. (A) A patient with an inactive toxoplasmosis gondii chorioretinal scar. (B) The same patient with a reactivated infection. The new, white retinal lesion can be seen just superior to the existing scar. A mild vitritis is evident from the haziness of the photo compared to the original. Small cells can be seen on close inspection of the optos photo as well as within the vitreous on the OCT B-scan (green arrows, C). This patient was seen very soon after acutely noticing new floaters in their vision and the vitritis in this case is more subtle than is often seen with toxoplasmosis.

hyalosis, synchysis scintillans opacities move freely within a liquified vitreous and will settle inferiorly when the eye is not moving.[43]

Vitritis/white blood cells. Acute-onset floaters may be the primary presenting symptom of ocular inflammation or infection, particularly in the case of vitritis, clinically evidenced by white blood cells within the vitreous. Whether physiologic, like floaters, or pathologic, an opacity impacts the inherent structural translucency. They may be clinically subtle or apparent and likewise for patients either visually symptomatic or asymptomatic. Causes include infection, inflammation, uveitis masqueraders, white dot syndromes and idiopathic disease (*Table 1*).

The appearance of these cells is also highly variable depending on the specific cause and length of time they have been present. When a patient is newly symptomatic for floaters, cells may be small, few in number and difficult to appreciate. Cells can also aggregate inferiorly and be considered "snowballs" or spread across the ora serrata and pars plana in what is known as "snowbanking."[50]

TABLE 1. POTENTIAL CAUSES OF VITRITIS *(Note: this list may not include all causes)*

Infectious	Inflammatory	Other	White Dot Syndromes
· Tuberculosis · Syphilis · Lyme disease · Progressive outer retinal necrosis · Toxocariasis · Acute retinal necosis · Toxoplasmosis · Diffuse unilateral subacute neuroretinitis · Bartonella · Leprosy · Whipple's disease · Cryptococosis · Endopthalmitis	· Multiple sclerosis · Sarcoidosis · Sjögren's syndrome · Behcet's disease · Tubulointerstitial nephritis and uveitis syndrome · Blau syndrome	· Neoplasms (particularly lymphoma) · Intraocular foreign body · Amyloidosis	· Acute posterior multifocal placoid pigment epitheliopathy · Birdshot retinopathy · Serpiginous choroidopathy · Multifocal choroiditis with panuveitis · Multiple evanescent white dot syndrome

Depending on the specific cause, deeper structures of the eye such as the retina or choroid are often involved as well.

Because vitritis is a nonspecific finding, workup includes a very detailed history, taking into account patient demographics, a full history of present illness and a comprehensive medical history. Bloodwork is often necessary for a definitive diagnosis, and the amount of tests ordered can be considerable. Often, this list can be narrowed down based on the history and physical examination. Take for example, a patient who presented with acute floaters (*Figure 8*). The clinical exam revealed a classic appearance consistent with active ocular toxoplasmosis gondii chorioretinitis infection. Toxoplasmosis is the most common cause of posterior uveitis and is associated with the classic "headlights in fog" appearance, with a new focal, white lesion visible through an often dense vitritis.[50] Bloodwork was positive for elevated toxoplasmosis antibody titers and negative for HIV, which is important to rule out, as reactivation of these lesions can occur in an immunocompromised state.

Takeaways

While often overlooked, the vitreous via its unique aging process, as well as its interaction with the retina, is susceptible to a variety of different conditions. Although bothersome, many vitreous opacities are harmless and need only patient education and yearly monitoring. On the other hand, entities such as pigment, hemorrhage or white blood cells within the vitreous can be cause for serious concern. It is our role to be able to differentiate the benign from the harmful and successfully manage accordingly. ■

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NUTRITION AND THE RETINA: HELP PATIENTS HELP THEMSELVES

The key to fostering neuroprotection from a variety of posterior segment diseases may lay in promoting dietary changes and supplementation.

BY SAIDIVYA KOMMA, OD
KERNERSVILLE, NC

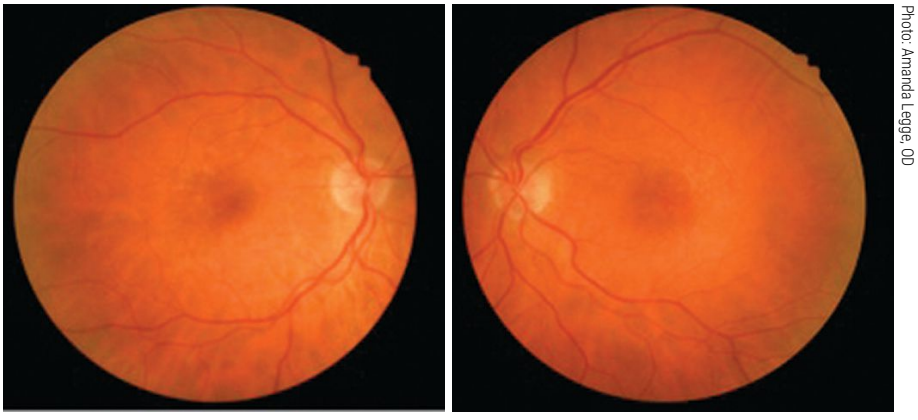

Photo: Amanda Legge, OD

This patient was diagnosed with early dry AMD in both eyes.

A question I get asked daily at the end of an exam is, "Doc, what can I do for my eye health?" Although the obvious answer lies in overall lifestyle maintenance efforts such as diet and exercise, it is important to tailor these recommendations based on the patient's unique ocular status. This step becomes increasingly crucial with the prevalence of eye diseases like age-related macular degeneration (AMD) and diabetic retinopathy (DR).

As of 2019, about 13% of US adults over the age of 40 were diagnosed with AMD.[1] It is the leading cause of blindness in adults over the age of 60 worldwide, with it affecting more than 190 million people in the global population as of 2020 and an estimated increase in prevalence to 288 million people by 2040.[2] DR was estimated to affect 463 million people worldwide in 2020, with a projected increase in prevalence to 700 million by 2045, making it the leading cause of preventable blindness in working age adults.[3]

AMD is estimated to cost the global healthcare system more than $300 billion at this time, while the ADA found that diabetes alone cost the US healthcare system $327 billion in 2017.[2,4] With the projected growth in prevalence of these diseases, the burden on the healthcare system will also continue to grow in the absence of preventative measures. The projected increase in prevalence correlates with the aging population, with age being a key risk factor in retinal disease and many other chronic health conditions.

Although aging is the result of multiple synergistic factors, a major factor is oxidative damage. The body releases reactive oxygen species during metabolism. A build-up of reactive oxygen species in cells can damage the DNA, RNA and proteins, eventually leading to cell death.[5] The retina, being an extension of the central nervous system, has highly metabolic processes.[6] Within the retina, the outer segments of the photoreceptors suffer from the most

About the authors

Dr. Komma serves as an externship coordinator and staff optometrist at the Kernersville VA Health Care Center. She graduated from the Pennsylvania College of Optometry and completed her ocular disease residency at the Salisbury VA Medical Center. She is a fellow of the American Academy of Optometry. She has no financial interests to disclose.

oxidative damage due to the process of phototransduction.[7] If these proinflammatory pathways are not offset by compensatory antioxidants, then the cells undergo prolonged stress and, ultimately, death.[5,8] The key to retinal neuroprotection may lay in promoting redox homeostasis to counteract the damaging effects of metabolism through dietary changes. Numerous studies have been conducted regarding the benefits of dietary supplements for eye health with a few notably consistent results.[8,9] Below are the major supplements that have been shown to delay, or in some cases possibly reverse, retinal disease.

Vitamins

These supplements appear to offer significant ocular benefits for patients. Bring this research up the next time patients ask for advice.

Vitamin A. This fat-soluble vitamin, in the form of 11-cis-retinal, is essential for normal phototransduction function in the retina.[10] The recommended daily dose for children is 400µg to 500µg (about 1,500IU), adult men 900µg (3,000IU) and non-pregnant adult women 700µg (2,333IU).[10] Low serum levels of vitamin A have been associated with nyctalopia, a condition that has been shown to be reversible with adequate supplementation, as well as Bitot's spots on the conjunctiva.[11]

Vitamin A deficiencies affecting the retina can be monitored through fundus autofluorescence, in which a deficient patient would exhibit generalized hypoautofluoresence.[10] Vitamin A supplementation in the form of retinyl palmitate has been shown to slow down the progression of retinitis pigmentosa at a dose of 15,000IU.[12]

In contrast, vitamin A supplementation has been shown to increase the risk of progression in Stargardt's patients, so it should not be recommended in these cases.[10] High-dose vitamin A supplementation is not recommended for routine patients due to the potential for toxicity, which can manifest as pseudotumor cerebri in the eyes.

TABLE 1. NUTRIENT-RICH FOODS[13]

Nutrients	Food
Lutein Zeaxanthin	Kale, spinach, romaine lettuce, broccoli, brussels sprouts, squash, pumpkin, avocado, corn Oranges, papaya, nectarines, corn Egg yolk
Omega-3 fatty acids	Fatty fish (salmon, mackerel, tuna, herring, sardines) Nuts and seeds (flaxseed, chia seeds, walnuts) Plant oils (soybean, canola)
Vitamin A	Herring, salmon, beef liver and other organ meats Spinach, sweet potatoes, carrots, broccoli, winter squash, cantaloupe, mangos and apricots Milk, cheese Eggs
Beta-carotene	Yellow, orange and green leafy fruits and vegetables (spinach, sweet potatoes, carrots, broccoli, winter squash, lettuce, tomatoes, cantaloupe)
Astaxanthin	Microalgae, yeast, salmon, trout, krill, crayfish
Vitamin C	Citrus fruits (oranges, grapefruit) Red and green peppers, tomatoes, broccoli, potatoes Strawberries, kiwi
Vitamin E	Vegetable oils (wheat germ, sunflower, safflower, corn, soybean) Nuts and seeds (peanuts, hazelnuts, almonds, sunflower seeds) Green vegetables (spinach, broccoli)
Zinc	Oysters Red meat, crab, lobsters Legumes, nuts, whole grains, eggs, dairy
Copper	Shellfish, seeds and nuts, organ meats, wheat-bran cereals, whole-grain products and chocolate (in moderation)

Vitamin C. This water-soluble vitamin is the primary antioxidant in the eye, and it can be found in the lens, aqueous, vitreous and cornea.[8] Dietary supplementation can be achieved in the forms of sodium ascorbate and ascorbic acid, although the former is not recommended for hypertensive patients due to the sodium content.

The recommended daily dose of vitamin C for adult males is 90mg and non-pregnant females is 75mg. Smokers need 35mg/day more than non-smokers.[13] Ascorbic acid has been shown to suppress vascular endothelial growth factor (VEGF) expression in the retinal pigmented epithelium (RPE), suggesting that it may be beneficial in DR.[14]

The AREDS study has also shown that 500mg/day of vitamin C supplementation reduced the risk of AMD progression. Due to the water-soluble nature of this vitamin, any excess intake is excreted in the urine, so there is low risk for overdose. This also suggests that vitamin C should be taken in equal doses throughout the day, rather than a single large dose per day. However, unless there is a deficiency, there are no clear benefits to high-dose vitamin C supplementation in routine patients at this time.[15]

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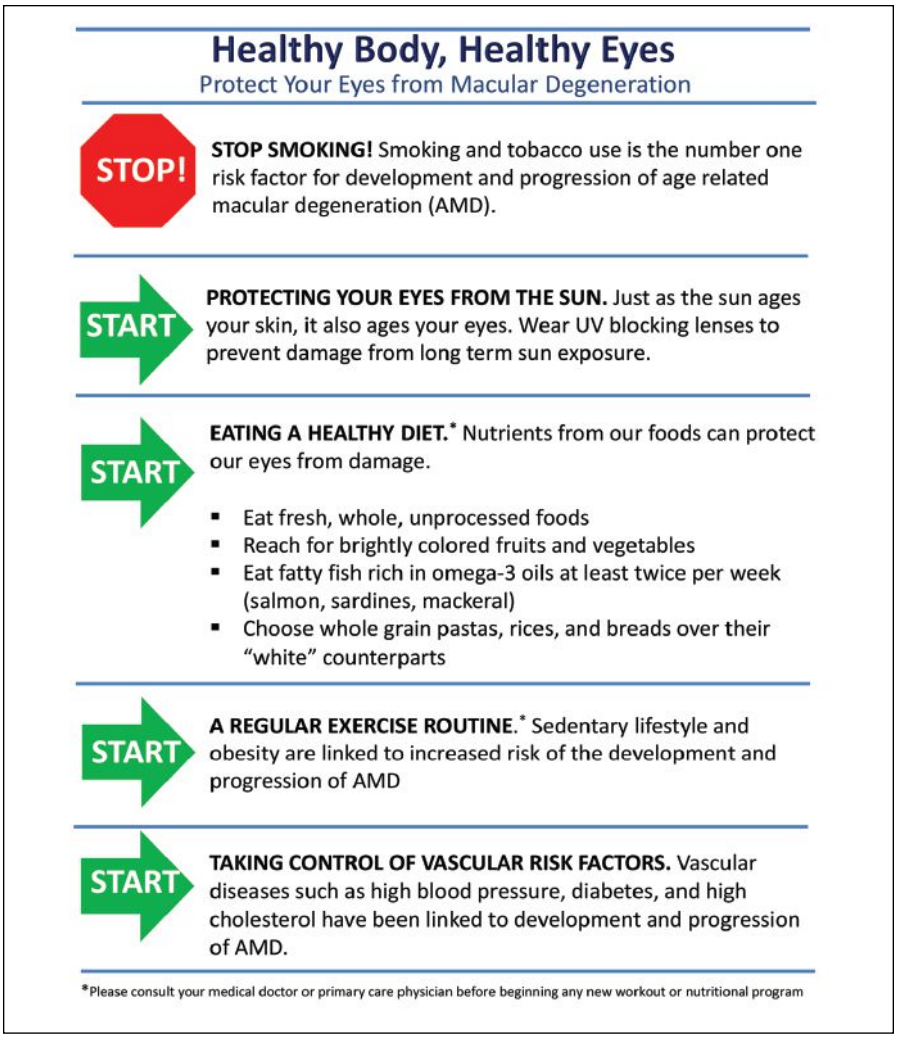

Healthy Body, Healthy Eyes

Protect Your Eyes from Macular Degeneration

STOP! **STOP SMOKING!** Smoking and tobacco use is the number one risk factor for development and progression of age related macular degeneration (AMD).

START **PROTECTING YOUR EYES FROM THE SUN.** Just as the sun ages your skin, it also ages your eyes. Wear UV blocking lenses to prevent damage from long term sun exposure.

START **EATING A HEALTHY DIET.*** Nutrients from our foods can protect our eyes from damage.

- Eat fresh, whole, unprocessed foods
- Reach for brightly colored fruits and vegetables
- Eat fatty fish rich in omega-3 oils at least twice per week (salmon, sardines, mackeral)
- Choose whole grain pastas, rices, and breads over their "white" counterparts

START **A REGULAR EXERCISE ROUTINE.*** Sedentary lifestyle and obesity are linked to increased risk of the development and progression of AMD

START **TAKING CONTROL OF VASCULAR RISK FACTORS.** Vascular diseases such as high blood pressure, diabetes, and high cholesterol have been linked to development and progression of AMD.

*Please consult your medical doctor or primary care physician before beginning any new workout or nutritional program

Give your patients a reference sheet of AMD lifestyle modifications such as this one. A full-page version of this form is available for download in the online version of this article.

Vitamin E. This fat-soluble vitamin is available in several isomers, but the one used in the AREDS formulation is alpha-tocopherol and has shown to decrease overall oxidative stress and inflammation by scavenging free radicals.[9] The recommended daily dose in adults, both female and male, is 15mg.[13] Avoid vitamin E in retinitis pigmentosa patients as it has been shown to increase the rate of progression.[12] This should also be avoided in patients on blood thinners since it increases their risk for bleeding and hemorrhagic stroke.[12]

There are conflicting results regarding vitamin E and its relationship to AMD. The Beaver Dam Eye Study found that a vitamin E deficiency was linked with large drusen formation the Blue Mountains Eye Study showed increased risk for AMD progression with vitamin E supplementation, and the Eye Disease Case-Control Study found no significant difference.[9] Given this, there is no compelling evidence to recommend high-dose vitamin E supplementation on its own to patients. However, due to its excellent anti-oxidative effects, many supplements include vitamin E in the formulation, including the AREDS formula with 400IU.

Carotenoids

Eyecare professionals should educate patients on these macula-enriching molecules.

Lutein and zeaxanthin. These pigmented xanthophylls are heavily concentrated in the macula and protect the area from oxidative stress caused by blue and UV light exposure.[8] The recommended daily dose in adults is 10mg for lutein and 2mg for zeaxanthin.[13] Lutein and zeaxanthin supplementation has shown to increase macular pigment optical density, improve visual acuity and increase contrast sensitivity on multifocal electroretinograms in both normal and diseased retinas.[8,9]

The Blue Mountains Eye Study also found that patients with high serum lutein and zeaxanthin had a significantly lower risk of AMD.[9] In animal models, zeaxanthin has been shown to inhibit DR.[8] Xanthophyll supplementation in patients with type 1 and type 2 diabetes has been shown to improve overall visual function and decrease symptoms of peripheral neuropathy, regardless of the level of retinopathy.[16] The benefits of lutein and zeaxanthin impact both diseased and healthy retinas with limited adverse reactions, so these may be one of the few supplements that can be recommended to all patients interested in ocular health maintenance and should be emphasized in patients at higher risk for retinal disease.

Beta-carotene. This is yet another potent antioxidant present in small amounts in the RPE and is a precursor to vitamin A. Since beta-carotene increases the risk for lung cancer in smokers, it was removed from the original AREDS formulation and replaced with lutein and zeaxanthin in AREDS2, which proved to be slightly more efficacious.[9] Although beta-carotene is a precursor of vitamin A, it does not carry the same risks as preformed vitamin A does in high doses.[12]

The recommended daily dose for adults is 15mg.[13] Separate beta-carotene supplementation seems to have little benefit at this time, given the prevalence, efficacy and improved safety profile of the other carotenoids. In addition, excess beta-carotene

(often found in supplement pills) has shown to be associated with an increased risk of AMD.[17]

Astaxanthin. One of the more powerful carotenoids is astaxanthin. This carotenoid has a chemical structure that fully spans cellular membranes with potent antioxidant and anti-inflammatory properties. By scavenging measures, it is a far stronger antioxidant than zeaxanthin, canthaxanthin, lutein, B-carotene and alpha-tocopherol.

Astaxanthin neutralizes single oxygen molecules and scavenges radicals to prevent chain reactions, thus preserving membrane structure. This carotenoid also crosses both the blood-retinal and blood-brain barriers and may be beneficial in cardiovascular, immune, inflammatory and neurodegenerative diseases.[18] While there is no recommended daily allowance for astaxanthin, studies have shown positive effects with between 6mg to 12mg daily.[19]

Fatty Acids

Proper supplementation of these in inflammatory diseases such as dry eye can offer patients relief and other advantages.

Omega-3s (docosahexanoic acid; DHA and eicosapentaenoic acid; EPA). These fatty acids form the phospholipid bilayer and outer segments of photoreceptors, making them crucial in retinal structure and function.[8,9] DHA and EPA are long-chain polyunsaturated fatty acids, with the most abundant in the retina being DHA.[8]

DHA levels were significantly lower in donor eyes with AMD in comparison to normal eyes.[8] Although studies have shown a reduced risk in choroidal neovascular membrane formation in red blood cells with high DHA concentrations, no improvement was noted in contrast sensitivity with DHA supplementation.[8] This suggests that omega-3 supplementation cannot improve retinal function, but may protect it from further decline. One study found that consuming at least 500mg per day of long-chain polyunsaturated fatty acids reduced the risk of sight-threatening diabetic retinopathy by 48%.[20]

The recommended daily dose is 1.6g for adult males and 1.1g for adult females.[13] Omega-3 supplementation has also been shown to help with vitamin A absorption in retinitis pigmentosa patients.[12] Take caution when using omega-3 supplementation in patients with clotting disorders or on blood thinners since high doses are known to increase prothrombin time.[8]

TABLE 3. DAILY DOSAGE RECOMMENDATIONS[13]

Nutrients	Adult Dose	
	Males	*Females*
Vitamin A	900µg (3,000IU)	700µg (2,333IU)
Vitamin C	90mg	75mg
Vitamin E	15mg	15mg
Lutein Zeaxanthin	10mg 2mg	10mg 2mg
Beta-carotene	15mg	15mg
Omega-3 Fatty Acids	1.6g	1.1g
Zinc	11mg	8mg

Minerals

The trace mineral zinc is necessary in over 300 enzymatic reactions, making it an important part of phototransduction.[9] Low levels of zinc have been associated with decreased night vision and RPE degradation.[8] The recommended daily dose is 11mg for adult males and 8mg for adult females.[13] Zinc can limit the absorption of fluoroquinolones and tetracyclines, while diuretics can limit the absorption of zinc.[13] It has been shown to reduce the risk of AMD progression with the exception of certain genetic variations of complement factor H, so it may be worth considering genetic testing to guide supplementation decisions in these patients.[9]

Zinc reduces the amount of copper your body absorbs, and high doses of zinc (like in AREDS) can cause a copper deficiency. For that reason, it is recommended to take 2mg of copper along with a zinc supplement.

The Mediterranean Diet

The Mediterranean diet has garnered a lot of attention for its promotion of longevity and chronic disease prevention. Studies have shown that adhering to this diet reduces the risk of cancer,

TABLE 2. AREDS VS. AREDS2 FORMULATION FOR AMD

Nutrient	AREDS formula*	AREDS2 formula
Vitamin C	500mg	500mg
Vitamin E	400IU	400IU
Beta-carotene	15mg	-
Copper (cupric oxide)**	2mg	2mg
Lutein	-	10mg
Zeaxanthin	-	2mg
Zinc	80mg	80mg

* Not recommended for current or former smokers.

** Added to avoid zinc-related copper deficiency.

Source: AREDS/AREDS2 clinical trials. National Eye Insistutute. Last updated November 19, 2020. Accessed April 25, 2023. www.nei.nih.gov/research/clinical-trials/age-related-eye-disease-studies-aredsareds2/about-areds-and-areds2.

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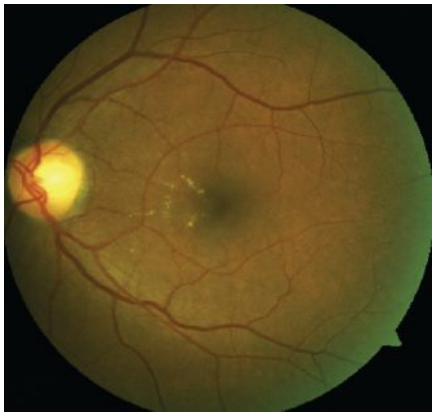

This fundus image shows clinically significant macular edema with best-corrected visual acuity of 20/20.

cardiovascular disease, and cognitive decline.[21,22] In fact, the Greek island of Ikaria and Italian island of Sardinia make up two of the five blue zones, meaning they contain one of the highest concentrations of centenarians.[22] So, what exactly is this heart-healthy diet made of?

- Olive oil, vegetables (leafy greens), fruits, breads/cereals: one to two servings per every meal
- Nuts (walnuts, hazelnuts, almonds, pistachios), dairy: one to two servings daily
- Legumes, fatty fish/seafood: two or more servings weekly
- Poultry, eggs: two servings weekly
- Red meat, sweets: <two servings/week
- Red wine: in moderation (one to two glasses/day for men; one glass/day for women)

The Mediterranean diet is rich in antioxidants and omega-3 fatty acids, making it ideal for disease prevention and longevity. Additionally, the diet depends on fresh ingredients, which are abundant in that area. Geographic atrophy has been seen to progress slower in patients adhering to a Mediterranean diet, further supporting its anti-inflammatory and neuroprotective characteristics.[23] In contrast, high glycemic diets (such as the Western diet) can lead to the accumulation of cytotoxic advanced glycation end products, which promote AMD and DR progression.[24]

Takeaways

Now that we have the information to share with our patients, how can we convince them to use this knowledge and implement potentially vision-saving lifestyle changes? Old habits tend to die hard, so it's important to emphasize the value of starting small. For example, swapping white bread with whole grain bread or eliminating desserts can significantly lower the patient's glycemic peaks without causing a shock to their dietary habits.[25]

Helping the patient find a convincing purpose to make these changes can also be a strong source of motivation. Visual impairment has been linked with depression, increased risk for falls, and early mortality.[26] Identifying reasons for the patient to keep seeing, whether it's their grandchildren, their hobbies or ability to function independently, may make a difference. The use of educational handouts outlining the relationship between adequate nutrition and eye health can also improve patient compliance.

It is important to consider whether there are any limitations in the patient's systemic health for proper nutrient absorption (*e.g.*, Crohn's disease, pancreatic insufficiency, cirrhosis, celiac disease, frequent use of proton pump inhibitors for gastroesophageal reflux, history of gall bladder removal) and to manage with their primary care provider accordingly.

Most vitamins and minerals are better absorbed by the body through food sources than supplements due to the inherent presence of enzymes and flavonoids.[8] However, supplements must be taken with food to maximize absorption. Comanaging patients with a nutritionist can also promote overall well-being. Although we are fortunate enough to have the tools to treat many retinal conditions today, prevention is always the best cure in the long-run. ■

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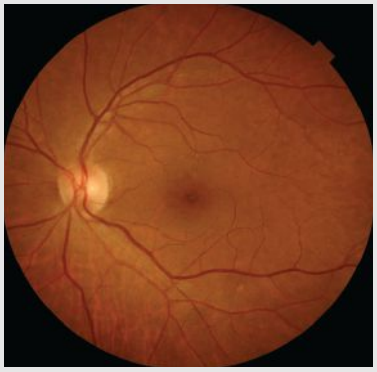
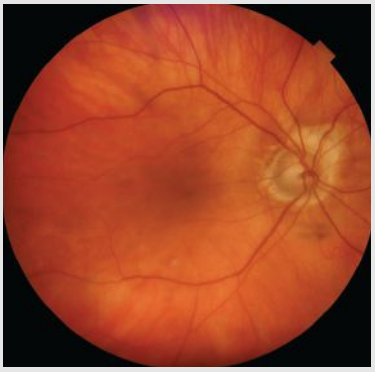
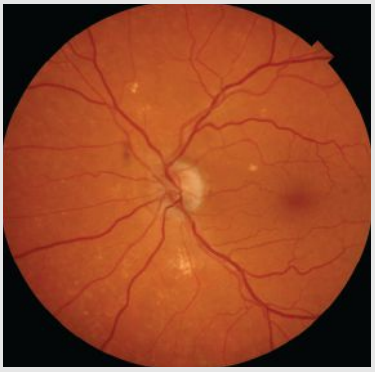


AMD: RPE Pigment Changes



Cataract, (ϕ)2mm Small Pupil



Cataract, Peripheral Drusen

Images courtesy of Silicon Valley Eyecare Optometry and Contact Lenses in Santa Clara, CA.

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CHOROIDAL FOLDS: A NEW WRINKLE IN RETINAL CARE

This often overlooked—yet important—clinical finding can have significant implications for a patient's overall health.

BY SARA WEIDMAYER, OD
ANN ARBOR, MI

While often overlooked as a non-contributory or irrelevant clinical finding, choroidal folds can be associated with serious conditions, both intraocular and extraocular. When we observe choroidal folds clinically, we should never just brush them off but rather ask ourselves what may have caused them to develop in the first place. In most cases, you will find an underlying source if you commit to looking for it.

There's a simple mnemonic for the basic differentials that may be the source of choroidal folds: THIN-RPE. As we discuss each of these broad categories, remember how important it is to think through the underlying condition causing choroidal folds—this is critical for comprehensive care and positive patient outcomes.

Anatomy & Pathophysiology

Choroidal folds are wave-like crinkles in the inner choroid, overlying Bruch's membrane (BM), the retinal pigmented epithelium (RPE) and often the outer retina. They present clinically as multiple thin parallel undulations under the retina, whose peaks and troughs make them appear as alternating light/dark bands where the crests are lighter.

When these striations involve the retina as well, they are called chorioretinal folds. Whether choroidal or chorioretinal, our thought process should be the same, so we will refer to them as choroidal folds for the purposes of this article. They are typically seen confined to the posterior pole, most often radiating horizontally from the optic nerve head through the macula; however, they can also be seen at different orientations and in other locations as well.

This clinical finding has patterns that stand out on the imaging modalities optometrists tend to use in-clinic. A classic wavy pattern of the choroid and RPE—and possibly outer retina—is seen on OCT when the line scans are perpendicular to the folds. On OCT angiography (OCT-A), tigroid lines of reduced signal may be seen, presumably due to some stretching and slightly decreased choriocapillaris perfusion along the folds' trough.[1]

Fluorescein angiography (FA) shows characteristic background choroidal fluorescence changes due to the RPE and choroidal waves; the troughs appear darker since folded RPE is relatively more dense in the trough, whereas the crests appear more hyperfluorescent because the RPE is slightly stretched and relatively more thin at the apex of the folds. This can be seen as early as the arterial phase or early in the arteriovenous phase on FA.[2]

On fundus autofluorescence (FAF), we see the opposite hyper-/hypofluorescent pattern as compared with FA. The lipofuscin in RPE cells hyperau-

About the author **Dr. Weidmayer** practices at the LTC Charles S. Kettles Medical Center, VA Ann Arbor Healthcare System in Ann Arbor, MI. She is also a clinical assistant professor for the Department of Ophthalmology and Visual Sciences, WK Kellogg Eye Center of the University of Michigan. She has no financial disclosures. *The views expressed by the author do not necessarily reflect the position of the US government or Department of Veterans Affairs.*

tofluoresces along the trough where the RPE is relatively more dense and hypoautofluoresces along the crest.

B-scan ultrasonography may not easily detect choroidal folds but can give information about retinal, choroidal and posterior scleral thickness, globe flattening and other relevant information regarding the optic nerve and its sheath, as well as choroidal or retrobulbar lesions that may be contributory. Even without all of these ancillary studies, on our standard fundoscopic exam, choroidal folds stand out when using a red-free (green) filter (*Figure 1*).

Choroidal folds are typically not associated with visual acuity changes or patient complaints but at times can cause blur or metamorphopsia, especially if the folds develop fairly quickly. However, symptoms may arise from refractive shifts or due to the underlying etiology of the folds.

Broadly, choroidal folds form due to changes in tension on the sclera, choroid, BM or RPE. This change in dynamic leads to the development of folds, largely due to the elasticity and collagenous composition of BM, which allows it to corrugate as it shortens.[3] Compressive stress may be due to physical pressure on or around these structures or size changes of these structures. Therefore, when folds are discovered, it is mandatory that they be investigated. The first question should always be, "Why are they there?"

Identifying the Underlying Cause

There are numerous reasons why choroidal folds may form. The mnemonic THIN-RPE can help you remember the broad differentials for the typical causes of choroidal folds, though as you will see, THIN-RPE is not all-inclusive; in fact, it is far from a comprehensive list. We will progress through these etiologies in order of the mnemonic, not in order of frequency, while adding more than the basic THIN-RPE to your list of differentials.

Tumor. Here, we consider choroidal tumors. Choroidal lesions—which take up space, displace the surrounding choroid, compress the sclera from the inside-out and compress the BM-RPE complex from below—can lead to choroidal fold formation.[4] Types of choroidal lesions that can lead to formation include cancerous tumors such as melanomas or metastatic lesions, benign tumors or masses such as osteomas or sclerochoroidal calcifications, vascular lesions such as hemangiomas, inflammatory masses such as granulomas and choroidal infiltration from any number of culprits.

T	Tumor (choroidal)
H	Hypotony, Hyperopia
I	Inflammation, Idiopathic
N	Neovascularization (choroidal)
R	Retrobulbar mass
P	Papilledema
E	Extraocular hardware

While not exactly in the choroid, we must not forget the suprachoroidal space. In this space, suprachoroidal hemorrhages may present. When more localized, they sometimes masquerade as a choroidal mass and can have a similar effect as any choroidal lesion, potentially causing choroidal folds.[5] Be especially suspicious of choroidal lesions if the folds do not emanate from the nerve in the typical radial pattern or if there is any adjacent choroidal thickening or color changes.

Choroidal Folds: A New Wrinkle in Retinal Care

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

Release Date: June 15, 2023

Expiration Date: June 15, 2026

Estimated Time to Complete Activity: two hours

Target Audience: This activity is intended for optometrists engaged in retinal management of choroidal folds.

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

- Comprehend the anatomy and pathophysiology of choroidal folds.
- Recognize choroidal folds via various imaging modalities.
- Identify the underlying causes of choroidal folds.
- Effectively manage patients who present with choroidal folds.

Faculty: Sara Weidmayer, OD

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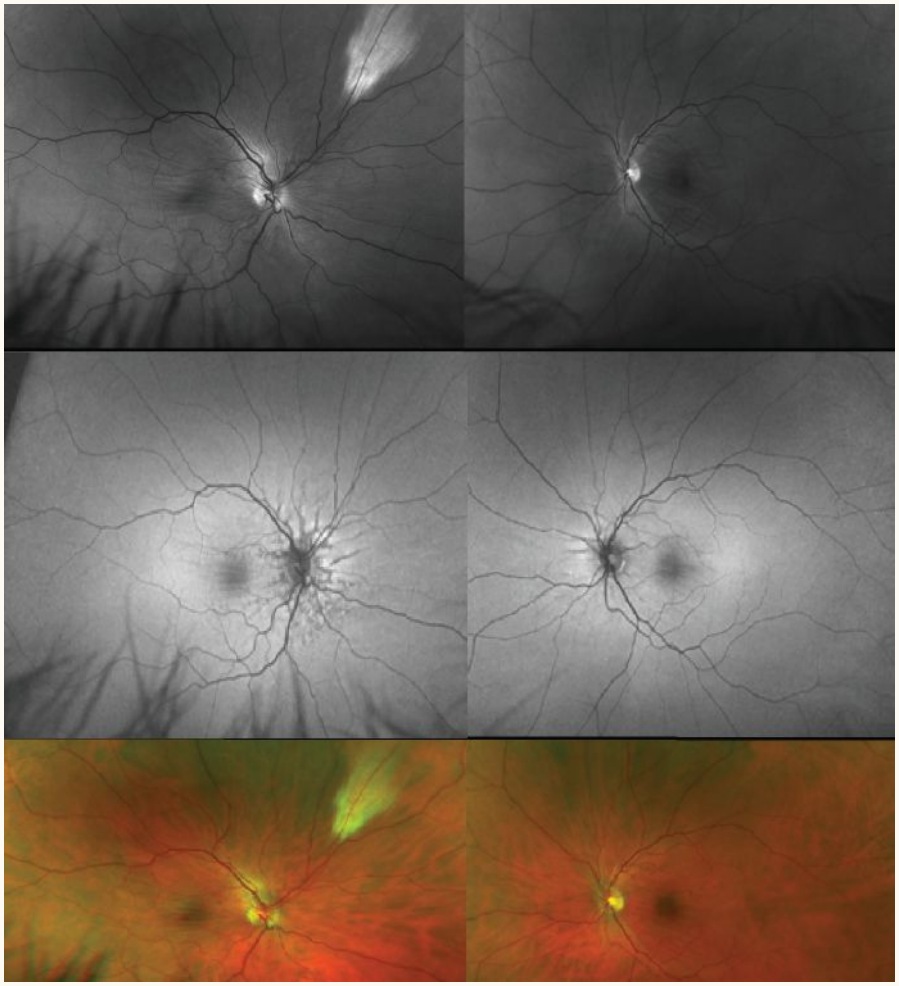

Fig. 1. Bilateral choroidal folds radiating from the disc and through the macula with a red-free (green) filter (top), on FAF (middle) and on fundus photos (bottom). Note this patient has angioid streaks around his optic discs OD>OS and myelinated nerve fibers in the superonasal midperiphery OD.

Hypotony or Hyperopia. Adequate intraocular pressure (IOP) is critical for maintaining the globe shape. When IOP is very low, there is insufficient counterforce inside the eye to balance the scleral compressive force, so the scleral wall bows or collapses inward. This leads to raisining of the chorioretinal tissue, which is particularly vulnerable in the macula where the thicker parafovea radially folds around the very thin fovea causing hypotony maculopathy (*Figure 2*).[2,6]

IOP of about 5mm Hg or less is typically considered hypotony; however, choroidal folds can still occur at pressures that are not quite hypotonous. It is important to note they can occur right before, or along with, the development of other significant side effects of hypotony, such as maculopathy, choroidal effusion or optic disc edema. Thus, choroidal folds seen with low IOP cannot be ignored.

In circumstances of low IOP, the choroidal folds are usually radial around the macula but erratically oriented elsewhere.[3] Low IOP-related choroidal folds also beg the question of why the eye is hypotonous (or nearly hypotonous). Here, we must consider factors such as cyclodialysis or intraocular volume loss related to trauma (*e.g.*, open globe) or intraocular surgery (*e.g.*, wound leaks or over-filtration).[4]

Conditions where choroidal volume is increased or compacted can also lead to choroidal folds. This concept will be touched on in multiple sections, but here we consider hyperopia. Primary hyperopes tend to have shorter axial lengths. This can be conceptualized as the intraocular contents being jammed into an overall smaller package. Choroidal congestion, likely in combination with some redundancy to BM, may cause choroidal folds.[7] Intuitively, this is often observed in nanophthalmos.[8] Secondary, or induced, hyperopia may also be associated with choroidal folds and will be discussed later.

Inflammation or Idiopathic. It is important to consider inflammatory conditions that affect choroidal thickness, either broadly, such as most forms of choroiditis, uveal effusion or Vogt-Koyanagi-Harada, or more locally, such as in solitary choroiditis. Choroidal folds can also be associated with central serous chorioretinopathy or other points along the pachychoroid spectrum where the choroidal volume is generally greater.

Another interesting scenario that could be causative is an increase in the normal pressure within the choroidal vasculature; carotid-cavernous fistulas involve carotid (arterial) blood flow ending up in the cavernous sinus (dural/venous

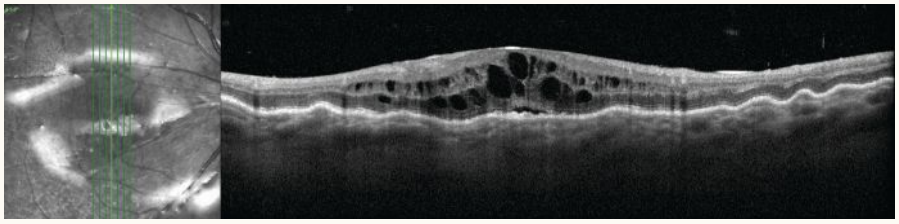

Fig. 2. This eye has hypotony maculopathy with choroidal folds, intraretinal and subretinal fluid and optic disc edema. His IOP was generally between 2mm Hg and 5mm Hg.

sinus). These can be high or low flow, direct or indirect and may form for a number of different reasons. When higher blood pressure is in the cavernous sinus, this elevates the normal venous pressure in the eye. We most commonly think of increases in the episcleral venous pressure as a result of this and the problems it can cause with IOP, but it could also increase the choroidal vortex venous pressure and vein size, creating a pachychoroid size and pressure effect, which we now know can induce folds (*Figure 3*).[9]

Inflammation can also lead to focal or global thickening and later, shrinkage of the sclera itself. One example is posterior scleritis (infectious, inflammatory).[4] When actively inflamed, the scleral wall thickens—often along with the choroid—and folds can be induced in this phase; however, as inflammation resolves, the sclera may contract, and folds can evolve as well. Furthermore, they may remain present indefinitely after scleritis resolves.

The overall rigidity of the sclera can also be decreased in some situations, influencing the eye wall's overall tension and dynamic with the choroid and surrounding structures. We see this especially in younger patients who have long axial lengths—myopes. Myopia isn't often thought of as a cause of choroidal folds; however, axial myopia and other causes of scleral thinning can also be the offender. Similarly, these folds can also be seen emanating from the edges of posterior staphylomas where the scleral wall is thin and bowed posteriorly.[10]

It is easy to assume that choroidal folds are idiopathic; however, idiopathic is a diagnosis of exclusion. Chalking choroidal folds up as idiopathic without a workup is bad practice—and a significant liability. Moreover, only about 15% of choroidal fold cases are actually idiopathic.[11,12]

Even with idiopathic folds, there are some thoughts as to the pathogenesis not being idiopathic at all but rather related to subclinical posterior scleral inflammation in childhood or even *in utero*. This ultimately causes the sclera to contract and choroidal folds to form.[4,13] The patient may also have had a prior—perhaps even insidious—inflammatory condition of the eye or orbit that could have led to scleral thickening or flattening causing folds.[4] Regardless, actual idiopathic cases are typically bilateral, confined to the central posterior pole through the macula, and found in visually asymptomatic, hyperopic males.[4]

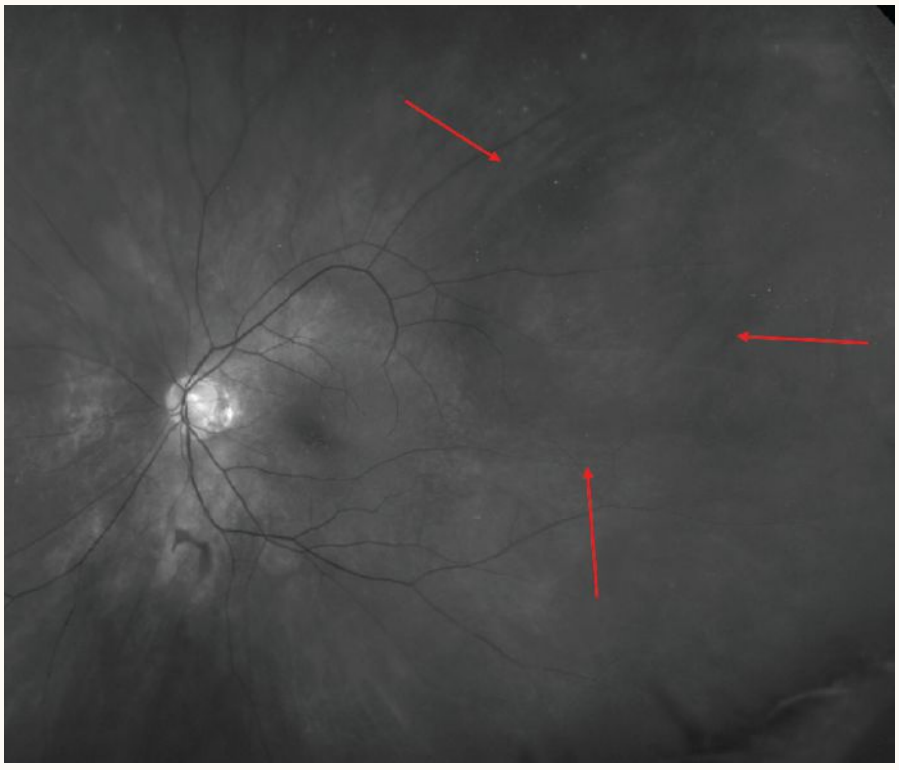

Fig. 3. This eye has choroidal folds surrounding a large vortex vein varix, which is more easily visible with a red-free (green) filter.

Neovascularization. Choroidal neovascularization (CNV) ultimately tends to cause membranes to contract and scar, either spontaneously or after treatment (*e.g.*, intravitreal injections), altering the adjoining anatomy with local choroidal and RPE tightening. Radial choroidal folds from the edges of the CNV can be seen in these cases.[13] As we know, CNV may develop due to a host of conditions. If an optometrist observes early folds but no clear evidence of CNV, such as subretinal fluid or hemorrhage, additional testing (*e.g.*, OCT-A or FA) may be warranted since choroidal folds should raise some suspicion for CNV.

There are other situations where we see outer retina/RPE/BM complex contraction or scarring, which also may be associated with choroidal folds. This has been reported after diode endolaser for a retinal tear, where deep choroidal and scleral thermal injury produced a chorioretinal scar associated with choroidal folds.[14] Likewise, choroidal folds are conceivable with other chorioretinal scars, iatrogenic or otherwise.

Similarly, another condition where there are alterations in BM is angioid streaks (*Figure 1*). Thickened but brittle BM may break, leaving these cracks in the structure. Not only is the local anatomy changed with the angioid streaks, possibly allowing choroidal folds (or CNV) to develop, but as we know, angioid streaks are associated with several metabolic

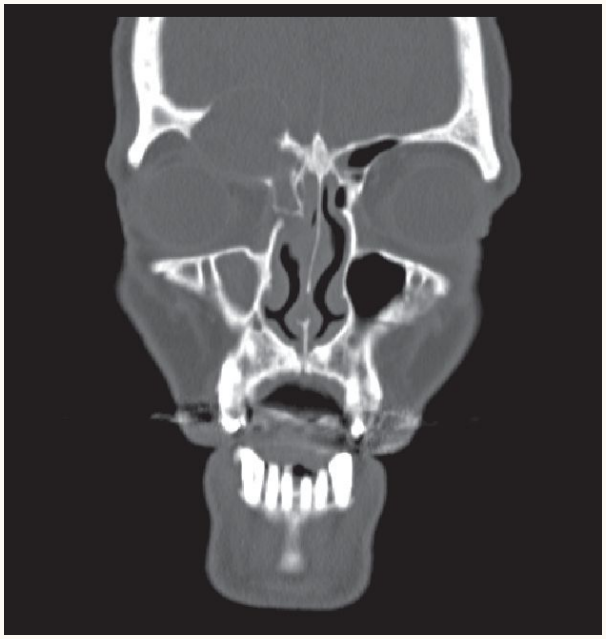

Fig. 4. This coronal CT shows a large right frontal sinus mucocele that eroded through the sinus into the orbit and was making contact with the globe. This is an example of an extraconal intraorbital mass that can cause choroidal folds.

diseases and connective tissue/collagen vascular disorders.[15] These conditions themselves may affect the collagenous and elastic structures of the eye (*e.g.*, BM and the sclera).

Conditions such as pseudoxanthoma elasticum, where the composition of BM or the sclera are abnormal, are prone to progressive calcification with loss of elasticity and fragmentation. This may predispose patients to choroidal folding, lacquer cracks and CNV.[16] Several of these systemic disorders may alter the collagenous structures of the eye. Examples include osteogenesis imperfecta, Marfan syndrome and others. Taking a pathophysiological approach to choroidal fold formation allows optometrists to expand their differentials, and when we think like doctors should, we will no longer need to memorize a laundry list of etiologies.

Retrobulbar mass. Space-occupying problems in the orbit that compress the globe or optic nerve from behind may also be the culprit behind choroidal folds. These may arise from the extraconal or intraconal space or from the optic nerve itself, such as in the case of optic nerve gliomas. There are a variety of retrobulbar mass types. They may include things like orbital tumors (*e.g.*, cavernous hemangiomas, meningiomas, metastatic lesions), mucoceles originating from the paranasal sinuses or retrobulbar hemorrhages (*Figure 4*).

Folds from these sources may be seen in non-central locales, though not necessarily at the site of the retrobulbar mass contact, and at unusual orientations, which should raise some flags of suspicion. Other clues may include refractive shifts, more likely hyperopic shifts from intraconal masses pushing the globe relatively more anterior, or astigmatic shifts from extraconal masses abutting the globe. This is most likely to be a unilateral problem—another red flag.

When it comes to retrobulbar masses, we should also consider an increase in pre-existing orbital tissue—processes like thyroid orbitopathy where we may see an increase in extraocular muscle or orbital fat mass, for example.[4] Other causes of orbital congestion or edema, such as orbital infections (*e.g.*, orbital cellulitis) or inflammation (*e.g.*, idiopathic orbital inflammation) could also cause choroidal folds to form. The lacrimal gland is another structure stationed around the eye that could enlarge and compress the globe. Any type of dacryoadenitis (infectious, infiltrative, inflammatory, neoplastic) would have to be significant; however, even an enlarged lacrimal gland can press on the globe and deform it enough to cause choroidal folds.[17]

Papilledema. Things that may cause the optic nerve head to be congested or swollen may lead to compression of the choroid adjacent to the optic disc—another possible culprit of choroidal folds. Any variety of optic disc edema (*e.g.*, ischemic optic neuropathy, papillitis, optic neuritis) can be the inciting factor, including papilledema—bilateral optic disc edema due to intracranial hypertension. Alarmingly, even in the absence of clinically detectable papilledema, choroidal folds can develop due to intracranial hypertension.[11,12] It is important to restate that there can be choroidal folds due to intracranial hypertension even though the optic discs may look normal.

The optic nerve is surrounded by cerebrospinal fluid (CSF) within its sheath, and increased CSF pressure can be induced by things such as dural venous sinus thrombosis, intracranial masses or idiopathic intracranial hypertension among others. Increased CSF pressure changes tautness on the optic nerve and at the optic nerve head, which causes the posterior globe flattening often seen in papilledema.

In some of these cases, we may also see a hyperopic shift. However, even without overt bilateral optic disc edema, increased—or increasing—CSF pressure may begin to have effects on the globe, including choroidal fold formation, even before any axonal swelling or axoplasmic stasis. And, as with other causes of choroidal folds, these folds may stay permanently, even after disc edema or papilledema resolves.

Here, along with choroidal folds radiating from the disc, the choroidal folds are often concentric, like rippling waves from a stone's throw, around the disc. This orientation of folds may be called Paton's Lines, though Paton's Lines can actually be peripapillary retinal folding rather than, or along with, choroidal folds. Optometrists should consider choroidal folds encircling the optic disc as another warning sign.

A brain MRI, magnetic resonance venogram and lumbar puncture may

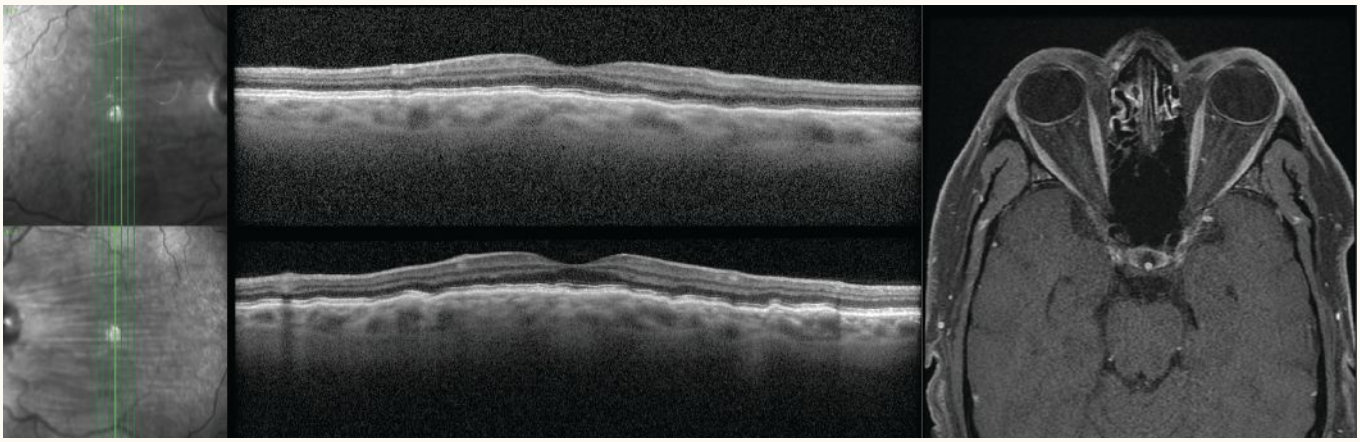

Fig. 5. These bilateral choroidal folds OS>OD were found in a male patient in his 40s. He was asymptomatic, moderately hyperopic and had no notable ocular history. His MRI (axial T1) showed posterior globe flattening OD>OS.

not be necessary to rule out intracranial hypertension in every patient without overt papilledema who has choroidal folds; however, all should be on your radar. In a prospective study, researchers reported that five out of six patients who had choroidal folds without papilledema were found to have intracranial hypertension on lumbar puncture.[12] Though only a small study with several limitations, it should still inform management.

In the absence of another clear cause of choroidal folds, patients should be asked about symptoms of intracranial hypertension such as headaches, nausea, vomiting and transient or positional visual obscurations. Baseline structural and functional measurements of the optic nerves should be taken (*e.g.*, OCT and visual fields) with short-interval serial testing to monitor for change. If available, B-scan ultrasonography can also be helpful here, assessing for posterior globe flattening, retrobulbar optic nerve or nerve sheath changes that may be suggestive of increased CSF pressure (*Figure 5*).

Extraocular hardware. Scleral buckles cinch the globe enough to lessen its circumference. However, narrowing its girth lengthens it in the opposite direction. A scleral buckle stretches the sclera and can cause choroidal folds perpendicular to the buckle's encirclement.[18]

Other examples of extraocular hardware may include glaucoma drainage devices or radiotherapy plaques. While not hardware, trabeculectomy also induces changes in the scleral structure and may lead to folds—even if the eye is not overfiltrated and has normal IOP.

One might also consider strabismus surgery. Relocation of typical muscle insertions could also influence the normal scleral tension balance. We may also see choroidal folds after therapeutic scleral windows have been made.

Takeaways

As optometrists, we must remember that choroidal folds are a key clinical finding and shouldn't be a stand-alone diagnosis—akin to how symptoms are symptoms, not diagnoses, and you must find the reason for them. When we begin to think mechanistically as to why choroidal folds may form, we no longer find ourselves needing to remember lists of possible causes.

A comprehensive knowledge of anatomy and pathophysiology is critical. Even as we use THIN-RPE as a framework for discussing choroidal folds, think outside of the THIN-RPE box about modes of genesis for folds, and internalize the fact that choroidal folds have a plethora of sources beyond the obvious or idiopathic. ■

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1. Choroidal folds may affect all of the following anatomic sites except which?
a. Choroid.
b. RPE.
c. BM.
d. Vitreous.

2. Choroidal folds appear as which of the following on OCT?
a. The line scans are parallel to the folds.
b. The line scans are perpendicular to the folds.
c. They are most evident on retinal nerve fiber layer scans.
d. They are most evident on ganglion cell layer analysis.

3. On FA, the troughs of the choroidal folds appear as which of the following?
a. Hypofluorescent.
b. Hyperfluorescent.
c. Isofluorescent.
d. To leak.

4. On OCT-A, the troughs of the choroidal folds appear to have which of the following?
a. Evidence of neovascularization.
b. Increased signal.
c. Decreased signal.
d. No change in signal.

5. On FAF, the troughs of the choroidal folds appear as which of the following?
a. Hypoautofluorescent.
b. Hyperautofluorescent.
c. Isoautofluorescent.
d. Variably fluorescent.

6. On fundoscopic exam, choroidal folds are most easily visible using what filter?
a. Unfiltered white light.
b. Cobalt blue filter.
c. Red-free (green) filter.
d. Dim illumination white light.

7. Which of the following choroidal tumors could cause choroidal folds?
a. Choroidal melanoma.
b. Choroidal osteoma.
c. Choroidal hemangioma.
d. All of the above.

8. Why does hypotony tend to cause notable choroidal folds specifically in the macula?
a. The sclera is thickest in the macula.
b. BM is thickest in the macula.
c. The retina is thick parafoveally and thin in the fovea.
d. Vitreous adhesion is strongest at the macula.

9. All of the following conditions are associated with hypotony except which?
a. Pigmentary glaucoma.
b. Cyclodialysis.
c. Open globe injuries.
d. Intraocular volume loss after eye surgery.

10. Scleral inflammation may lead to choroidal folds due to which of the following?
a. Focal or global thickening and later, shrinkage of the sclera.
b. Increased IOP.
c. Decreased choroidal volume.
d. Thinning of the choroid.

11. Choroidal folds are idiopathic in approximately what percentage of cases?
a. 85%.
b. 50%.
c. 25%.
d. 15%.

12. Idiopathic choroidal folds tend to be which of the following?
a. Bilateral.
b. Peripheral.
c. Visually symptomatic.
d. Found in myopes.

13. An increase in orbital tissue volume may be seen in which of the following?
a. Orbital cellulitis.
b. Thyroid orbitopathy.
c. Idiopathic orbital inflammation.
d. All of the above.

14. Retrobulbar masses are not very likely to cause which of the following?
a. Hyperopic shifts.
b. Myopic shifts.
c. Astigmatic shifts.
d. Bilateral refractive shifts.

15. Optic nerve swelling is more likely to cause choroidal folds _____.
a. In the periphery.
b. In the contralateral eye.
c. Adjacent to the optic disc.
d. Only when the edema is due to ischemic optic neuropathy.

16. Intracranial hypertension may cause choroidal folds _____.
a. Only when there is clinically detectable papilledema.
b. Even in the absence of clinically detectable papilledema.
c. Only when papilledema is due to an intracranial mass.
d. Only when papilledema is due to idiopathic intracranial hypertension.

17. Increased CSF pressure may be associated with all of the following except which?
a. Myopic shifts.
b. Posterior globe flattening.
c. Paton's Lines.
d. Hyperopic shifts.

18. What symptoms are typically associated with intracranial hypertension?
a. Nausea, vomiting.
b. Headache.
c. Transient or positional visual obscurations.
d. All of the above.

19. Extraocular hardware may cause choroidal folds _____.
a. Because it may change the scleral shape or tension.
b. Only if the hardware lowers IOP.
c. Only if it pierces the sclera.
d. Due to the hardware increasing IOP.

20. Aside from THIN-RPE, other possible causes of choroidal folds include all of the following except which?
a. Suprachoroidal hemorrhage.
b. Decreased scleral rigidity.
c. Thyroid orbitopathy.
d. Diabetic retinopathy.

Examination Answer Sheet

Choroidal Folds: A New Wrinkle in Retinal Care

Valid for credit through June 15, 2026

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

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Jointly provided by PIM and the Review Education Group.

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. Determine when to prescribe a glaucoma drug for ocular hypertension. ① ② ③ ④ ⑤

22. Recognize choroidal folds via various imaging modalities. ① ② ③ ④ ⑤

23. Identify the underlying causes of choroidal folds. ① ② ③ ④ ⑤

24. Effectively manage patients who present with choroidal folds. ① ② ③ ④ ⑤

25. Based upon your participation in this activity, do you intend to change your practice behavior? (Choose only one of the following options.)

Ⓐ I do plan to implement changes in my practice based on the information presented.

Ⓑ My current practice has been reinforced by the information presented.

Ⓒ I need more information before I will change my practice.

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

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

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

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

Ⓐ Very confident Ⓑ Somewhat confident Ⓒ Unsure Ⓓ Not confident

29. Which of the following do you anticipate will be the primary barrier to implementing these changes?

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

30. Additional comments on this course: _____

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

First Name
Last Name
E-Mail
The following is your: ☐ Home Address ☐ Business Address
Business Name
Address
City State
ZIP
Telephone # - -
Fax # - -
OE Tracker Number

Rate the quality of the material provided:

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

31. The content was evidence-based. ① ② ③ ④ ⑤

32. The content was balanced and free of bias. ① ② ③ ④ ⑤

33. The presentation was clear and effective. ① ② ③ ④ ⑤

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

Signature _____ Date _____ Lesson 123899 RO-OSC-0623

Will Epi-on Take Off?

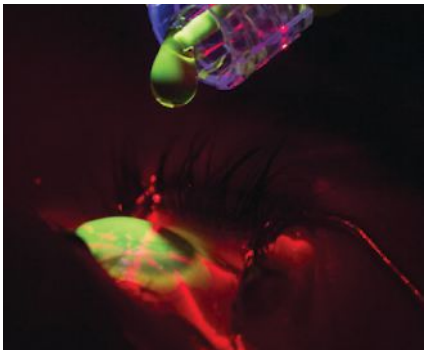
The challenges of crosslinking an intact cornea.

Q What are the differences between epi-on and -off CXL and how are protocols being devised to ensure comparable efficacy with the newer procedure?

A The paradigm of progressive keratoconus management has shifted tremendously with FDA approval of epithelium-off collagen corneal crosslinking (CXL) in 2016. Previously, we managed patients with an arsenal of optical devices for visual rehabilitation. However, nothing could prevent patients from disease progression—leading to a loss of their best-corrected visual acuity, corneal scarring, contact lens intolerance and inevitably a corneal transplant, Drs. Lawrence Nguyen, OD, and Mitch Ibach, OD, of Vance Thompson Vision in Sioux Falls, SD explain. Now, epi-off CXL can halt the progression of corneal ectasia. The epithelium is debrided to allow better diffusion of the riboflavin into the stroma.[1] The riboflavin is then coupled with UV light to stabilize the biomechanically weakened ectatic cornea.

Off's Limits

Dr. Nguyen emphasizes that with epi-off CXL, post-op care is crucial, especially pain management. "Our first line is to ensure the bandage contact lens is in place, then to advise frequent lubrication and oral NSAIDs," she says. Reassuring patients that their decreased acuity is normal is also important, she adds. Unlike in laser refractive surgeries, CXL patients are not expected to have a profound visual recovery; it is simply not the goal of this procedure. However, studies have shown improvement of uncorrected visual acuity up to 2.7 Snellen lines after two years.[2]



Riboflavin loading in CXL.

There is no question that epi-off is efficacious at halting the progression of corneal ectasia. The Avedro Phase III clinical trials demonstrated 1.6D of flattening in Kmax after one year compared to the sham group, which steepened in Kmax.[3] Other studies have shown similar outcomes in Kmax flattening.[2,4] From a safety standpoint, epi-off CXL is minimally invasive with few risks, including temporary corneal haze, punctate keratitis, corneal scarring/infiltrates and infectious keratitis.[3]

On the Radar

Now with epithelium-on CXL on the horizon, the goal is to lessen postoperative pain, shorten procedural duration and significantly reduce the risk of corneal haze and infections, Dr. Ibach notes. "Epi-on CXL emphasizes the patient's quality of life and can allow the patient to return to their normal routine postoperatively at a much faster rate. From a surgical standpoint, this can also shorten the duration of the procedure as well."

The biggest concern remains whether epi-on is as efficacious compared to epi-off CXL, Dr. Ibach warns. Oxygen, riboflavin and UV light are all necessary for CXL. The intact epithelium adds a barrier that poses a threat to efficacy. Despite epi-off providing superior results in most studies, epi-on CXL has still shown great potential to halt progression through multiple platforms.[5,6]

Glaukos's epi-on CXL is currently in an FDA Phase III trial and met the primary endpoint of a 1.00D difference in Kmax between the treatment group and the control group at six months.[7] To circumvent the epithelium, there are many variations to epi-on CXL to increase its effectiveness. Some strategies include pulsed UV light treatments, riboflavin loading sponges and specialized goggles to increase oxygen levels. Others are aimed at increasing riboflavin penetration through various formulations, including the addition of sodium iodine or norepinephrine.[8,9]

"Regardless, CXL has become a valuable and life-changing procedure for patients. It will be exciting to see epi-on available in the US, as each technique will still have its situational advantages in progressive keratoconus patients," Dr. Ibach posits. ■

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About Dr. Shovlin

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

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BY JESSICA STEEN, OD

No Pressure, Really

Here's how I found myself experiencing hypotony—and why this rare complication of intravitreal injection should be on our radar.

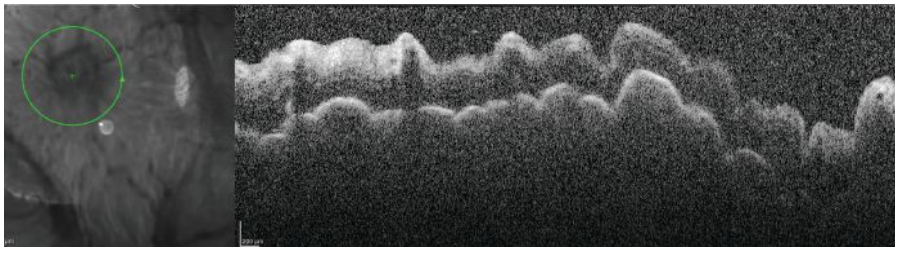
Managing complex ocular cases through the application of clinical knowledge, high-quality data and experience can maximize patient outcomes—but when we ourselves become the patient, we can pass along the perspective gained and nuanced features learned to our colleagues and form the basis for delivering true person-centered care.

As a retina patient myself, I've received periodic intravitreal bevacizumab injections followed by anterior chamber paracentesis for a number of years for the treatment of chronic low-grade cystoid macular edema following a complex retinal detachment repair (a tale for another time). As an experienced intravitreal anti-VEGF injection patient, I know what to expect post-injection: ocular surface irritation and tearing and burning for 15 to 45 minutes, followed by visual improvement and relief in ocular comfort throughout the day.

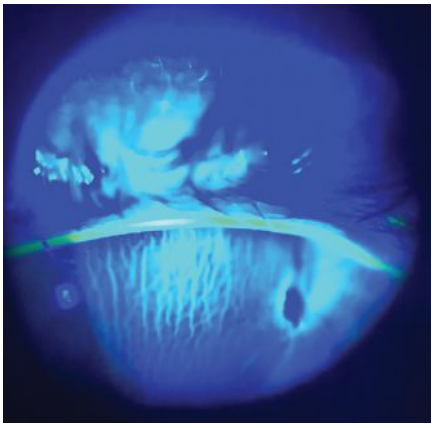
So, the post-procedure experience of significant unilateral tearing and the feeling of a very "full" tear film, in the absence of redness or irritation after the resolution of surface-related symptoms, was something new—and concerning. By the sixth hour following the injection and paracentesis, now back in the retina surgeon's office, my intraocular pressure (IOP) was 2mm Hg, and my vision had dropped from 20/40 to 20/400 with a positive Seidel sign—not from the scleral site where the intravitreal injection was performed but from the peripheral corneal paracentesis site, with the presence of chorioretinal folds in the posterior pole.

Background

Treating retinal pathology with intravitreal injections has transformed long-term outcomes for patients with sight-threatening disease associated with exudation and neovascularization. While adverse events associated with intravitreal injections are most often centered on vision-threatening complications such as rare inflammatory and infectious events, the most common adverse effect, transient elevated IOP, and its potential long-term consequences on optic nerve health, are often left out of the discussion.



Peripapillary OCT demonstrating chorioretinal folds in the setting of hypotony.



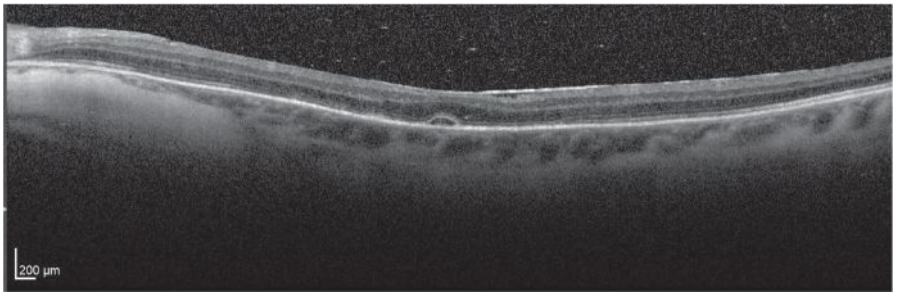
Positive Seidel test and anterior corneal folds.

Transient elevated intraocular pressure is an expected phenomenon following intravitreal injection and is reported to typically range from 28mm Hg to 55mm Hg but has been reported to be as high as 87mm Hg. The proposed mechanism for elevated IOP is increased volume of fluid within the vitreous cavity, so higher-volume intravitreal therapies, including the newly approved Syfovre (pegcetacoplan, Apellis Pharmaceuticals) 0.15mg/0.1mL, may be expected to have a greater impact on post-procedure IOP in comparison with most intravitreal anti-VEGF agents, which have a volume of 0.05mL.[1-5]

While the long-term impact of this immediate, sudden IOP spike on retinal vascular circulation and optic nerve function, especially in individuals with a high burden of treatment for chronic retinal pathology, has yet to be completely understood, the expected impact on the risk of development or progression of glaucomatous optic neuropathy has led treating surgeons to explore additional options to minimize or prevent the IOP rise.[1,6-10.]

About Dr. Steen

Dr. Steen is an assistant professor at Nova Southeastern University College of Optometry where she serves as director of the Glaucoma Service, coordinator of the Primary Care with Emphasis in Ocular Disease Residency and teaches courses in glaucoma and ocular pharmacology. Her financial disclosures include Bausch & Lomb, Santen, Ocuphire and Carl Zeiss Meditec.



Resolution of chorioretinal folds with remaining focal serous detachment at day five.

Anterior chamber paracentesis has been cited as an effective means of mitigating the IOP spike either immediately prior to or following intravitreal injection.[1,7-9] In a series of 1,661 patients who received anterior chamber paracentesis following intravitreal injection of either bevacizumab or triamcinolone acetonide, the mean post-injection IOP was 9mm Hg with a median 210µL of aqueous removed.[7] Importantly, no instances of endophthalmitis, wound leak or negative outcome were observed in any of the cases.[7]

The risk of hypotony following anterior chamber paracentesis appears to be very rare, with one case series of five eyes of five patients described in the literature.[9] Hypotony risk has been suggested to be associated with serial paracentesis due to long-term degradation of corneal integrity with repeat trauma, which may make cases more complex to manage in comparison with persistent wound leak following cataract surgery due to "coring" of corneal tissue and resulting poor wound closure.[9]

Previously reported cases of hypotony following anterior chamber paracentesis associated with intravitreal injections resolved with a range of treatments of escalating invasiveness, which range from bandage contact lens placement, pressure patching and corneal sealant use to corneal suturing.[9]

Swift Solutions

After a persistent wound leak from the paracentesis site was determined, topical moxifloxacin for prophylaxis of infection was instilled in-office, and a tight pressure patch was placed over my left eye for two days. One disposable eye patch was folded in half and placed under a second patch adhered with three strips of very tightly stretched surgical tape.

> **Early recognition and aggressive treatment led to a quick recovery and excellent visual outcome.**

For pressure patch placement, paper-based tape with limited stretch is not preferred, as it makes it difficult to apply the patch tightly. Following removal of the pressure patch, difluprednate 0.05% was instilled every three to four hours, moxifloxacin was instilled 0.5% TID and cyclopentolate 1% was instilled once daily.

A tight pressure patch was placed each night and removed in the morning with the goal of keeping the eye closed with limited motility under the patch.

My vision subjectively improved rapidly after the first 48 hours and was 20/60 with a fully formed anterior chamber, closed wound leak and IOP of 28mm Hg after five days.

At five days, the chorioretinal folds had resolved, but a subtle, focal serous detachment remained. The topical steroid and antibiotic were discontinued, and intraocular pressure, visual acuity and macular anatomy all returned to baseline by 14 days.

Takeaways

Early recognition and aggressive treatment, with a combination of medical therapy and a tightly applied pressure patch, led to a quick recovery and excellent visual outcome.

Everybody has "something." When that something overlaps with the conditions that we diagnose and manage in our own patients, that personal experience and intimate understanding of management considerations—as well as the ability to share that experience with colleagues—ultimately elevates the care that we continue to provide to our patients. ■

I remain so grateful to Timothy Murray, MD, Aaron Gold, OD, and the team at Murray Ocular Oncology and Retina for their ongoing exceptional care and constant pursuit to improve treatment outcomes.

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BY JAMES L. FANELLI, OD

Glaucoma, But Not

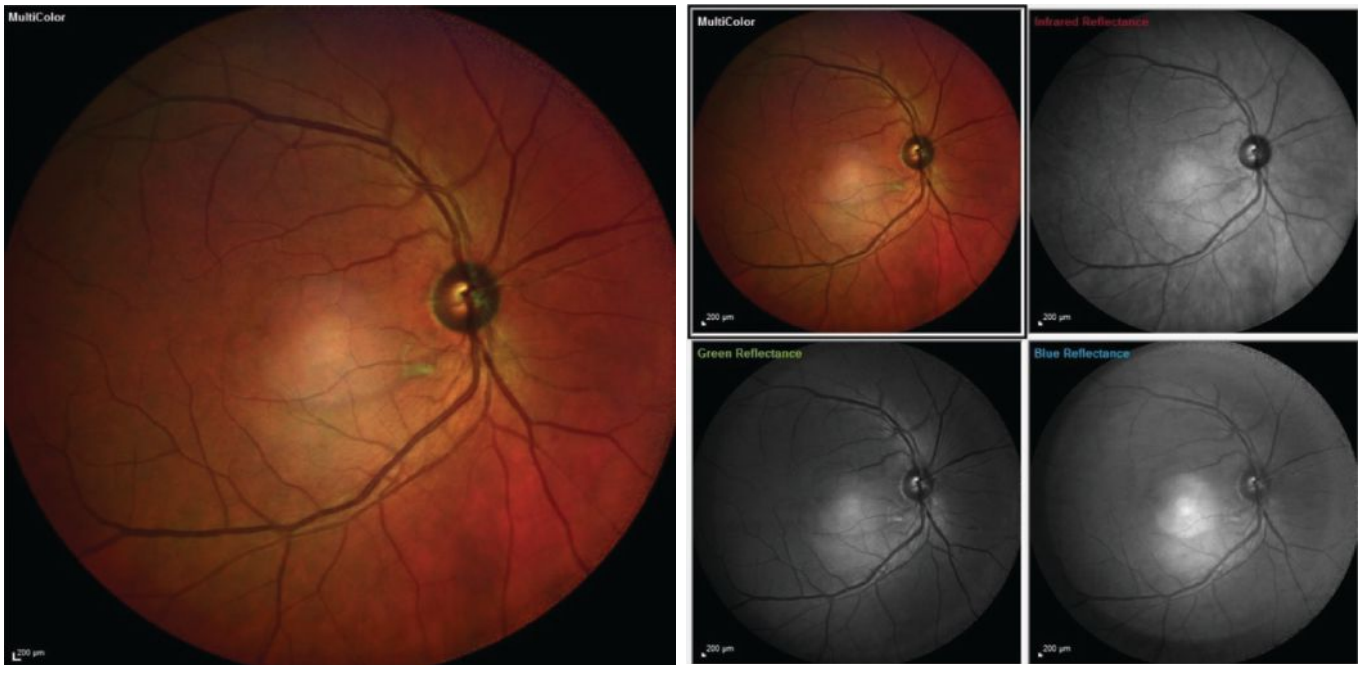
Sometimes the damage is already done, but there's still a job to finish.

A 63-year-old Caucasian female whom I've been seeing for several years was recently referred for cataract surgery, as her cataracts had gradually progressed over the past several years and were beginning to affect her quality of life.

I first met this patient in 2018 when she was referred to me by her primary care provider with complaints of transient visual obscurations. At that time, she also complained of right-sided eye discomfort and transient visual obscurations OU lasting for a few minutes several times each day. The onset of symptoms had begun two weeks earlier, and when they did not subside, she sought the care of her primary care provider who referred her to me.

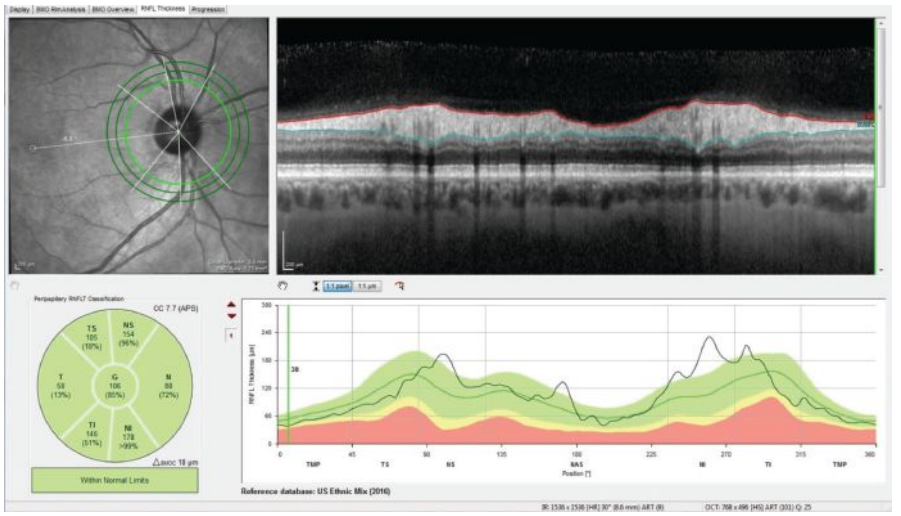
Ultimately, the patient presented with neovascularization of the iris in the right eye and visual acuities of 20/25 OD and 20/20 OS. Medication included crestor, synthroid and albuterol PRN. Pupils were ERRLA with no afferent pupillary defect noted. The anterior segment was entirely normal otherwise OU, with clear crystalline lenses. Prior to dilation, threshold visual fields were performed and showed generalized depression OU; there were no neurological field defects respecting the horizontal or vertical meridians. Applanation tensions were 18mm Hg OD and 17mm Hg OS. Cup-to-disc ratios were 0.50 x 0.60 OD and 0.50 x 0.55 OS. There was no neovascularization of the disc nor elsewhere in either eye. There was one isolated cotton wool spot (microinfarct) along the inferior arcade in the right eye, and vasculature OU was characterized by mild arteriolarsclerosis. The peripheral retinal examination was unremarkable. Baseline multimodal fundus images were obtained.

Auscultation for carotid bruits was positive on both the right and left sides. Given the clinical findings, the patient was scheduled for a carotid Doppler evaluation followed by an MRI and MRA. The studies demonstrated >90% occlusion of the right internal carotid artery and >75% of the left. MRI findings demonstrated



The patient's initial visit in 2018 showed only one isolated cotton wool spot along the inferior arcade, which is consistent with the iris neovascularization, ocular ischemia and complaints of visual disturbances.

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



Baseline RNFL 3.5mm circle scans were obtained following the carotid artery stenting and failed PCA stenting. This image was taken three months after initial presentation.

several areas of focal atrophy consistent with small vessel occlusive disease, and the MRA demonstrated stenosis of various branches of the cerebral vasculature, in particular the left posterior cerebral artery (PCA).

The patient was scheduled for a right carotid stenting procedure, which was completed without complication. During the carotid stenting, a cerebral angiogram was performed, which verified the stenosis of the left PCA. The patient was subsequently scheduled for stenting of the left PCA. She returned for this procedure; however, intraoperatively it was found that this vessel was not amenable to stenting, so the procedure was aborted.

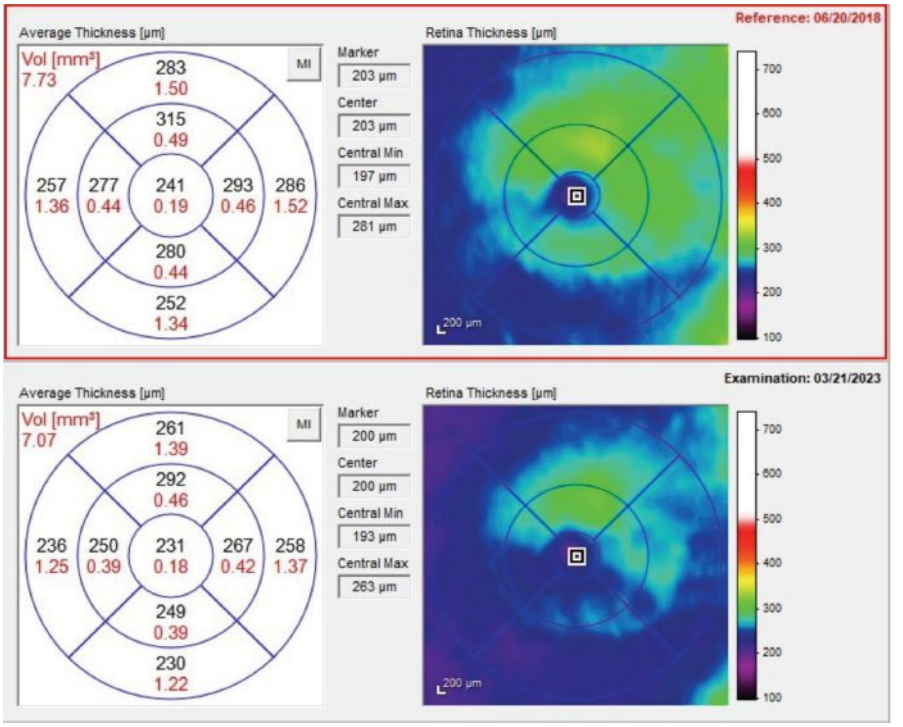
Approximately three months following the carotid stenting, the patient's neovascularization had cleared, as did the microinfarct seen funduscopically in the right eye on initial presentation. Also, she was free of symptoms related to visual obscurations. Baseline OCT scans were obtained.

Following two years of close surveillance of her ophthalmic and visual status and with no complications, the patient was scheduled to be seen yearly and was compliant with the visits. No other neurological symptoms developed in this timeframe, though she did begin to develop cataracts, and as time progressed, the cataracts began to affect her quality of life.

As is always the case in our office, a pre-cataract surgical evaluation was completed prior to referral to the cataract surgeon. This included a detailed and dilated fundus examination, OCT scans of the optic nerves and maculae, tonometry, A-scan ultrasonography and IOL calculations. There were no noted contraindications to cataract surgery, and a preliminary appointment was made with the cataract surgeon. Medications at this point in early March included the previous listed medications along with dabigatran, olmesartan and carvedilol.

Following the evaluation of the patient by the cataract surgeon, she was referred back to me for evaluation of glaucoma OD prior to scheduling lens extraction with IOL placement. Of note was the cataract surgeon's concern that there was RNFL thinning on the right eye consistent with glaucomatous damage.

The patient returned to my office for evaluation. Intraocular pressures were found to be 18mm Hg OD and 19mm Hg OS. Her cup-to-disc ratios were unchanged from previous visits, and glaucoma OCT scans were obtained. There were no changes from scans of the past four years. Gonioscopy showed open angles with no neovascularization and minimal trabecular pigment OU. Threshold



Retinal thickness changes seen from the baseline scan in 2018 to the most recent scan in 2023. Note the significant thinning of the retina in the inferior temporal arcuate region.

field studies were essentially unremarkable.

While there were no recent changes in her OCT scans, there were changes in her right eye that were attributable to the ocular ischemia she presented with in 2018. As the scans showed, there were changes in the macular thickness scans in the right eye that are sometimes suggestive of glaucoma.

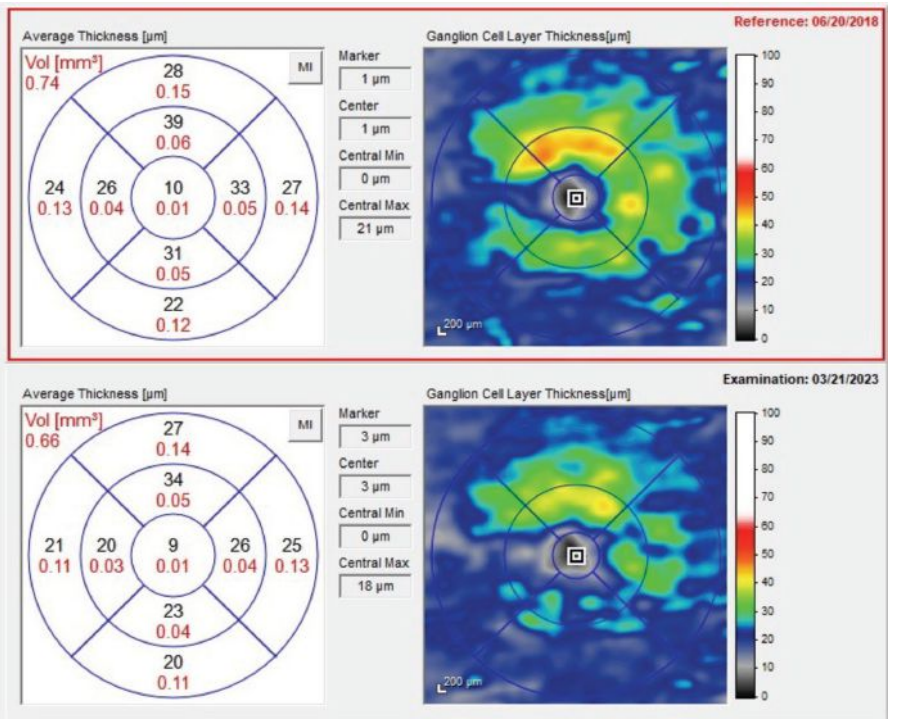
Keep in mind that when viewing the macular scans, the cataract surgeon was only seeing the current scans, which do in fact show thinning of the inferior temporal arcuate region; this finding is what raised the alarm with the cataract surgeon that glaucoma may exist. Closer examination of her earlier scans also shows some early changes seen in the same scan in 2018. Remember too, that this 2018 scan was taken about three months following the carotid stenting on the same side, and the OCT from 2018 is consistent with early ischemic damage to the retina consistent with the ocular ischemia that was ongoing at that time.

> **Glaucomatous damage can certainly result in loss of the RNFL and GCL in the macula, but it also manifests in loss of neuroretinal rim tissue at the disc.**

The GCL shows similar changes. Note the relatively robust GCL thickness in the 2018 scan, relatively early in this disease process.

The neuroretinal rim in the right eye at the current time is stable, with no glaucomatous tissue loss. There is some subtle pallor in the temporal aspect of the rim, owing to the ischemic tissue loss secondary to the ocular ischemic syndrome that initiated sometime prior to our first visit in 2018.

And herein lies the lesson of this case: glaucomatous damage can certainly result in loss of the RNFL and GCL in the macula, but it also manifests in loss of neuroretinal rim tissue at the disc. Various OCTs measure the neuroretinal rim tissue in different ways, but looking closely at the minimum rim width adjacent to Bruch's membrane opening is very helpful in noting erosion of neuroretinal rim tissue. This patient had none, indicating that these changes were associated with neuropathy that is not glaucomatous in origin.



The baseline and current GCL thickness maps. Note the significant loss of thickness seen in the 2023 scan.

There are other conditions that can cause loss of the ganglion cell and RNFL layers without neuroretinal rim loss, and they include the nonglaucomatous optic neuropathies, retinal artery occlusions and other vascular ischemic events, to name a few. In these cases, the loss of the RNFL and GCL occurs quickly, usually within six months of the event. Neuroretinal rim tissue is not lost, but it often becomes pallid.

Outcome

While a quick look at a macular scan preoperatively raises the spectre of glaucoma, the back history of this patient tells us the whole story: what we are looking at today is damage that occurred five years ago due to perfusion issues, which have remained stable and since been rectified.

What kind of visual recovery should this patient expect following cataract surgery? Well, her best-corrected visual acuity two years ago (prior to significant cataract progression) was 20/25 OD and 20/20 OS. Snellen acuity post-lens extraction (barring any complications) will probably be good, perhaps 20/25 OU, but I would suspect that the patient will always see a difference between the eyes, with the left being subjectively better than the right. This was discussed with the patient again before she was referred back to the cataract surgeon for the lens extractions.

The damage has been done; now let's get the patient seeing to her best ability while still surveilling for progressive atherosclerosis of the visual system. ■

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BY JEROME SHERMAN, OD, AND SHERRY BASS, OD

OCD Pays Off

Few patients keep a health diary, but such recordings may benefit your surviving family members and loved ones.

A 45-year-old presented for new glasses because of reduced vision at distance and near in both eyes. He had no other symptoms or contributory history. Best-corrected visual acuity (BCVA) was 20/25- in each eye at distance and near with only a minimal change in prescription. The external exam including confrontation visual fields (VFs) was recorded as normal. Slit lamp revealed mild cataracts in each eye. IOPs were normal, as was the fundus exam in each eye.

The ophthalmic clinician advised the patient that all was well except for the mild cataracts and that cataract surgery may be indicated at a future date. As instructed, the patient returned in two years for a re-evaluation. The record indicated that the patient had the same symptom of reduced vision in both eyes as two years earlier. BCVA was again noted as 20/25- in each eye, and the external exam was unremarkable. Slit lamp exam revealed mild cataracts in both eyes as noted two years earlier.

Bear in mind that this patient, with obsessive compulsive disorder (OCD), kept a detailed health diary with notes about each visit with all health clinicians. He included his symptoms, tests performed, results of both the tests and the exam as explained by the doctor, differential diagnoses, medicine prescribed, possible side effects of the medicine and dates of future visits.

On his second visit with this eye clinician, the patient recorded and dated in his diary blurred vision, worse in the left eye than the right eye and worse in the left field. His chief complaint was far more detailed in his health diary than the chief complaint recorded by the examining doctor.

As recommended by this eye doctor, the patient presented another two years later. He complained that his vision was a bit worse than the previous visit but he denied any other symptoms. Best-corrected VA was 20/25- in each eye, and the external exam was again recorded as within normal limits. Slit lamp exam revealed mild cataracts once again in each eye but noted as slightly worse than before. IOPs were normal and unchanged, and the fundus exam noted the same 0.3 cup-to-disc ratio in both eyes as previously recorded. Refraction revealed a very mild change in the prescription, but the patient decided to get a new frame and lenses. He was reassured that there was no serious problem and again told that the cataracts will eventually need to be removed at some distant date.

As recommended, the fourth visit occurred two years later. The patient was previously seen in 2002, 2004, 2006 and now in 2008. He never missed an appointment!

Surgical Input

During the eighth year following his initial complaint, the patient felt his vision was worsening. He eventually decided to obtain a consultation from a highly respected cataract surgeon. Part of the initial consultation was a screening VF, which indicated a bi-supero-temporal field defect, somewhat worse in the left eye.

The cataract surgeon then performed a routine dilated exam and informed

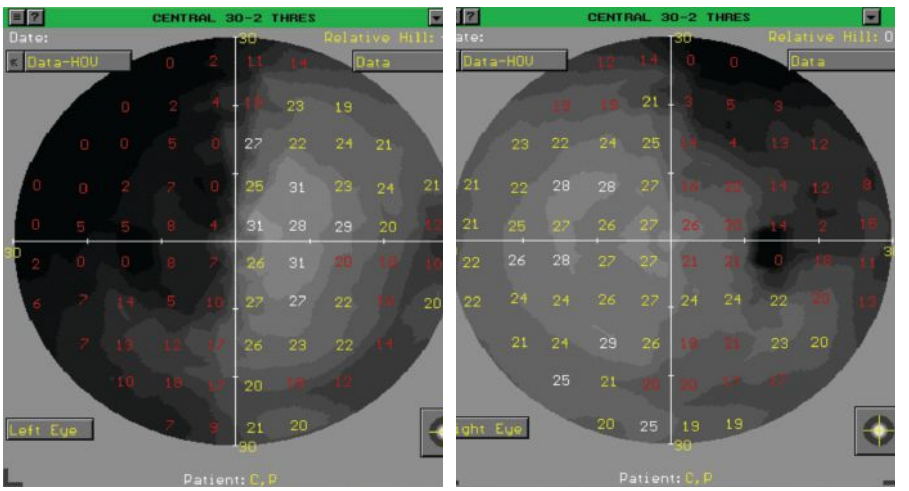

Fig. 1. Fields from a different patient than the case described who had vague complaints. Note the temporal loss in the left eye is worse than the temporal loss in the right eye.

About Drs. Sherman and Bass

Dr. Sherman is a Distinguished Teaching Professor at the SUNY State College of Optometry and editor-in-chief of *Retina Revealed* at www.retinarevealed.com. During his 52 years at SUNY, Dr. Sherman has published about 750 various manuscripts. He has also served as an expert witness in 400 malpractice cases, approximately equally split between plaintiff and defendant. Dr. Sherman has received support for *Retina Revealed* from Carl Zeiss Meditec, MacuHealth and Konan. **Dr. Bass** is a Distinguished Teaching Professor at the SUNY College of Optometry and is an attending in the Retina Clinic of the University Eye Center. She has served as an expert witness in a significant number of malpractice cases, the majority in support of the defendant. She serves as a consultant for ProQR Therapeutics.

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the patient that his cataracts were only mild and certainly did not require surgical removal. The surgeon then told the patient that the VF screening test strongly suggested a problem in the head. Threshold VFs were then performed and confirmed the screening field results, which were highly suggestive of a pituitary tumor and that an MRI should be obtained. Within several days, the MRI was obtained and the neuro-radiologist confirmed a large pituitary adenoma.

The patient was immediately referred to a prominent neurosurgeon at a major medical center. This specialist discussed two very different surgical approaches. The first was much less invasive, required an incision above the upper teeth or through the nose and then through the sphenoidal sinus and did not require major brain surgery. The second procedure, described to the patient as "cracking the skull" was more invasive, had a better chance of success but included greater risk.

The patient decided to have the transsphenoidal adenomectomy surgery. Scanning the patient a day after surgery revealed that much of the tumor remained. The patient obtained several neurosurgical consultations, and all agreed that the more invasive, and more dangerous, procedure was now indicated. One month after the "successful" removal of the remaining mass, the patient died in the hospital, never regaining consciousness after the second procedure. Cause of death: complications of the second surgery.

You Be the Judge

Should the eye clinician have considered a diagnosis beyond mild cataracts?

Should an automated VF screening be obtained on any of the four visits?

Do confrontation VFs give the doctor and patient a false sense of security?

Did the eye clinician take and record a detailed history of chief complaints?

Is the eye clinician culpable of malpractice?

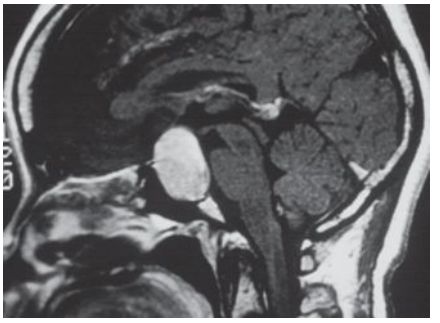

Fig. 2. Mid-sagittal MRI from a different patient that demonstrates a pituitary adenoma. This current patient underwent successful surgery, and the fields returned to normal.

Would you judge the doctor culpable of malpractice if the patient did not keep a detailed health diary?

Follow-up

Family members knew the patient had a health diary and reviewed it several weeks after the funeral. They then contacted a malpractice attorney who accepted the case. An optometrist (JS) was asked to review all the documents, the all-important diary and furnish an opinion.

The entry by the patient relating to the second visit stated, blurred vision, worse in the left eye than the right eye and worse in the left field, which appeared to have been totally disregarded by the eye clinician. Since the patient recorded this chief complaint in his health diary, we assume this was also reported to the doctor. Since the fundus exam was noted to be normal, blurred vision in the left eye worse in the left field strongly suggests a temporal field loss in the left eye. With a normal retinal exam, etiologies beyond the globe must be considered.

Our Opinion

Pituitary adenomas are common tumors, and if they grow large enough below the chiasm, the inferior nasal fibers at the optic chiasm will be impaired and typically result in a superior-temporal field loss, usually bilateral, but not always symmetric (*Figures 1 and 2*). Many patients with early to moderate bitemporal field loss are unaware of the VF defect, most likely due to the fact that the temporal field loss of the right eye is filled in by the normal nasal field of the left eye and the temporal field loss of the left eye is compensated by the normal nasal field of the right eye. With both eyes open, the field is essentially normal, and many patients do not cover one eye and then the other.

The records of far too many malpractice allegation cases of failure to detect brain lesions contain "confrontation VFs recorded as normal." In several of these cases, it is unclear whether confrontation VFs, although recorded as normal, were ever performed. We believe that normal confrontation VFs can give the doctor and patient a false sense of security.

Many patients in their mid-forties have mild cataracts but most are asymptomatic. Persistent symptoms require a complete work-up, and this includes automated VFs. One could argue that this patient could still be alive if automated visual fields were performed and interpreted properly.

Without the health diary documenting the blurred vision, worse in the left eye than the right eye and worse in the left field, the malpractice allegation would be far more tenuous.

Prior to a jury trial, the case was settled for an undisclosed amount, but in the realm of a million dollars. A jury trial could have resulted in a much larger award. ■

NOTE: This article is one of a series based on actual lawsuits in which the author served as an expert witness or rendered an expert opinion. These cases are factual, but some details have been altered to preserve confidentiality. The article represents the authors' opinion of acceptable standards of care and do not give legal or medical advice. Laws, standards and the outcome of cases can vary from place to place. Others' opinions may differ; we welcome yours.

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Hitting the Bullseye

The finding of this pattern hinted to this patient's condition.

BY RAMI ABOUMOURAD, OD, AND KALIE LEONE, OD
MIAMI

A 62-year-old Hispanic female presented with gradual progressive central vision loss of both eyes over five years. Her past medical history included rheumatoid arthritis that was controlled with tofacitinib and hypertension that was controlled with lisinopril. She reported drug allergies to codeine and thiopental.

Her best-corrected visual acuity was 20/50 OD and 20/60 OS. Pupils were equally round and reactive to light with no relative afferent pupillary defect, confrontation visual fields were full to finger counting and extraocular motilities were full. IOP was 19mm Hg OD and 17mm Hg OS by applanation. The patient had total color vision deficit by Ishihara color plate testing. Anterior segment examination revealed trace nuclear sclerosis OU. While fundus photos were not obtained, clinical exam revealed a pigmentary bullseye maculopathy OU and macular chorioretinal scar OS. Fundus autofluorescence and OCT imaging are below for review.

Take the Retina Quiz

1. Which of the following best describes the OCT retinal imaging of both eyes?
a. There is hyperreflectivity of the inner retina.
b. There is perifoveal loss of the inner segment/outer segment junction (IS/OS) and retinal pigment epithelium (RPE).
c. There is subfoveal loss of the IS/OS and RPE.
d. There is subretinal fibrosis.

2. Which of the following is NOT a differential diagnosis for bullseye maculopathy?
a. Cone dystrophy.
b. Hydroxychloroquine toxicity.
c. Presumed ocular histoplasmosis syndrome.
d. Stargardt's disease.

3. What is the appropriate treatment for this patient's retinal condition?
a. Broad spectrum oral antibiotics.
b. Cessation of inciting agent and/or observation.
c. Genetic testing.
d. Oral corticosteroids.

4. What is the expected visual prognosis?
a. Complete loss of peripheral vision.
b. Complete resolution to baseline.
c. Progression to no light perception.
d. Stability or slowly progressive decline in central visual acuity.

5. Which of the following is least helpful in detecting early hydroxychloroquine retinal toxicity?
a. Dilated fundus examination.
b. Fundus autofluorescence.
c. Multifocal electroretinogram.
d. Spectral-domain (SD-OCT) of the macula.

For answers to the quiz, see page 98.

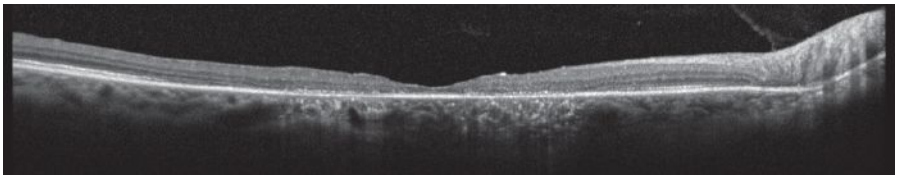

Fig. 1. Heidelberg SD-OCT of the right macula.

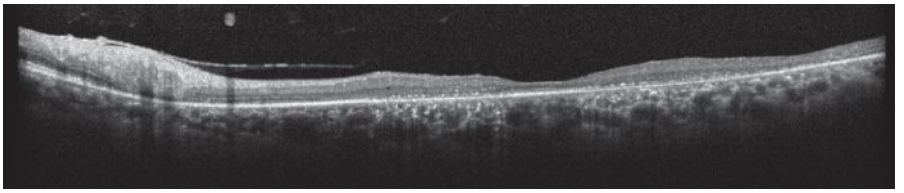

Fig. 2. Heidelberg SD-OCT of the left macula.

Diagnosis

Dilated fundus examination revealed pigmentary changes of the macula in a bullseye pattern in both eyes. Additionally, there was a hyperpigmented chorioretinal scar present in the superonasal macula of the left eye secondary to a reported history of toxoplasma retinochoroiditis 20 years prior. OCT confirmed perifoveal IS/OS and RPE loss, evidenced by perifoveal hypertransmission, with a thin sliver of remaining photoreceptors and RPE underlying the fovea (*Figures 1 & 2*). Fundus autofluorescence demonstrated perifoveal hypoautofluorescence in both eyes and a dense hypoautofluorescent lesion in the superonasal macula consistent with the chorioretinal scar observed on fundus exam (*Figures 3 & 4*).

Further questioning revealed that the patient was previously on hydroxychloroquine for 18 years for

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

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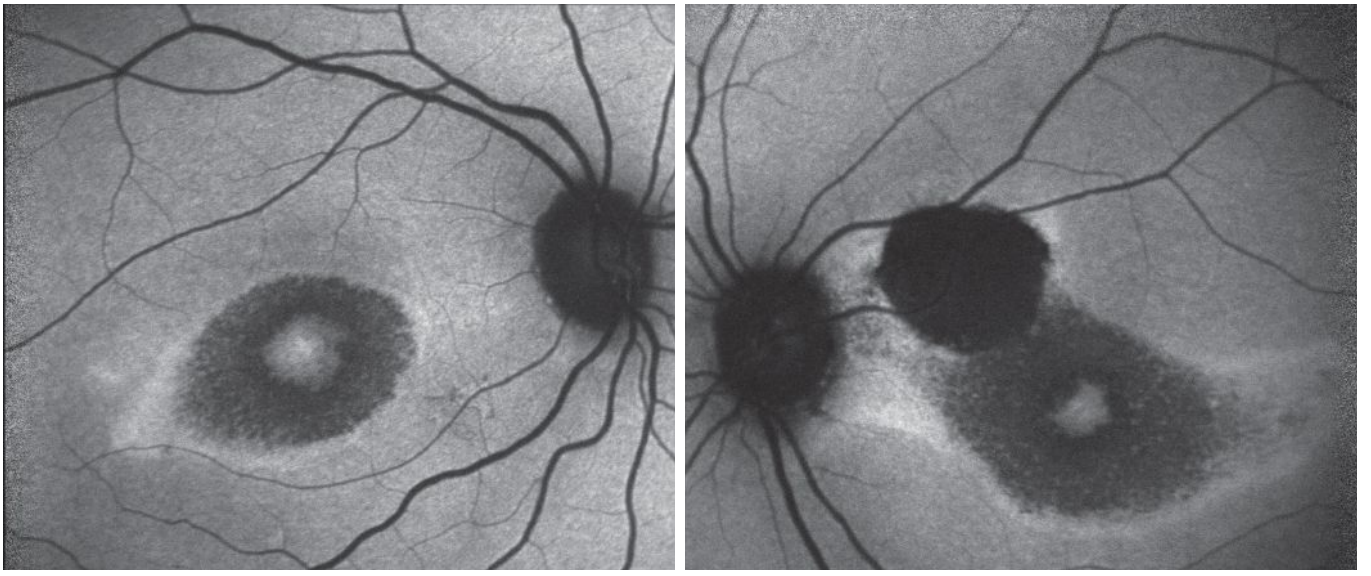

Figs. 3 and 4. Heidelberg fundus autofluorescence of the right (left image) and left (right image) eyes.

rheumatoid arthritis. Retinal toxicity was noted six years prior to her presentation to our institute and the hydroxychloroquine was discontinued at that time.

Discussion

Hydroxychloroquine is a disease-modifying anti-rheumatic drug commonly used for the management of autoimmune diseases such as rheumatoid arthritis and systemic lupus erythematosus.[1,2] Retinal toxicity is thought to occur by accumulating within the RPE, which interferes with photoreceptor outer-segment phagocytosis and produces destruction of the perifoveal outer retina (rods, cones, RPE).[3] Furthermore, *in vitro* studies have shown that the medication alters the pH of RPE lysozymes, thereby causing higher levels of lipofuscin which is associated with photoreceptor damage.[2]

The risk of developing retinopathy is most strongly related to daily dose and duration of use.[4] The recommended daily dose for prevention of retinal toxicity is less than 5mg/kg of real weight. At this dose, the risk of retinopathy is less than 1% during the first five years of use, less than 2% for 10 years of use and increases to 20% after 20 years of use.[4] Other factors that increase risk of ocular toxicity include cumulative lifetime dose greater than 1,000g, renal disease, concomitant tamoxifen use and underlying macular disease.[2,4]

Screening and Detection

The hallmark clinical presentation of advanced hydroxychloroquine retinal toxicity is bilateral pigmentary changes encircling the fovea, termed a bullseye maculopathy.[3] This is an advanced finding, and the goal of screening is to detect retinopathy before it presents clinically on fundus examination.[4] A bullseye maculopathy without a history of high-risk medication use should prompt other differential diagnoses such as cone-rod or cone dystrophy, Stargardt's disease, fenestrated sheen macular dystrophy, neuronal ceroid lipofuscinosis or an atypical presentation of macular degeneration.[3]

The American Academy of Ophthalmology updated their screening guidelines in 2016 to include a baseline fundus examination at the time of medication initiation to rule out underlying macular disease, then annual screenings begin after five years.[4] Presence of risk factors, including daily dose in excess of the recommendations, may warrant sooner or more frequent screenings.[4] Annual screenings should include 10-2 visual field testing for detection of early paracentral depressions and SD-OCT of the macula to look for perifoveal IS/OS.[4] The flying saucer sign is an OCT finding characterized by an intact subfoveal IS/OS layer with perifoveal IS/OS loss, creating an appearance of a subfoveal "flying saucer."[5] It is important to note that patients of Asian descent may present with extramacular toxicity, so 30-2 or 24-2 visual fields should be used in addition to 10-2 field testing.[4,6]

Additional testing can include fundus autofluorescence (bullseye hyperautofluorescence in the acute stage and hypoautofluorescence in the late atrophic stage) and multifocal electroretinography (looking for weak parafoveal responses).[4] Previous screening tests such as color vision and Amsler grid testing have fallen out of favor with the advent of more advanced ancillary tests that are more sensitive and specific to detect earlier disease.[4]

Crucial Communication

Once there is any evidence of retinal toxicity, communication should be initiated with the patient's rheumatology team informing them of the findings. Ultimately, the decision to continue or stop the drug should be made by the patient and their rheumatologist; how-

ever, retinopathy can still progress, even with prompt cessation of the medication due to the slow clearance of the drug and entrapment within the RPE.[7] The earlier the retinal toxicity is detected and medication is stopped, the less likely the damage is to progress.[1] In patients with minimal RPE atrophy, the retinopathy is likely to stabilize within one year of cessation, but in patients with more advanced RPE damage, progression can continue for as long as 20 years after cessation.[7]

Optometrists play a key role in screening for retinal toxicity in patients taking hydroxychloroquine and should be well-prepared to detect early changes in order to prevent patients from becoming symptomatic.

Since retinal toxicity is irreversible even after drug cessation, this patient was counseled on the state of their disease and simply observed as there are no further indicated interventions. Communication with the patient's rheumatology team would have been indicated had she still been on hydroxychloroquine at the time of presentation. This patient's vision is expected to remain stable or to slowly decline, as there was already extensive perifoveal RPE damage.[1] ■

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ABOUT THE AUTHOR

Drs. Aboumourad and Leone currently practice at Bascom Palmer Eye Institute in Miami. They have no financial disclosures.

The Cornea in Crisis

Learn to identify and manage cases of edema following cataract surgery.

BY MICHAL REYGAN MARTIN, OD
AUSTIN, TX

Following cataract surgery, patients will present for their one-day post-op visit. The exam will primarily consist of a visual acuity check, tonometry measurement and slit lamp evaluation. One finding you may come across is a hazy appearance to the cornea. What is it, and how do you act on it?

Corneal Edema

Patients will already be using their postoperative antibiotic and anti-inflammatory drops, and in most cases, this is enough. In less than a week's time, the cornea will clear up, visual acuity will improve and no additional drops are needed. This is due to corneal edema, commonly caused by increased inflammation and intraoperative decreased endothelial cell function.[1]

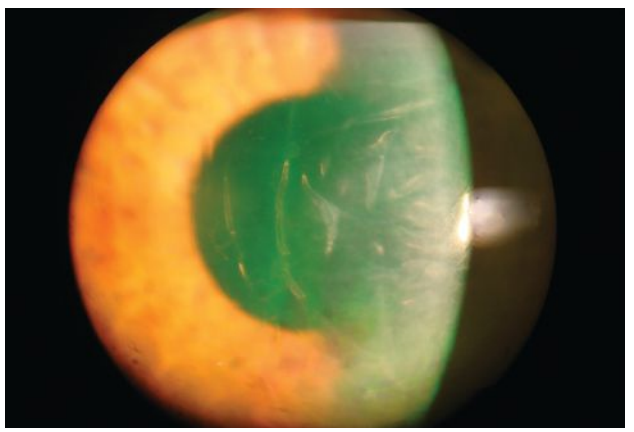


Photo: Derek Cunningham, OD

Corneal swelling and endothelial folds at a one-day cataract surgery post-op.

In cases where epithelial microcysts are present, this is due to excessive corneal edema and can be caused by elevated intraocular pressure (IOP). Diligently measure the pressure, and if the patient is in pain, the secondary port can be burped to mechanically remove aqueous, lowering the IOP in-office.[2] Be aware that the IOP reading will be inaccurate and typically underestimated due to the softened edematous cornea.[3] In some cases, the patient may be okay to use pressure-lowering drops and monitored closely. If elevated pressures persist, treat the underlying cause (*e.g.*, retained lens fragment, trabeculitis).

The cornea's transmissibility is possible due to the unique structural arrangement of each corneal layer. At the endothelial level, barrier and pump functions promote deturgescence and maintain tissue thickness.[5] Endothelial tight junctions' barrier function is heavily dependent on calcium and adenosine. Irrigation solutions and drugs used intraoperatively can cause insult to the pump's ability to function and limit calcium ion availability, both of which contribute to corneal edema.[2]

Intraoperative risk factors include procedural trauma from instrument use, irrigation techniques/materials and surgery duration. Inflammation and the generation of free radicals have been found to cause endothelial cell dysfunction, which has led to the use of irrigating solutions such as viscoelastic and balance salt solution. These contain sodium hyaluronate which has less impact on the calcium-dependent tight junctions.[5]

Pre-existing risk factors that are likely to increase the severity of corneal edema include low endothelial cell count, corneal endothelial dystrophies, iridocorneal endothelial syndrome and pseudoexfoliation syndrome. This last one which may lead to pseudoexfoliative material adhering to the corneal endothelium, which disrupts its appearance and function.[2]

Edema initially gives the cornea a dull and hazy appearance with the concurrent presence of increased corneal thickness. The hazy presence is not to be mistaken for residual viscoelastic, which will have a more

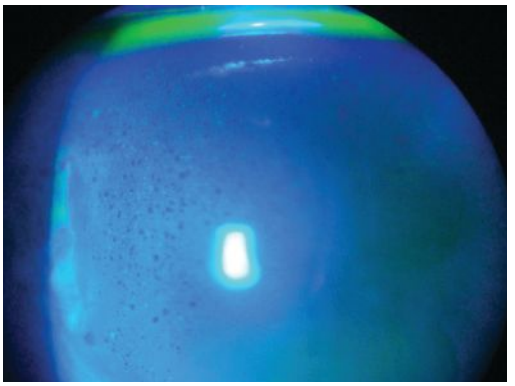
Differential Diagnoses to Rule Out

- **Endophthalmitis**: this is usually present three to five days after surgery; corneal edema associated with lid swelling, hypopyon, vitreous exudates, blunting of red reflex and retinitis
- **Herpetic endotheilitis**: keratic precipitates, reduced corneal sensation and past history of recurrent attacks
- **Endothelial dystrophies**: prominent guttae present in fellow eye
- **Toxic endothelial cell destruction (TECD) syndrome**: significant corneal edema within 24 hours after surgery characterized by star-shaped Descemet's folds and count fingers visual acuity
- **Toxic anterior segment syndrome**: similar to TECD, with less edema, a marked inflammatory response and an occasional hypopyon

About Drs. Cunningham and Whitley

Dr. Cunningham is the director of optometry at Dell Laser Consultants in Austin, TX. He has no financial interests to disclose. **Dr. Whitley** is the director of professional relations and residency program supervisor at Virginia Eye Consultants in Norfolk, VA. He is a consultant for Alcon.

Photo: Derek Cunningham, OD



Microcystic edema from corneal swelling at a one-day cataract post-op.

clustered, less diffuse appearance on the posterior endothelial surface. As the edema persists, the cornea may start to reveal the appearance of microcysts, bullae and scarring.[2]

Management

This includes the use of hypertonic agents such as sodium chloride 5% eye drops to help draw water out of the edematous cornea, but doing so can lead to irritation and may have little impact in reducing stromal edema. Anti-inflammatory drops help speed up the reduction of postoperative inflammation, typically given in the form of a topical steroid.[2]

Surgical management is rarely required. In cases of Descemet's membrane detachment, spontaneous reattachment is commonly seen within days following surgery. In more severe cases, surgical intervention is needed in the form of an air or gas injection. When there is severe irreversible endothelial damage, as a last resort endothelial keratoplasty or penetrating keratoplasty surgeries are performed.[6]

Overall, cataract and refractive lens procedures have evolved tremendously, with all the advancements and even the most proficient surgeons, postoperative corneal edema is still commonly present.[2] Often, it's self-limiting and seldom a long-term issue. A thorough preoperative consult, minimal intraoperative endothelial insult and vigilant postoperative care will assist in limiting poor outcomes and unsatisfied patients. ■

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ABOUT THE AUTHOR

Dr. Martin is an ocular disease resident at Dell Laser Consultants in Austin, TX. He has no financial interests to disclose.

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BY ANDREW S. GURWOOD, OD

DIAGNOSTIC QUIZ

One False Move

Beauty products can have damaging effects on the eyes and/or surrounding tissue.

A 34-year woman presented complaining of a painful upper eyelid OD of two days' duration following application of false eyelashes. She had removed the lashes the day before and explained that her lid remained swollen, "purple" and painful. She denied trauma, systemic disease or allergies of any kind.

Clinical Findings

Her best-corrected entering visual acuities were 20/20 in the right eye and 20/20 in the left at distance and near. Her external examination was remarkable for a painful and swollen right upper eyelid, tender to the touch, which is demonstrated in the photograph below on the left. Her extraocular motilities were full, her confrontation visual fields were intact and there was no evidence of afferent pupillary defect. Her posterior segment findings were normal and Goldmann applanation tonometry measured 17mm Hg OU.

Additional Testing

The patient was further assessed by palpation of the injured area and inspection of the tissue for firmness, intactness (puncture wound), bleeding or suppurative oozing. The lashes were inspected for lingering "glue." The fornix regions were evaluated for foreign material. The cornea was inspected for superficial injury.

Your Diagnosis

What would be your diagnosis in this case based on the findings presented? What's the likely prognosis? Which interventions, if any, would you recommend? To find out, read the online version of this article at www.reviewofoptometry.com. ■

The patient's initial presentation is shown at left and her appearance after resolution at right. What do you see here?

About Dr. Gurwood Dr. Gurwood is a professor of clinical sciences at The Eye Institute of the Pennsylvania College of Optometry at Salus University. He is a co-chief of Primary Care Suite 3. He is attending medical staff in the department of ophthalmology at Albert Einstein Medical Center, Philadelphia. He has no financial interests to disclose.

Retina Quiz Answers (from page 90)—Q1: b, Q2: c, Q3: b, Q4: d, Q5: a

NEXT MONTH IN THE MAG

In July, we present our annual issue devoted to glaucoma. Articles will include:

- Avoid These Common Glaucoma Mistakes
- What to Do When You See Progression
- Understanding Angle Mechanics and Mishaps
- Glaucoma Care Beyond the Basics
- Optic Nerve Disorders: How They Manifest and What They Mean

Also in this issue:

- Unpacking the TFOS Lifestyle Report on Dry Eye

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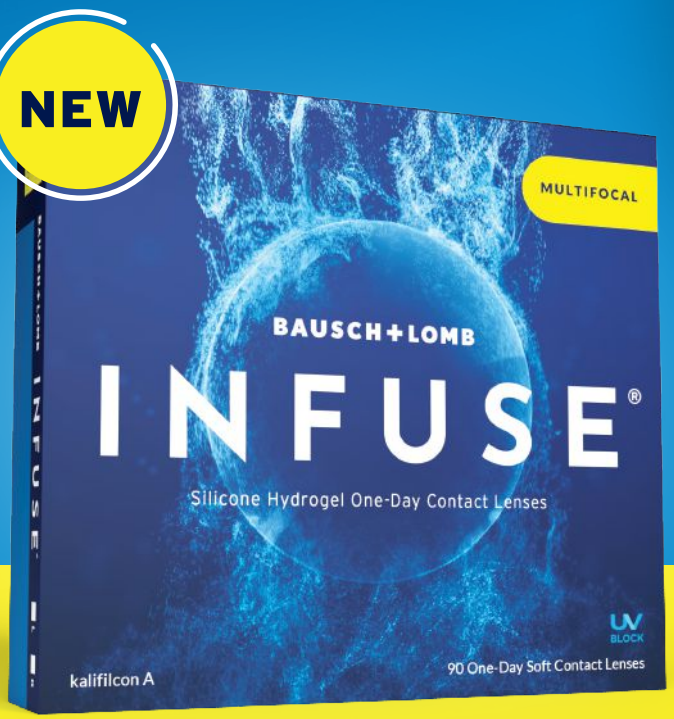

BAUSCH+LOMB

Keep an eye out
for the root cause of blepharitis.

Demodex mites are the cause of chronic inflammation and associated with two-thirds of blepharitis cases.[1,2]
Demodex blepharitis (DB) is an important part of eyelid health.[3,4]

JEANETTE, real DB patient

SEE THE SIGNS OF DB FOR YOURSELF

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