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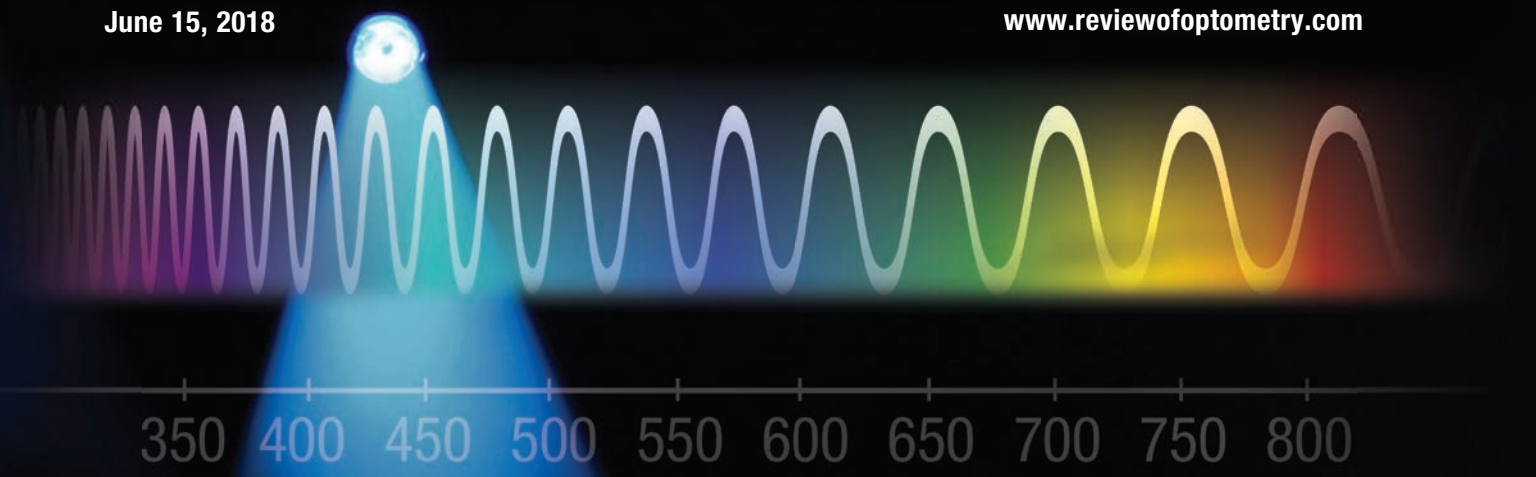

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IN THE NEWS

Using **ultrasound biomicroscopy (UBM)**, investigators were able to **identify positive findings of ocular toxocariasis (OT)** in 92.3% of study participants diagnosed with OT. UBM also revealed 12 of the peripherally involved eyes also had posterior pole granulomas, which the researchers called a 'combined type' of OT. The imaging modality uncovered vitreous strands in 67.9% of study eyes, peripheral tractional retinal detachment in 52.6% and tractional cyclodialysis in 41.0% of eyes.

Chen Q, Gu J, Jiang R, et al. Role of ultrasound biomicroscopy in diagnosis of ocular toxocariasis. Br J Ophthalmol. April 9, 2018. [Epub ahead of print].

Rural areas of low-resource countries bear the biggest burden of vision impairment from presbyopia, according to a new study. The investigators also reported the **global unmet need for presbyopia correction in 2015 was estimated to be 45%**. The investigators estimated there were 1.8 billion people, globally, with presbyopia in 2015, 826 million of whom had near vision impairment because they had no, or inadequate, vision correction.

Fricke TR, Tahhan N, Resnikoff S, et al. Global prevalence of presbyopia and vision impairment from uncorrected presbyopia. Ophthalmology. May 2018. [Epub ahead of print].

Clinicians should be careful to examine patients with keratoconus for eyelid conditions such as blepharitis, new data suggests, as researchers found a much **higher rate of blepharitis in keratoconus patients than in healthy patients: 24% vs. 2.8%**. Beyond that, a higher percentage of keratoconus patients also claimed they were rubbing their eyes more than once a day and experiencing red and tired eyes.

Mostovoy D, Vinker S, Mimouni M, et al. The association of keratoconus with blepharitis. Clin Exp Optom. 2018;101(3):339-44.

Linked: Topical Glaucoma Meds, CME

Mainstay drugs trigger inflammatory mediators that lead to edema.

By Rebecca Hepp, Managing Editor

Clinicians should do their best to avoid topical glaucoma therapies, especially those preserved with benzalkonium chloride (BAK), post-cataract surgery, if they can, new data suggests. Canadian researchers looked at 508 cataract surgery patients and 5,080 controls and found the incidence of post-op pseudophakic cystoid macular edema (CME) was associated with topical therapy with either prostaglandin analogs (PGAs) or beta-blockers.

The pathophysiology of post-op CME entails production of inflammatory mediators such as prostaglandins and cytokines in the aqueous, which then disperse into the vitreous. "These substances in turn cause damage to the blood-retinal barrier and result in the stimulation of the retinal capillaries and the development of edema over time as serum leaks from the vessels and pools in the retinal tissue," the authors wrote. The authors suggest that "contact of BAK with lens epithelial cells and other intraocular cells undergoing wound healing is thought to enhance the synthesis of prostaglandins in the aqueous humor. Subsequent disruption of the blood-aqueous barrier and blood-retinal barrier is thought to be thereby exacerbated, thus enhancing the development of edema."

But the results provided some good news, too. While the risk of CME was similar with bimatoprost and travoprost, the association between CME and the postoperative use of latanoprost was not statistically significant.

"This study reminds us that there is a small but real risk of CME with the use of topical PGAs," says Richard Trevino, OD, director of Residency Programs at the Rosenberg School of Optometry, University of the Incarnate Word. "This risk must be balanced against the IOP control that these agents provide. Special precautions in patients undergoing cataract and other surgical procedures can help to decrease the risk of CME and minimize the potential for vision loss."

At the same time, the "greatest strength of this study is also its greatest weakness," Dr. Trevino adds. "By analyzing almost 20,000 records in an insurance claims database, the researchers have managed to conduct the largest study to date on the association between topical glaucoma medication and pseudophakic CME. However, such databases do not contain clinical information such as how and when the medications were used."

Wendel C, Zakrzewski H, Carleton B, et al. Association of postoperative topical prostaglandin analog or beta-blocker use and incidence of pseudophakic cystoid macular edema. J Glaucoma. 2018;27(5):402-6.

CXL Corneas May Reduce Infection

Researchers recently took a closer look at the antimicrobial and antikeratolytic properties of corneal collagen crosslinking (CXL) in the context of treating fungal keratitis, and the study results are promising.

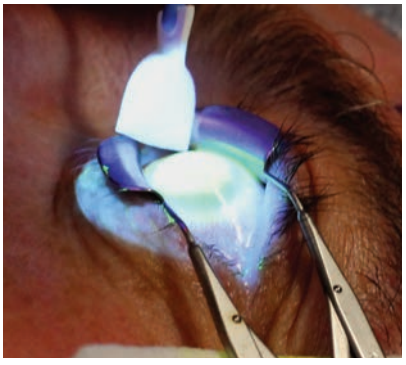

Photo: Marshall Ford, MD, Pacific Cataract and Laser Institute.

Donor corneas that have undergone CXL may provide suprising benefits for fighting off infection.

They enrolled 53 patients with confirmed fungal keratitis and randomized them into two groups: those who underwent therapeutic keratoplasty (TPK) using CXL-treated donor corneas, and those who had TPK with non-CXL treated donor corneas. After analyzing the postoperative incidence of graft infection, graft clarity, visual acuity, deep vascularization of the graft and other complications, they found none of the CXL group developed infection compared with six in the non-CXL group.

They also found 80.8% of patients in the CXL group had clear grafts at six months post-op compared with 22.2% of the non-CXL group. As for corrected visual acuity, 50% of patients in the CXL group achieved 20/200 or better, compared with only 7.4% in the non-CXL group.

"CXL-treated donor corneas may help to reduce the incidence of graft infection after TPK in fungal keratitis, and a beneficial effect is observed in terms of visual acuity and graft clarity," the study authors said. "Further long-term multicenter prospective randomized trials with larger sample sizes and in which optical grade donor tissues are used are required to confirm the results of our study and to elucidate the functional outcomes of TPK with CXL-treated donor corneas."

Titiyal JS, Karunakaran A, Kaur M, et al. Collagen cross-linked therapeutic grafts in fungal keratitis. Ophthalmology. May 5, 2018. [Epub ahead of print].

Visual Gains Lost After AMD Trial

After concluding the ASSESS prospective trial performed at Cole Eye Institute, researchers were encouraged to learn that switching patients with age-related macular degeneration (AMD) from Avastin (bevacizumab, Genentech) or Lucentis (ranibizumab, Genentech) to a fixed bimonthly intravitreal injection of Eylea (aflibercept, Regeneron) for two years provided statistically significant improvement in central subfoveal thickness and vision compared with baseline. However, following the 32 participants for another 12 months as they moved back into routine care provided disheartening results.

"While fixed interval injections may produce sustainable anatomic and visual gains, it is not typical of clinical practice, where treat-and-extend or as-needed clinical protocols based on medical judgment are the norm," says Sara Weidmayer, OD, clinical assistant professor in the Department of Ophthalmology and Visual Sciences at the University of Michigan.

The routine care, mostly consisting of variable treatment regimens, led to a slow decrease in visual gains. At baseline, 22% of participants had visual acuity of 20/200 or worse, which fell to just 5.5% at 24 months of fixed bimonthly treatment. After the next 12 months of follow-up, that percentage jumped back up to 16.7%.

"While this deterioration is not desirable, it also is not always practical to maintain long-term fixed interval injections due to treatment burden for the patient, such as travel, scheduling, cost and overall patient health," Dr. Weidmayer says. Until future controlled studies with larger number of patients and longer follow-up periods can help to develop an effective but less burdensome follow-up protocol, "we must continue to encourage our patients to follow the advisement of their retinal specialists regarding their injection regimen," she adds.

Want to see more? Check out *Review of Optometry*'s daily News Feed at www.reviewofoptometry.com/news or scan the QR code.

Conti FF, Silva FQ, Srivastava SK, et al. 36-month evaluation of intravitreous aflibercept injection for wet age-related macular degeneration in patients previously treated with ranibizumab or bevacizumab. Ophthalmic Surg Lasers Imaging Retina. 2018;49(3):179-85.

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References: **1.** Alcon sales data on file. **2.** Wirtitsch MG, Findl O, Menapace R, et al. Effect of haptic design on change in axial position after cataract surgery. *J Cataract Refract Surg.* 2004;30(1):45-51. **3.** Visser N, Bauer NJ, Nuijts RM. Toric intraocular lenses: Historical overview, patient selection, IOL calculation, surgical techniques, clinical outcomes, and complications. *J Cataract Refract Surg.* 2013;39(4):624-637. **4.** Potvin R, Kramer BA, Hardten DR, Berdahl JP. Toric intraocular lens orientation and residual refractive astigmatism: An analysis. *Clin Ophthalmol.* 2016;10:1829-1836.

News **Review**

MGD Behind Ocular Discomfort

A new study is providing hard data for a claim optometrists have been making for years: meibomian gland dysfunction (MGD) contributes substantially to the burden of ocular discomfort. The research, published in the May edition of the Icelandic journal *Acta Ophthalmologica*, looked at the experiences of 1,372 dry eye patients and found that a whopping 70.3% of them had signs of MGD.[1]

This study retrospectively used medical records from an Austrian clinic to evaluate symptoms and objective tear film and ocular surface parameters. The researchers classified patients into categories of pure MGD, pure aqueous tear deficiency (ATD), MGD combined with ATD, pure anterior blepharitis, Sjögren's syndrome (SS) without MGD and SS together with MGD.

"Although the intensity of subjective complaints was similar to all other subgroups, pure MGD exhibited the lowest severity of signs of ocular surface damage and also affected younger people," the study says.

"The results of this study demonstrate the importance of thorough evaluation of the meibomian glands in all of our patients," says Chandra Mickles, OD, the Dry Eye Service coordinator at Nova Southeastern University. "We are fortunate today to have diagnostic tools from the simple meibomian gland expression to the meibography which are tremendously useful in MGD detection."

Optometrists can identify MGD by looking for notching—particularly in the lower eyelid, which is responsible for the majority of oil in the tear film—that is a sign of gland atrophy. Additionally, an OD can use a wet cotton-tip applicator or one of several devices on the market to aid in the expression of the nasal to central lower eyelid glands, and observe both how many glands express and the quality of the meibum. According to Paul Karpecki, OD, "healthy meibum is clear like olive oil and easily expresses, barely noticeable when rolling off the eyelid. A turbid expression or paste-like or non-expressive glands are indicative of further progression of the disease."[2,3]

"Careful assessment of MGD in the mindset of clinicians should lead to appropriate clinical management and ultimately improve patient outcomes," Dr. Mickles says. ■

1. Rabensteiner D, Aminfar H, Boldin I, et al. The prevalence of meibomian gland dysfunction, tear film and ocular surface parameters in an Austrian dry eye clinic population. Acta Ophthalmologica. 2018. onlinelibrary.wiley.com/doi/abs/10.1111/aos.13732. Accessed May 22, 2018.
2. Karpecki P. Finding MGD in DED. Rev Optom. 2015;152(7):24-8.
3. Hom MM, Silverman MW. Displacement technique and meibomian gland expression. J Am Optom Assoc. 1987 Mar;58(3):223-6.

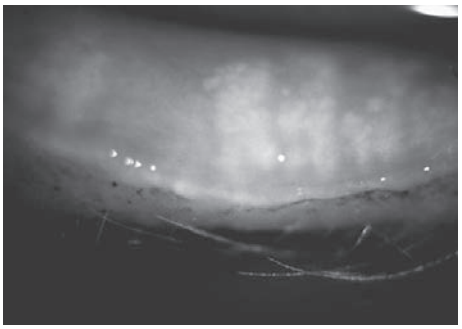

Photo: Gregory Moore, OD

Meibography shows significant meibomian gland structural loss in a patient with moderate to severe MGD.

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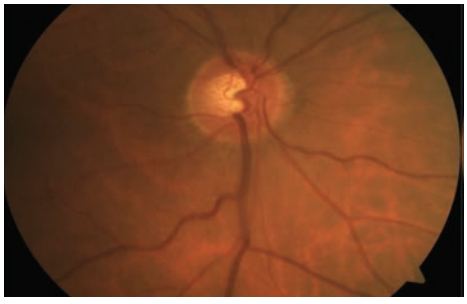

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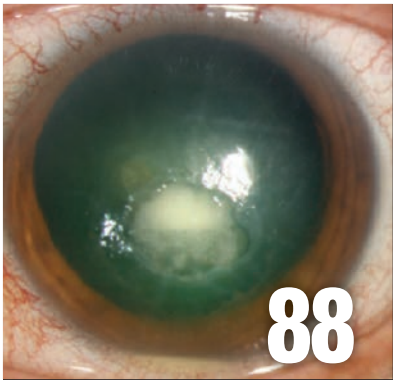

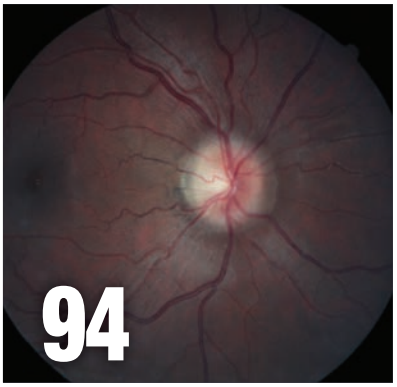

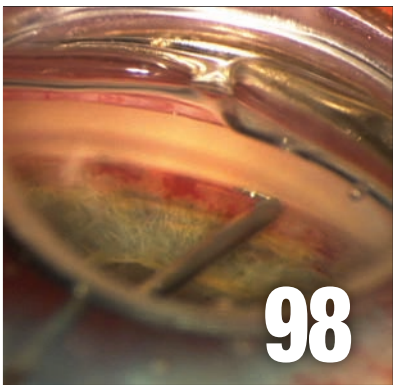

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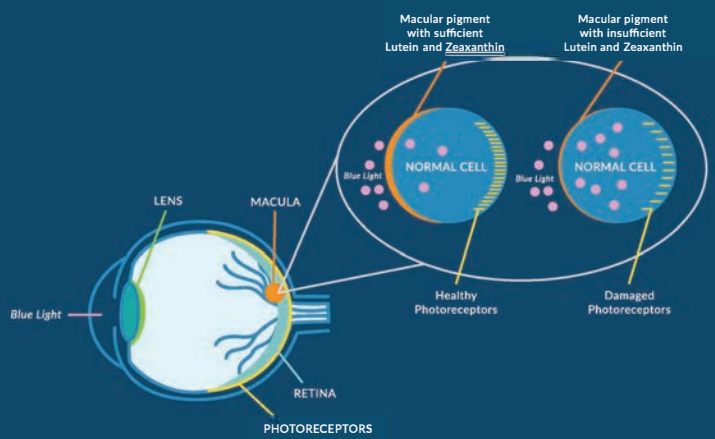
What About All Three Carotenoids?

In 2014, I was fortunate enough to be asked to be a part of a group[1] that reviewed the scientific evidence and nutritional best practices surrounding zeaxanthin, lutein, and meso-zeaxanthin. Drs. Stuart Richer, Steven Ferrucci, Jeffry Gerson, Elizabeth Johnson, Joseph Pizzimenti, Diana Shechtman, and I discussed the role of these carotenoids in eye health and what we believe the best nutritional practices are.

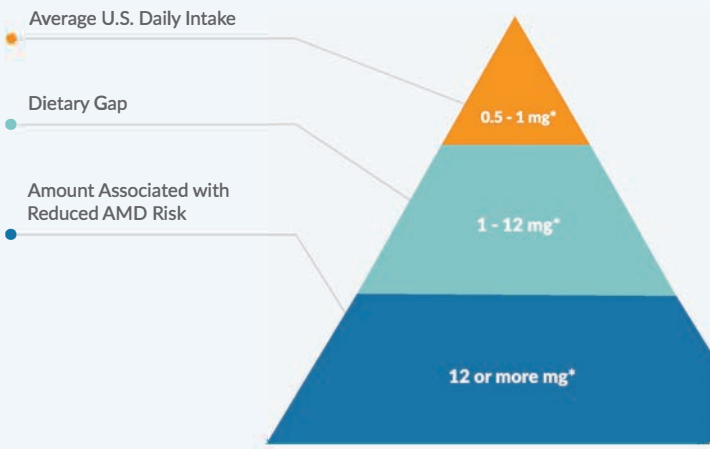
Zeaxanthin & Lutein

First, we discussed the dietary carotenoids, zeaxanthin and lutein, that make up the protective macular pigment. Macular pigment absorbs harmful light waves and stops them from reaching the delicate photoreceptors and causing oxidative stress. Zeaxanthin was first detected in macular pigment in 1985,[2] and these two antioxidants were included in the National Eye Institute's Age-Related Eye Disease (AREDS) 2 study.

Zeaxanthin and Lutein's Effect on Macular Pigment



Zeaxanthin and Lutein Dietary Gap



*According to the National Eye Institute's AREDS 2 study

Lutein can be found in dark, leafy greens like kale and spinach, and zeaxanthin can be found in foods like peppers and corn. While these two nutrients are critical to eye health, the average American diet is sparse when it comes to including them. Dr. Ferrucci said, "...while it is possible to increase lutein and zeaxanthin intake by dietary modification, it is unlikely that most patients will begin eating sufficient quantities of the healthy foods that are rich in these nutrients. Supplementation with lutein and zeaxanthin is important to fill in their dietary gap."

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Meso-Zeaxanthin

We then moved on to the third and more controversial carotenoid, meso-zeaxanthin. Meso-zeaxanthin is a stereoisomer of zeaxanthin and a metabolite of lutein, meaning the body converts lutein to meso-zeaxanthin. While some may think meso-zeaxanthin is a comparable substitute for dietary zeaxanthin, the chemical makeup is not the same.[3]

Despite often-referenced common chirality, meso-zeaxanthin is difficult to find in isolation in nature. While one group of researchers claimed to find it in fish skin and turtle fat,[4] Dr. Johnson and her colleagues tried to replicate their findings using fish skin but had no success. In fact, the amount of clinical evidence supporting meso-zeaxanthin is lacking when compared to the years of research backing dietary zeaxanthin and lutein. Only 21 human clinical studies have been conducted using meso-zeaxanthin, and all but 2 have been performed by the same group of researchers.[5]

Some might argue that the studies reviewing dietary zeaxanthin and lutein also include meso-zeaxanthin, but this is only possible due to impurities during the manufacturing process. Dr. Gerson explained, "...the amount of meso-zeaxanthin in the supplements administered in those studies was never measured and therefore cannot be definitively stated as being present. Furthermore, the presence of meso-zeaxanthin in such variable, negligible amounts does not provide a basis for making any valid statements about its efficacy and safety."

A Battle for Absorption

Beyond the ingredient's effectiveness, there is a concern for safety when talking about meso-zeaxanthin. It's been shown that the three carotenoids compete for absorption, and meso-zeaxanthin can actually reduce systemic levels of dietary lutein and zeaxanthin especially when in high doses.[6,7]

As a strong advocate for the eyes acting as markers for what's happening in the brain, I believe more research needs to be done to know how meso-zeaxanthin affects carotenoid levels in the brain.

Nutritional Guidance in Clinical Practice

Some eye care professionals may feel uneasy prescribing dietary supplements of any kind, but we as practitioners play an important role in our patients' well-being. Getting the proper nutrients is imperative for eye and overall health, and nutritional supplementation can help bridge the dietary gaps that may be present. While multivitamins are a popular suggestion, they often do not contain dietary zeaxanthin and lutein and other key ingredients that support long-term eye health. As Dr. Gerson said in our discussion, the decision for what nutraceutical to prescribe should be based on "what we know from good science" and be manufactured to the highest standards.

Dr. Hitchmoth is Chief of Optometry and former Director of Residency at the VAMC White River Junction. She is a Vision Source private-practice owner and is the first consulting optometrist at University of Massachusetts medical school Practice Transformation Network (SNE-PTN). Dr. Hitchmoth is a nationally-recognized award-winning professor, lecturer and educator. She is a past State President and has served in leadership roles in the American Optometric Association over the past 24 years. She is VA residency trained and a Fellow of the Academy and the Optometric Retina Society. She is also on several pharmaceutical, nutraceutical and ophthalmic device scientific advisory boards. Her publication and research interest centers on retinal biomarkers of systemic disease and disease prevention. Dr. Hitchmoth is recognized as the nation's expert on retinal multi-spectral imaging.

Disclosures: Dr. Hitchmoth serves on the EyePromise Scientific Advisory Board and received honoraria and consulting fees from EyePromise.

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Outlook

By Jack Persico, Editor-in-Chief

Welcome to the Jungle

The contact lens market is growing again! But newbies are beset by conniving retailers. Look out for them.

A recent market report about the vision care industry contained a pleasant surprise: for the first time in what seems like ages, contact lens sales are up. The Vision Council reports that revenue increased 7.5% in 2017 vs. 2015. That took some people (or me, at least) by surprise. For years, the mood surrounding contact lenses has been a weary resignation that the market is stagnant and probably will just stay that way. Although lots of new wearers enter the category—the oft-cited number is 16% new fits annually—an equal number of existing wearers drop out every year, promptly wiping out any gains.

Not any more. "Contact lens sales increased most among all major markets mostly due to an increase in contact lens usage among the US adult population, which saw an increase of 1.1 million new additional users," the report noted. The contact lens population stands at 41.6 million wearers, with a value of $5 billion. Impressively, the category's growth rate is more than double that of the overall vision correction market. In 2017, 192.4 million Americans used some form of correction, representing $40 billion. But the contact lens category outpaced the market as a whole.

Some of this growth came at the expense of an old nemesis—LASIK, down 9.2%—but also from a decline in the number of uncorrected ametropes out there, whose ranks dropped 3.1% as these procrastinators finally did something about their need for vision correction. The rest was from organic growth of the market as the US population increased.

Of course, there are still reasons to be skittish about the contact lens rebound. The Vision Council notes "39.8% of the revenue from online optical sales comes from the sales of contact lenses." That's not 40% of contact lens sales going online, it's 40% of the *total dollar volume of online sales of any kind*. Yikes.

Online contact lens sales grew by 8% in transactions and 10% in revenue last year, the report states, further eroding the market.

And while ODs have battled for years against the misinformation and shady tactics of 1-800-CONTACTS, newer outlets have gotten even more brazen. Witness the tag line for online retailer Simple Contacts: "Lenses you need. Office visits you don't." OK, then! No need for eye health exams, or even prescription checks. Got it.

Such is the atmosphere that greets new lens wearers as they're forming their attitudes and habits; it's also steadily undermining the doctor's relationship with existing wearers.

I'd like to be optimistic and think online sales growth can be reversed. But that's probably naïve. "Contact lenses is the most established online optical market in the US," the Vision Council report soberly reminds us. Better, then, to manage the process and give people the convenience they're looking for without the risks that online retailers downplay or disregard. The AOA is fighting hard on your behalf, and perhaps some shady business dealings will go away. But ultimately it falls to you to look out for patients, both old and new. Your oath is to Hippocrates. Online retailers are inspired only by Croesus. ■

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Sherlock Holmes With a Lab Coat

Put your thinking cap on—it's time to solve some ocular mysteries.

By Paul M. Karpecki, OD, Chief Clinical Editor

Being an optometrist can feel a lot like being a detective. A patient presents with reduced vision—and it could be almost anything. Sleuthing out the underlying cause is often what makes our clinic experience so enjoyable and challenging.

The importance of diagnosis is pervasive this month in our *9th Annual Retina Report* and part two of our new series, *Take Charge of Glaucoma*. Here's a preview.

How to Handle Hemorrhages

All vitreous hemorrhages would require a referral, but the timing is a puzzler that calls for a little detective work to understand what's at stake. Technology can be a huge help. If you can get a B-scan to ensure the retina is intact, for example, you can more safely monitor the condition.

Here are some insights from my colleague John Kitchens, MD:

If the patient has no history of diabetes, the hemorrhage is likely secondary to a posterior vitreous detachment (PVD), unless trauma or another obvious cause is present.

With a history of diabetes, if the fellow eye has mild nonproliferative diabetic retinopathy (NPDR) and the patient has good glucose control, be more concerned about a PVD or even a horseshoe tear, especially with symptoms of floaters or flashes.

If the patient has severe NPDR, it may be the cause of the hemorrhage.

You should refer any patient who does not have diabetes but presents with a hemorrhage in the macula. The chances of a small branch retinal vein occlusion (BRVO) or early choroidal neovascular membrane (CNVM) are too high to monitor.

Hemorrhages outside of the macula are a different story. A diabetes patient with a large preretinal hemorrhage likely has neovascularization and thus early PDR. If multiple hemorrhages exist in one particular quadrant in a patient without diabetes, think extra-macular BRVO.

With a normal OCT, a referral within two to three weeks is reasonable. If hemorrhages are associated with PVD symptoms, refer as if the patient may have a tear. A patient with a one-to-four disc area subretinal hemorrhage at the equator and drusen or peripheral reticular changes likely has an eccentric CNVM. Refer the case, as these can result in breakthrough vitreous hemorrhage.

The most common scenario when you will *not* want to send to retina is a single flame-shaped hemorrhage in a patient without diabetes. Call the primary care provider and ask them to order a complete blood count and check blood pressure. See the patient back in a few weeks. And remember: it can take six to eight weeks for the hemorrhage to resolve.

See the (Blue) Light

This is another tricky clinical case, both in terms of its visual effects and how to educate patients properly. Giles Duffield, a PhD at the University of Notre Dame, has dedicated much of his research to circadian rhythms and the effects of blocking blue light. He notes how blue light from digital devices resets the internal clock, so to speak—a major reason why children are not getting enough high-quality sleep. According to Dr. Duffield, the brain perceives a device's late-day blue light as morning light, creating a jet-lagged effect.

This month's CE article delves into the details to help you understand blue light's impact on AMD patients and anyone using digital devices.

Gear Up for Glaucoma

I was managing glaucoma patients quite well 15 years ago—until I started using OCT. It completely changed our understanding and management of the disease and now allows us to make a diagnosis years earlier, preventing vision loss in many patients. Today, OCT technology continues to advance, adding progression analysis and even swept-source scanning. But other technologies, such as corneal hysteresis, are now changing our understanding of glaucoma, too. Like a good detective, the technology looks where others failed to. By measuring the shock absorption capability of the cornea, hysteresis has been shown to be predictive for visual field loss progression in ways superior to central corneal thickness and even intraocular pressure readings.

Optometry is clearly a field where diagnostic technologies do not stand still. With all of these tools and clinical knowledge at your fingertips, you can be both an optometrist *and* a detective. ■

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B-SCAN PROBE FOR IMAGES OF THE POSTERIOR SEGMENT OF THE EYE

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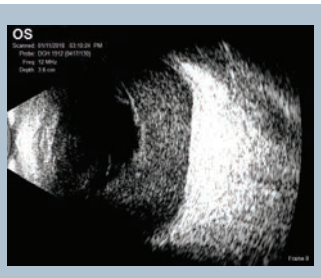
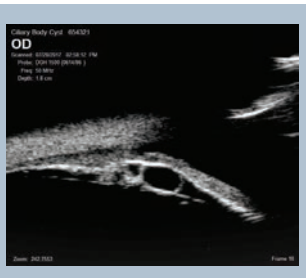
UBM PROBE FOR IMAGES OF THE ANTERIOR SEGMENT OF THE EYE

The UBM probe provides imagery of the anterior segment of the eye, including features behind the iris that optical devices can't see. This is particularly useful in diagnosing plateau iris and other pathologies hidden by the iris.

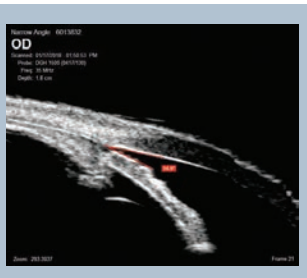
Scanmate Flex A-scan, showing the Flex user interface. The basic interface is the same for all three probe types.



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



Visualizing ciliary cysts with the Scanmate Flex UBM probe.



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Sure I'm Sure, Aren't You?

As docs, we have to at least pretend we know what's going on around here.

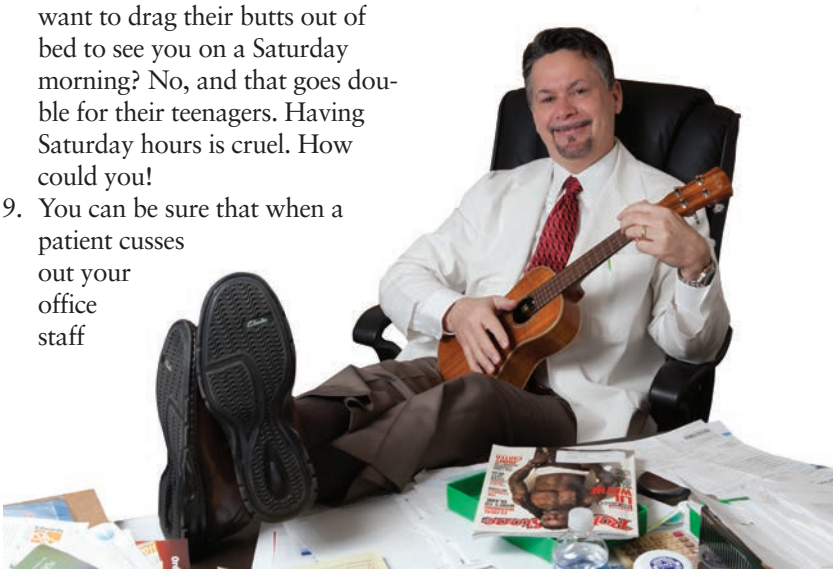
By Montgomery Vickers, OD

Are you one of those annoying doctors who is always sure? Well, me too, and that's why I know the only thing sure docs can be sure of is that we are mostly just pretending.

But, since we are the doctors and it all flows from the top, we ODs have to pretend to be sure so the rest of the office and all of the patients don't burst into flames at any given moment. I want to help you by reminding you of some things that we can be sure about and some things that we can be sure we should not be sure about. Here goes:

1. You can be 100% sure that a patient who shows up 20 minutes late for his appointment is "in a hurry" because he has an appointment with someone else who is much more important than his eyesight—such as his barber. (Remedy: take a pupillary distance, refract him and send him and his hair on their way.)
2. You can be sure that the number of no shows is directly related to the hours of sunshine on any given day. Double book beautiful days, and if they all show up, refer to the remedy above.
3. I am sure staffers think bonuses are actually salary, and when they don't get the bonus they think they took a pay cut. How's that working for you?
4. It's a sure bet that when your contact lens sales rep gripes to you off the record about one of your competitors, that his company gives that jerk a better rate than you. Just be the jerk!
5. We can all be sure that the final patient of the day will opt for dilation rather than retinal mapping. I just throw mapping in or use 2% atropine so they'll never do that again.
6. Are you really, truly sure that you will go out of business if you don't accept that crappy vision plan? You might, but studies show that halitosis is a more common cause of your business failing, so drop the plan and use the empty time to floss.
7. Don't be so sure you will ever get around to reading any journals you have stacked up five feet high beside your desk. I'll save you the trouble… contact lenses are totally different than they were when the journals were published in 1994.
8. Are you sure that patients really want to drag their butts out of bed to see you on a Saturday morning? No, and that goes double for their teenagers. Having Saturday hours is cruel. How could you!
9. You can be sure that when a patient cusses out your office staff because they got billed for that $11 of unmet deductible you deserved. You should have collected $20 extra on the front end and just reimbursed them $9.
10. I am 100% sure that when a patient has their exam in September and comes back the next July to complain they never could wear those glasses that (a) they did not buy them from you, and (b) you will love the look on their face when you ask "have you had a phone at your house over the past 10 months?"

It's a good thing to be sure of yourself, and I applaud you for working toward that goal. Combine that with a well-developed sense that you don't actually have control of any facet of your life and you will seem almost normal to most people around you. ■



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A Coding Crystal Ball

The comprehensive error rate testing program can provide valuable claims data to help you stay ahead of the game. **By John Rumpakis, OD, MBA, Clinical Coding Editor**

Proper claims payments may seem like a simple numbers game, but the Centers for Medicare and Medicaid Services (CMS) fee for service (FFS) program continues to battle improper payments, whether it's a claim that was paid when it should have been denied or a claim that was over- or underpaid. To help track the FFS improper payment rate, CMS developed the comprehensive error rate testing (CERT) program in 1996.[1,2]

Program Basics

To calculate this rate of error, the program requests records from practitioners and analyzes the claims data. Each reporting period, a stratified, random sample of approximately 50,000 claims are selected for review. Because the selection process ensures statistically valid random samples, it provides data indicative of all claims processed by CMS during the reporting period.

For the selected claims, the submitting clinician must provide all supporting documentation, which is then scrutinized by independent medical reviewers to determine if the claim was paid properly under CMS coverage, coding and billing rules. If the documentation does not show all rules were met, the claim is counted as a total or partial improper payment. These errors are then grouped into one of five categories:

1. No documentation
2. Insufficient documentation
3. Medical necessity
4. Incorrect coding
5. Other

If you are one of the random providers chosen to provide documentation for CERT, you will get a letter requesting the proper information; it is critical that you respond in a timely fashion. The request comes in a clearly marked envelope and the specific reporting requirements are spelled out clearly in the official correspondence.[3]

If you do not reply or fail to provide the correct documentation as indicated in the request letter, the CMS contractor will automatically initiate claims adjustments or overpayment recoupment for these undocumented services, so be sure to follow the CERT request guidelines closely.

CERT for You

While this sounds like nothing more than the government doing internal quality control and checking to make sure that all CMS carriers and providers are following the rules, it can have significant implications for your practice. This internal study identifies areas of concern for CMS regarding improper payments made for certain claims types based upon statistical evidence—possibly leading to closer monitoring.

For example, if the program identifies a specific CPT code that has increased in frequency, or a particular ICD-10 code that is being used improperly, it could increase the frequency of audits on the local carrier level. If you were a practitioner who submitted those CPT codes often, this could put you on the short list for an audit.

While CERT may make your heart beat a little faster for fear of more audits, also consider the wealth of information this data provides—for free. CERT data is available to all practitioners on the CMS website and is published on an annual basis.[3]

This is a great crystal ball, of sorts, that allows you to know what CMS considers areas of concern within their payment system. By reviewing this information, you can remain aware of any codes you use regularly in your office that have been flagged by CERT. This is a great way to identify areas that require further attention within your own practice.

The CERT process benefits you both as a taxpayer and as a provider. It is a transparent process that can provide you with useful information that can help identify areas of concern within your practice's coding and billing patterns in a timely fashion and allow you to potentially modify behaviors to be compliant before a larger issue surfaces. ■

Send questions and comments to rocodingconnection@gmail.com.

1. Centers for Medicare and Medicaid Services. Comprehensive error rate testing (CERT). www.cms.gov/Research-Statistics-Data-and-Systems/Monitoring-Programs/Medicare-FFS-Compliance-Programs/CERT. Accessed April 29, 2018.
2. AdvanceMed. About the comprehensive error rate testing (CERT) program. https://certprovider.admedcorp.com/Home/About. Accessed April 29, 2018.
3. AdvanceMed. Sample CERT Letter. https://certprovider.admedcorp.com/Content/SampleLetters/PartB/PartBInitialLetter.pdf. Accessed May 2, 2018.

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If It's Not One Thing It's Another

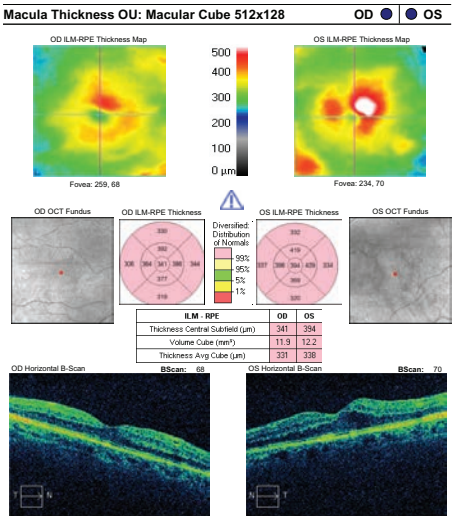
When a cataract patient presents with unexpected DME, the case becomes a balancing act. Here's how to handle the situation. **Edited by Paul C. Ajamian, OD**

Q A cataract patient sent to our practice also had diabetic macular edema (DME). How do I sort these issues out and to whom should I send the patient first, the retina specialist or the cataract surgeon?

A "Of critical importance for any patient being sent for cataract surgery consultation is a thorough exam, and in particular careful retinal evaluation," says Jeffry Gerson, OD, of Grin Eye Care in Olathe, KS. In Dr. Gerson's practice, patients have macular optical coherence tomography (OCT) done as part of the pre-op workup to find any subtle changes that may not have been noticed during the clinical exam. "These 'hidden' findings may become quite significant, and are especially important to document if your patient is considering a premium intraocular lens (IOL)," he adds.

At times, you or the cataract surgeon will want a patient to see a retinal specialist prior to undergoing cataract surgery for "clearance" if there is high myopia, lattice degeneration, diabetic macular edema or any other questionable pathology, Dr. Gerson continues.

"Epiretinal membranes are easily missed. While unlikely to be a reason to cancel cataract surgery, their identification is important in order to temper expectations," says Dr. Gerson. "Macular edema, on the other hand, is a reason to cancel surgery, because it can be worsened by surgery and needs to be controlled before anything else is done." In these cases, send patients to a retina specialist to further assess and treat the macular edema. Once the condition is stable and the patient is cleared by the retina doctor, then you are on safer ground to proceed with cataract extraction, Dr. Gerson says. "Of note, these patients often should be placed on nonsteroidal anti-inflammatory drug (NSAID) drops preoperatively for longer than the average patient, and will likely be on the NSAID longer postoperatively than most patients in order to prevent later onset edema."



This patient was only referred for cataract consultation but also ended up with a diagnosis of macular edema.

Modern Knowledge

Historically, many believed cataract surgery would "speed up" conversion of dry age-related macular degeneration (AMD) to wet AMD, Dr. Gerson says. However, today we know this is not the case. "It is safe for patients with dry macular degeneration to undergo cataract surgery," says Dr. Gerson. Just keep expectations realistic, especially about viable IOL options. For example, it probably would not be prudent to use multifocal IOLs that could affect contrast in a patient who already has reduced contrast sensitivity from AMD, Dr. Gerson adds. "As long as OCT is 'dry,' a retinal consult is not generally necessary, but may make sense in questionable cases."

Since cataract surgery increases the risk of retinal detachment, practitioners should be sure to provide extra education for patients with high myopia, since they are already at a higher risk, says Dr. Gerson. While no preventative treatment applies to these cases, more frequent postoperative follow-up may be required. Additionally, practitioners should educate patients to call immediately if any symptoms are noted.

In one last scenario, a retina surgeon may request that the cataract be removed before they perform a procedure. "Two reasons for this would be better visualization for the retinal surgery, but also to see how much of the visual decline is due to cataract and how much is retina," says Dr. Gerson. "If functional vision returns after cataract surgery, the more invasive procedure may not need to be performed." ■

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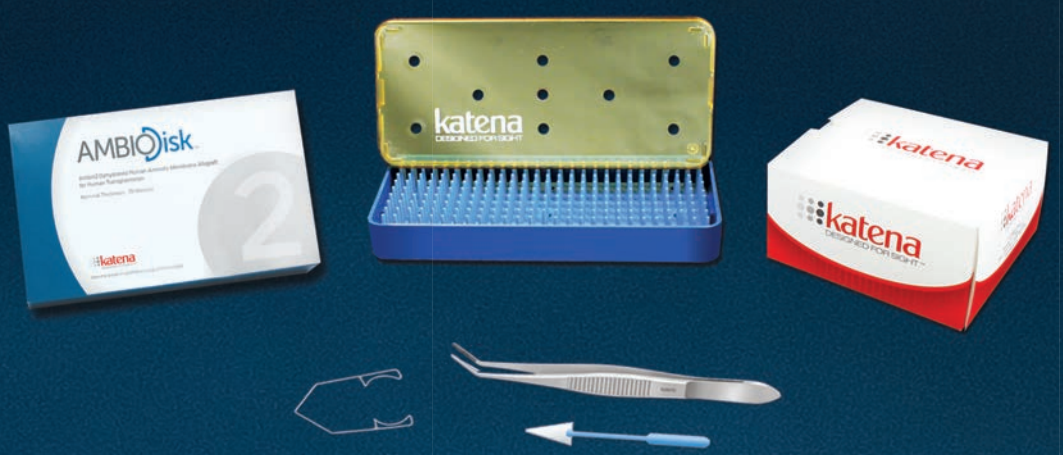

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REFERENCE: Craig JP, et al., TFOS DEWS II Report Executive Summary, The Ocular Surface (2017), http://dx.doi.org/10.1016/j.jtos.2017.08.003

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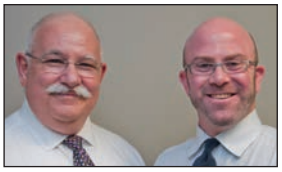
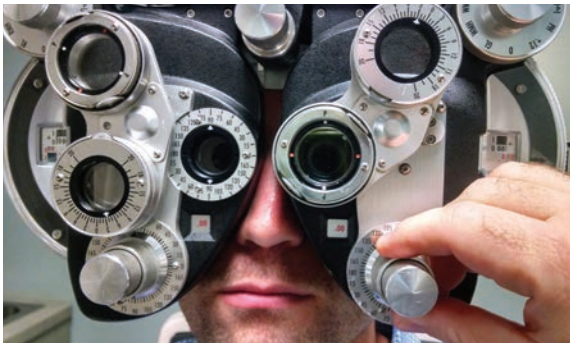


Home on the Range

There's no one ideal refraction. Prescriptions are as diverse as the patients in your chair.

By Marc B. Taub, OD, MS, and Paul Harris, OD

Optometric educators may have noticed a disturbing trend developing. It seems optometry students are under the misconception that a single, ideal refraction—*the* refraction, let's call it—exists for each individual, and that the goal of what we teach students is to find it. Once found, the refraction can be prescribed with impunity. If the patient has trouble with their glasses, it is assumed, the refraction could not be blamed.



Here's a tip for matching a patient to a prescription: talk to them. You're required to do more than simply sit them in front of a phoropter and jot down results.

How many times have we heard, "These are the wrong glasses" or "Who thought this prescription would be right for you?" In fact, refractive procedures should not spit out a single 'correct' number, but rather, a range of lenses. The key is recognizing what to do with that range.

The Skeffington Analytical Sequence

One early method of teaching optometrists, the Skeffington Analytical Sequence, has two key endpoints of refraction—7 and 7a. Today, these endpoints are known (at least at Southern College of Optometry) as the "binocular balance" and the "manifest," though many call the second endpoint the "best visual acuity (BVA)." Their definitions are:

- "The 7," or binocular balance: The most plus/least minus to the first good 20/20.
- "The 7A," or BVA: Change from the first endpoint derived by reducing plus or adding minus binocularly to the set of lenses through which visual acuity is optimized.

Why then, if every optometrist is trained to get these two endpoints, does the myth of *the* refraction persist? Breaking this habit may be a challenge, but take a look at the benefits of taking the time to find the two endpoints and to then discuss options with patients.

Reaching the Endpoints

The binocular balance is the endpoint most are familiar with. After retinoscopy, the optometrist finds the proper level of accommodative stimulation to move to the astigmatism testing. Then, the actual binocular balance is performed. Assume the patient has equal visual acuities through both eyes and no pathology or amblyopia.

A well-performed binocular balance is an OD's best tool to make sure that, when we do prescribe, we know the maximum plus at distance before seeing lens blur, as well as the minimum minus to restore a good 20/20.

In searching for the other endpoint—the BVA— many optometry students change the chart to 20/15 and go one or two clicks binocularly, reducing plus or adding minus, to see if now the patient can see the 20/15 line. Most students have yet to develop a standard system by which they come this other endpoint. That is to be expected, as they are still forming a refraction routine. However, we often embed many of the procedural schemes we learn early, and they may be hard to change later.

Independent Study

Years ago, a group of ODs—Dr. Harris included—participated in a study group that decided to investigate the usefulness of questions they asked patients in finding the second endpoint, BVA. They asked:

- Which is blacker?
- Which is clearer?
- Which is bolder?
- Which is blackest and clearest?
- Which of these looks larger?

During their monthly meetings, the group discussed the reactions they got and how helpful or not the question was, in getting to a second endpoint that was meaningful and stable.

In the end, most patients responded positively to the last question. The 20/20 line was kept in place and the patients were asked to compare two options based on size. Interestingly, this goes against what we all know about optics. How could less plus or more minus, added binocularly, cause the perception of the letters to be larger? Most likely, it was a perceptual response, but we may never know exactly. The key point was, we kept taking plus away or adding minus until the person noticed a slight reduction in size or the size of the letters stopped getting larger. Then, we tested visual acuity through this "largest" (large only in the size perception of the patient of the same 20/20 letters they were looking at) lens and many could now get to 20/10 or even better.

Often, this second endpoint (7a, BVA, manifest) was 0.50D to 0.75D less plus or more minus than the binocular balance. In some instances, the first click caused a shrinkage of the size of the letters and in those instances, only one endpoint was recorded. This was rare.

From a Certain Point of View

"All prescribing is a negotiation between the doctor and the patient," Gregory Kitchener, OD, once said. So, having the range sets the stage for negotiation to begin. Neither binocular balance nor this second endpoint is the prescription. The prescription results from the needs of the patient, both short-term (e.g., to see clearly, to see more efficiently or to reduce asthenopia) and long-term (e.g., to reduce dependency on eyewear for certain tasks) which were expressed by the patient to the doctor, combined with the optometrist's knowledge.

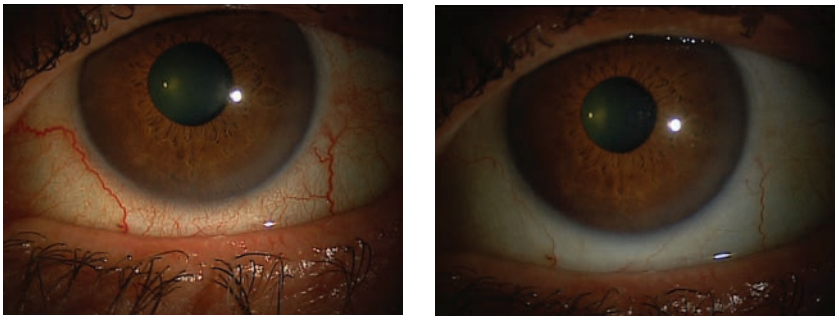
As an example, assume the following range found on two different 14-year-olds. The binocular balance is -1.50D and the BVA is -2.25D. Child #1 plays basketball and is an excellent player hoping to make the varsity team, and together we find that she shoots much more accurately with the full -2.25D. Child #2 is rather bookish and both parents are well above -6.00D in their corrections. Child #2 has been treated before with vision therapy, has been in a bifocal since the third grade and been rather stable in her myopia progression. So, child #1—our basketball player—gets fit with the full -2.25D in contact lenses for full-time wear and might be given some plus for near for studying and test taking. Child #2 gets the -1.50D with an add determined by many factors, with her stress-point retinoscopy being the key factor, giving us a +0.75D add in a bifocal for full-time wear to continue stabilization.

So much of selecting a final set of numbers depends a lot on various frames of references and points of view. One of the keys in working with guidelines or directives is to know when to follow them—and when to let them go. ■

White: Not Just for Brides

The FDA recently approved a new over-the-counter alpha-adrenergic receptor agonist designed to clear your patients' red eyes. **By Paul M. Karpecki, OD**

Many patients have told me their red eyes have kept them from going out with friends or family, and they were tired of being asked if they were crying, drinking, sleep-deprived or on drugs. One patient told me that she had more than 35 interviews for a job and felt she was well qualified, but that her chronic red eyes prevented her from being hired (whether it was affecting her confidence or the interviewer's perception). I had her try a new over-the-counter red eye reducer—one I've been involved with as a consultant for Bausch + Lomb—and she reported being offered a job after her very next interview. Although just one anecdotal experience with a plethora of underlying factors at play, it exemplifies the significant personal and social connotations of chronic red eyes for our patients.



This patient's red eyes cleared up after instilling Lumify, and they can expect the effects to last six to eight hours.

The new red eye reducer I used was Lumify (brimonidine tartrate ophthalmic solution 0.025%, Bausch + Lomb), which could have significant implications for many of your patients.

Something New

Recently FDA-approved for the relief of ocular redness due to minor eye irritations, Lumify is a low-dose (0.025%) brimonidine tartrate and is a highly selected α-2 adrenergic receptor (AR) agonist. It is approved for ages five and older, works within 30 to 60 seconds and lasts six to eight hours with no known rebound hyperemia or tachyphylaxis. As an aside, a non-ocular formulation exists, 0.33% brimonidine gel, for the treatment of persistent facial erythema secondary to rosacea.[1]

We've had almost 20 years of experience with high doses of brimonidine tartrate when managing glaucoma patients. It is the active ingredient in Alphagan P (Allergan), for example. However, the concentration of Alphagan P is 0.1% and generic forms are 0.2%. The higher dose is associated with hyperemia and allergic dermatitis or ocular allergy; very low doses, however, are known to have a whitening or blanching effect.[2-4] Researchers realized reducing the concentration four- or eight-fold avoids IOP changes while enhancing the whitening effect.[5]

Why It's Different

Red eye reducers on the market thus far affect the α-1 receptors alone or α-1 and α-2 receptors non-selectively. Visine (tetrahydrozoline, Johnson & Johnson) is an example of a selective α-1 AR agonist, while mixed α-1 AR and α-2 AR agonists include naphazoline—available as ClearEyes (Prestige Brands), Opcon-A (Bausch + Lomb) and Naphcon-A (Alcon)—and oxymetazoline, marketed as Visine LR (Johnson & Johnson) and Ocu-Clear (Elder Pharmaceuticals).

Long-term use of these redness reducers is limited due to rebound hyperemia, tachyphylaxis, pupil dilation, corneal toxicity and systemic side effects such as dizziness.[6,7] Research shows ocular toxicity due to extended use can be so severe as to mimic ocular cicatricial pemphigoid.[8] The rebound hyperemia and tachyphylaxis is due to the preponderance of α-1 ARs in arteries.[8] Their presence results in constriction of arterial blood flow and oxygen delivery. The body's response is to initiate down-regulation of α-1 ARs, resulting in tachyphylaxis. The vasoconstrictor-induced tissue becomes ischemic and the body's response is vasodilation—hence the rebound redness.[9]

In contrast, Lumify is a selective α-2 AR agonist, with minimal action at the α-1 AR site. This, provides pref-

erential venule constriction rather than affecting oxygen flow through the arteries, eliminating the resultant ischemia.[10] Constriction, which affects the veins—a significant anatomical target, given the concentration of vortex veins and capillary bed volume—results in an increased whitening effect without the risks of tachyphylaxis or rebound hyperemia.[10] Furthermore, since the drug lasts six to eight hours, it can typically be dosed once or twice a day, further decreasing the risk of toxicity and medicamentosa.[11] In FDA Phase II and III trials, neither tachyphylaxis or rebound hyperemia were noted.[12]

The ocular adverse events, such as burning on instillation, were minimal and mild and resolved spontaneously. Patients reported the drop as 'very comfortable,' and researchers detected no systemic safety signals in either the efficacy studies and or the safety study of pediatric, adult and geriatric subjects.[11,12]

Clinical Outlook

So far, I have found this product helpful for patients with conjunctival redness due to ocular irritation such as dry eyes, allergies, rosacea, meibomian gland dysfunction and especially for chronic hyperemia patients.

In this era of information-on-demand for patients, we must always be aware of new therapeutics such as low-dose brimonidine. Patients will see this drop on the shelf next to their current eye whitener, and may come to you seeking advice and information about its effects.

We now have a tool at our disposable to provide these patients whiter eyes without the complications and systemic risks of other vasoconstrictors. This provides yet another opportunity to increase your patients' confidence in your management of their ocular condition and, even more important, increase their own confidence in social circumstances. ■

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Reasons to Reach for Silicone Hydrogel 1-Days

Expert views on the use of silicone hydrogel for 1-day wearers

Silicone hydrogel has become the first choice material in most practices, with ECPs fitting 90% of their monthly and two-week contact lens wearers in this material[1]. This statistic aligns closely with practitioners' concerns about hydrogel materials. For example, 83% worry that frequent replacement hydrogel lens wearers' corneas aren't getting all the oxygen they require. But doctors aren't only concerned about material choice for their frequent replacement wearers. Research shows that 77% of ECPs worry that their hydrogel 1-day lens wearers' corneas aren't getting all the oxygen they require.

Research shows that even daily disposable wearers often don't follow safe wearing instructions and have lifestyles that present significant challenges to safe, comfortable lens wear.* When you consider modern lifestyle challenges, including digital device use, as well as how many hours 1-day patients typically wear their lenses, it becomes quite obvious that replacement schedule shouldn't dictate material choice.

We spoke with three optometrists who advocate the use of silicone hydrogel for 1-day wearers. In the interviews that follow, they explain why they are so committed to the material and how it has improved their relationship with patients as well as their practices.

The Practitioners

Melanie Frogozo OD, FAAO, FSLS
The Contact Lens Institute of San Antonio – San Antonio, Texas

Ethan E. Huisman OD, FAAO
Elite Eye Care
West Des Moines, Iowa

Steve Rosinski OD
Crozet Eye Care
Charlottesville, Virginia

From a material standpoint, why do you reach for silicone hydrogel?

Dr. Huisman: In all patient populations, and especially in younger eyes, long-term eye health is paramount. Silicone hydrogel delivers meaningful ocular health thanks to outstanding oxygen permeability. In fact, 92% of ECPs say silicone hydrogel 1-day lenses are the best choice to safeguard their patients' eye health related to contact lens wear. That's a significant majority. Beyond oxygen benefits, the material also allows for easy insertion and removal as well as all-day comfort. About 80% of ECPs agree that silicone hydrogel 1-day lenses are more comfortable than hydrogel 1-day lenses. Finally, because silicone hydrogel is so comfortable and doesn't dry out, patients aren't rushing home from work to remove their lenses.

Dr. Frogozo: Almost 90% of ECPs agree that if they were to create the perfect lens or if they could only fit one material it would be silicone hydrogel. Obviously, most of us know that silicone hydrogel is a healthier choice than hydrogel material, as evidenced by the changes we've made in material selection for frequent replacement wearers. Our 1-day wearers are no different—there's no reason to deprive them of silicone hydrogel. This is one of the reasons why we have only one hydrogel set in our whole practice and our 1-day fits are entirely in silicone hydrogel material. We strongly believe in the benefits this material provides.

Dr. Rosinski: When any new lens comes out, I like to do my research to see what the studies have shown. I also solicit feedback from my colleagues. But, even if the reviews are glowing, it has to make my patients happy to earn space on my shelf. Silicone hydrogel, in general, and the clariti® 1 day family and MyDay®, in particular, have made my patients happy from a comfort perspective and from a handling perspective, and both have likewise proven to be outstanding from a clinical perspective.

What patient populations are particularly well suited to silicone hydrogel lenses?

Dr. Rosinski: Most, if not all, types of patients are good candidates for silicone hydrogel 1-day lenses. We now have a very broad range of options in this category, including a wide range of sphere, multifocal and toric parameters. I'm not sure why you wouldn't start with a silicone hydrogel 1-day lens as long as you were able to. Explain the benefits to the patients and let them try it.

Dr. Huisman: Almost every patient who can be fit in a soft lens is well suited to silicone hydrogel. There is no logical reason to limit silicone hydrogel fittings to frequent replacement wearers. Most 1-day wearers use their lenses full-time for as many hours as their frequent replacement lens-wearing counterparts. Clearly, the demands are every bit as great and require a lens that offers a healthier lens wearing experience.

84% of the ECPs agreed

that if they could, they would like to prescribe all **new contact lens wearers** with silicone hydrogel 1-day lenses.

About 80% of ECPs agree

that silicone hydrogel 1-day lenses are more comfortable than hydrogel 1-day lenses.

87% of ECPs say

that if they want to keep patients from dropping out of lens wear, they would refit them with 1-day silicone hydrogel lenses.

92% of ECPs say

silicone hydrogel 1-day lenses are the best choice to safeguard their patients' eye health related to contact lens wear.

Dr. Frogozo: There's a misconception that 1-day wearers don't need silicone hydrogel when, in fact, this is the perfect population for the material. These patients have already demonstrated that they value eye health and wellness. Furthermore, a lot of 1-day hydrogel wearers have end-of-day dryness and redness that interferes with daily life. I put all of my new sphere fits in silicone hydrogel and I refit patients who are currently using hydrogel 1-day lenses into 1-day silicone hydrogel.

Why should you switch to a silicone hydrogel material if your patient seems happy with their current 1-day lenses?

Dr. Rosinski: Patients come to me to get the most innovative product they can find. Silicone hydrogel 1-day lenses are exactly that. I sometimes hear colleagues say these lenses cost too much, but cost is relative. We should never make assumptions about what a patient can or can't afford. Offer the best every time. Even if a patient turns it down, you've made it clear that you know what's best and you make it available to your patients. This goes a long way toward building a good reputation. And at the very least, it plants the seed for future visits.

Dr. Huisman: Switching to silicone hydrogel is preventative care. Recent research shows that 87% of ECPs say that if they want to keep patients from dropping out of lens wear, they would refit them with 1-day silicone hydrogel lenses. If a patient is wearing hydrogel on a full-time basis, proactively switching to silicone hydrogel can save the patient from the same type of symptoms you'd expect in a full-time frequent replacement hydrogel wearer.

Dr. Frogozo: I agree. Also, if the patient is currently wearing hydrogel lenses, they may already have hypoxia—in which case I point this out to illustrate why I'm making the change. If they don't yet have signs of hypoxia, they soon may. I don't want patients dropping out if there's something I can do to prevent it from happening in the first place.

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1 Industry third-party data.
2 Manufacturer stated oxygen transmissibility values (Dk/t): MyDay® daily disposable (100), clariti® 1 day (86), 1-DAY ACUVUE® MOIST® (25.5), SofLens® daily disposable (24).
3 With higher oxygen transmissibility than hydrogel materials, silicone hydrogel contact lenses minimize or eliminate hypoxia-related signs and symptoms during lens wear.
*It is essential that patients follow eye care practitioner's directions and all labeling instructions for proper use of lenses.
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Clear the Air—and the Vitreous

PPV for floaters is controversial. But maybe it's time to take a closer look at this management strategy. **By Diana Shechtman, OD, Rashid Taher, MD, and Jay M. Haynie, OD**

By far, floaters are the most common complaints in a retina practice. Chronic floaters may significantly impact a patient's quality of life, affecting everything from reading to driving. Their causes can include any number of processes, such as posterior vitreous detachment (PVD), myopic vitreopathy, vitritis, vitreous hemorrhages, retinal detachment, retinal breaks and vitreous liquefaction.

Managing these underlying causes remains the primary treatment approach for the associated floaters. Still, most floaters are benign and are treated simply with observation, which does not always address the patient's symptomology.

The surgical treatment of floaters remains a topic of conflict, especially given that many patients are young, phakic and have good preoperative visual acuity. Although treatment is associated with significant resolution of symptoms, there is a small but not insignificant rate of complications such as retinal, vitreous or suprachoroidal hemorrhages, cataract formation, endophthalmitis, macular edema and iatrogenic retinal breaks and/or retinal detachments.

In our practice, Dr. Rashid Taher has found floaterectomy—pars plana vitrectomy (PPV) for the treatment of floaters—to be a viable option for well-selected patients, yielding favorable outcomes, high surgical success rate and minimal associated complications. The key to our success lies in our careful assessment of individual inclusion criteria prior to surgical intervention. This ensures we meet patient expectations while also mitigating the risk of complications. Such criteria can include, but are not limited to, complaints exceeding three months, as well as a perceivable, significant impact on quality of life, often measured with the NEI-VFQ-25 questionnaire. To diminish the possibility of iatrogenic retinal breaks or detachments, we typically evaluate for the presence of an observable PVD. Additionally, any vitreoretinal pathology, such as a retinal break, is typically treated during the procedure with endolaser barrier. Core vitrectomy is implemented with preservation of the anterior hyaloid face to delay cataract formation.

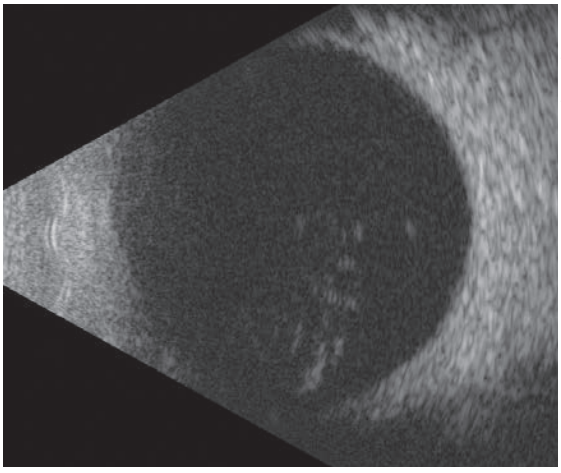

Photo: Jay M. Haynie, OD

An ultrasound of symptomatic vitreous floaters in a patient considering surgical management.

We also use ultrasonography as an objective measurement to classify the state of the vitreous. Subjectively, pre-testing contrast sensitivity may have a substantial contribution to patient satisfaction and aid in determining the need for floaterectomy. Sebag's studies show that floaters have a significant impact on contrast sensitivity, which can correlate to patient complaints and will dramatically improve following the surgical procedure.

Floaters Be Gone

By Drs. Shechtman and Taher

A 56-year-old white male presented to our clinic with a four-month complaint of floaters affecting his left eye more than his right. Past medical history was unremarkable. His best-corrected visual acuity was 20/20 OD and 20/25- OS. Slit lamp evaluation revealed mild cataracts in both eyes. Dilated fundus exam revealed pink, distinct optic nerves with physiological cupping. The macula was flat and intact OU, and the periphery did not reveal any pathology in either eye. The vitreous revealed syneresis OU, and a PVD was apparent in the left. Ultrasound revealed numerous floaters OU and no evidence of retinal detachments, retinal breaks or mass.

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The patient was advised of the risks and benefits associated with floaterectomy, after which he opted to schedule surgery.

The patient was followed at one day and one week postoperatively, the fundus showed cleared vitreous with no peripheral pathology. Ultrasonography revealed resolution of floaters with no evidence of retinal detachments, retinal breaks or mass. The patient expressed satisfaction regarding the resolution of floaters and was asked to return to clinic within one month. He was educated on the signs and symptoms of retinal breaks and detachments and was asked to return to the clinic sooner if he noticed them.

How Our Practice Compares

By Dr. Haynie

Managing symptomatic floaters in my practice first involves differentiating acute vs. chronic symptoms. Acute symptoms secondary to a PVD will often resolve over time, and the discussion to consider surgical removal is not part of the management strategy. The priority for acute symptoms is to ensure there is no peripheral retinal breaks or holes that could be associated with a higher risk of complications such as a retinal detachment. In most cases, the vitreous floaters will settle and resolve, providing resolution of symptoms.

Management of chronic floaters—present for one year or more in my practice—can include laser vitreoloysis or vitrectomy surgery. Although laser vitreolysis has been available for two decades, my practice has yet to embrace this as a treatment for symptomatic floaters. In my area, a few anterior segment surgeons offer this treatment. Several of my patients have had laser vitreolysis and, unfortunately, continue to have symptoms; in some cases, the number of floaters increased. Of course, I likely only see those patients for whom treatment failed, not those who are satisfied with their outcomes. Some complications of vitreolysis I have seen personally include posterior capsule tears, retinal burns, foveal burns, choroidal rupture and choroidal hemorrhages. In addition, the predictability of success is highly variable. Thus, my surgeons prefer PPV as the definitive treatment for chronic floaters.

Evaluating a patient for vitrectomy surgery for symptomatic floaters first involves patient selection. This begins with a clinical examination of the vitreous to determine if a PVD has occurred. In the absence of a PVD, the surgeon will need to mechanically induce a PVD, which can increase postoperative complications or peripheral retinal breaks. We next determine the magnitude and effects of floaters. I like to look at patients with a 20D condensing lens on the visual axis to see if I can appreciate any shadows of floaters cast on the retina. If I can, then it is likely the patient will have fewer symptoms following surgery. Other ways of demonstrating the "shadow effect" are widefield red-free photography or optical coherence tomography.

Once we have determined surgery is appropriate, we provide the patient a detailed explanation of the risks, benefits and alternatives. The risks of vitrectomy surgery parallel those of cataract surgery; fortunately, the risk of any sight-threatening complication is quite low. In addition, if the patient is phakic, it is important that they understand that having vitrectomy surgery will accelerate the formation of a nuclear cataract earlier than the natural history.

One hang-up with vitrectomy for symptomatic floaters from a referring doctor's stand-point is the risk of the surgery itself; however, the evolution of cataract surgery minimizes even this concern. Thirty years ago, when cataract surgery was associated with a higher incidence of complications, patient selection was key, and it was not indicated for everyone. Today's cataract surgery has had major upgrades and is now the most common ocular surgery performed—and with great success in the majority of cases.

Likewise, retina surgery has also evolved. Current systems use much smaller-gauge instruments (25 or 27) compared with the 20-gauge needles used less than 15 years ago. Today's smaller vitrectomy instruments allow for sutureless procedures, less inflammation, fewer complications and, in the end, a much greater success rate.

For each patient presenting with floaters, think about someone with cataracts and why that surgery is being done: in most cases, the patient has developed functional vision loss and secondary symptoms of glare and night driving difficulties that are impacting their quality of life. Similarly, the retina patient with symptomatic floaters also has functional vision loss with symptoms of reading difficulties, loss of contrast acuity and the burden of distraction. Vitrectomy surgery is the definitive option, and patients with symptoms greater than one year in duration deserve the opportunity to discuss surgery with a vitreoretinal surgeon.

We never think twice about referring someone for a cataract evaluation; it's time we provide similar enthusiasm for patients looking to manage their chronic floaters. ■

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Pathways for Achieving Premium Outcomes

Harnessing advanced IOL technology & co-management opportunities to take eye care to the next level.

Dear Reader,

Optometrists will play an increasingly critical role in helping patients achieve the refractive outcomes they expect from advanced intraocular lenses (IOLs). As eye care professionals who will oversee much of the pre- and postoperative care for our patients, it is incumbent upon us to take the right clinical steps surrounding refractive procedures. In addition, we must work collaboratively with cataract surgeons to help ensure the best possible outcomes and high patient satisfaction.

For one thing, we must be aware of how astigmatism can potentially impact refractive results and recommend options to effectively correct it. With new toric and presbyopia-correcting IOL designs, we have an invaluable opportunity to educate patients about advances that can improve their vision. In doing so, we can expand our role in medical eye care and in co-management of our patients with the cataract surgeon.

In addition, research has shown us the importance of the pre-corneal tear film as a refractive interface. With the understanding that tear film irregularities can significantly degrade retinal image and vision quality with multifocal IOLs, we must screen patients for ocular surface disease and manage dry eye before referring them for surgery.

It's clear that proactively informing our patients about new technologies available to them, making referrals when appropriate, and co-managing ocular surface care surrounding refractive procedures will offer us additional ways to further the continuum of care for our patient base. This will serve to build trust with our patients and cement doctor-patient relationships well into the future.

Supported by independent medical educational grants from Johnson & Johnson Vision and Carl Zeiss Meditec

Managing Astigmatism With Innovative IOL Technology

Ways to help match astigmatic patients with advanced refractive solutions.

By Paul Karpecki, OD, FAAO

Approximately 4 million cataract procedures were performed in 2017—a number that will continue to grow. More than 70% of patients have more than 0.50D of preoperative astigmatism, which must be addressed to achieve optimal uncorrected visual outcomes from cataract surgery.[1]

We are an essential part of this process. Cataract surgery is the number-one growth area of medical procedures, but the number of surgeons is declining.[2] Therefore, our role in medical eyecare and co-management will increase.

With new toric intraocular lens (IOL) designs, we have an invaluable opportunity to educate patients about advances that can improve their vision.

Although many astigmatic cataract patients are within the treatable range of toric IOLs, only approximately 10% receive them.[1,3-5] Barriers include cost and a lack of awareness.[6]

PRECISE DIAGNOSTICS

Keratometry and autorefractors are usually sufficient to assess astigmatism power and axis, although optical biometry, corneal topography, and optical coherence tomography can be useful in some cases (*Figure 1*). Using more advanced diagnostics and related assessments enables doctors to justify certain fees for service with premium IOLs.

For biometry, the IOLMaster (Zeiss) provides keratometry at 2.5-mm diameter, six data points, and three keratometry measurements, and the Lenstar (Haag-Streit) provides keratometric readings at 1.65 and 2.3 mm, four keratometric scans on two concentric rings, and 32 data points.

Additional tests, such as the IOL Master, should be performed if measurements are inconsistent or the patient has high astigmatism in either eye, small or large eyes, or steep or flat eyes.

To reduce error, we need to perform measurements before drops are instilled and diagnose and treat dry eye, which can impact measure-

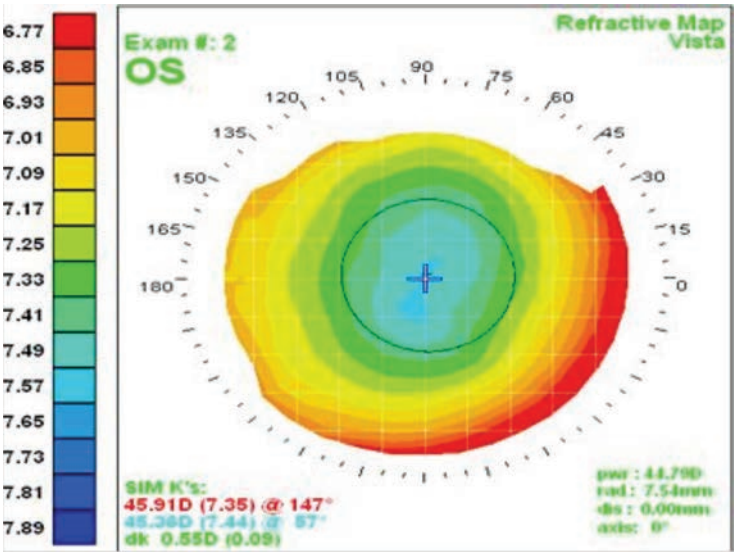

Figure 1. Corneal topography shows 0.50D of corneal astigmatism, whereas the manifest refraction shows 1.75D of overall astigmatism.

ments and surgical outcomes.

Patient satisfaction correlates with cylinder correction. According to a study performed at Optical Express Centers in the United Kingdom, approximately 82% of 4,970 eyes that had refractive lens exchange with no postoperative cylinder had 20/20 uncorrected distance vision, but approximately 54% of those with 1.00D cylinder had 20/20 vision.

If the IOL is rotated only 1 degree, patients lose 3.3% of the effect of the toric IOL.

MAKING THE MOST OF TORIC IOLS

Patient selection is about meeting each patient's visual needs. Toric IOL recipients should seek good uncorrected distance vision and accept that they may need glasses for near.

In a study of monofocal toric IOLs, 88% of patients achieved 20/20 or better monocular corrected distance vision, and in a study by Daniel Black, MD, 94% of eyes achieved 0.50D or less residual refractive cylinder with these lenses.[7,8]

Patients have high expectations and need to know all available choices, which include monofocal IOLs with incision correction, toric IOLs, and presbyopia-correcting toric IOLs (multifocal, extended depth of focus).

Although many patients have at least 0.50D of astigmatism, only those with corneal astigmatism benefit from toric IOLs.

Toric IOLs work well in eyes with regular astigmatism, but eyes with irregular astigmatism will not achieve the same results.

We have more flexibility in with-the-rule (WTR) astigmatism, which slowly shifts toward against-the-rule (ATR) with time, but ATR astigmatism has

"With new toric intraocular lens designs, we have a great opportunity to educate patients about advances that can improve their vision." — *Paul Karpecki, OD, FAAO*

more impact on vision and may worsen. Most patients benefit if we correct smaller amounts of ATR astigmatism. If a person has 0.75D or 1.00D of ATR or oblique corneal astigmatism, we consider a toric IOL.

Posterior corneal astigmatism works like a minus lens, creating net plus power along the horizontal meridian.[9,10] The impact of posterior WTR astigmatism is similar to ocular ATR astigmatism.

MANAGING RESIDUAL ASTIGMATISM

In patients with postoperative refractive error, we need to repeat the manifest refraction multiple times to be sure it is stable and search for other conditions, such as ocular surface disease (OSD) or cystoid macular edema. Significant OSD should be treated and stabilized before the manifest refraction is repeated. We also need to be sure toric IOLs are positioned correctly.

Postoperative residual astigmatism can be corrected with glasses or contact lenses, limbal relaxing or arcuate incisions, LASIK or PRK, IOL rotation, or IOL exchange. If rotation or explantation is necessary, it should be performed early and before Nd-YAG.

HELPING PATIENTS ACHIEVE OPTIMAL OUTCOMES

As we look to the future, optometrists will play an increasingly critical role in helping patients achieve the refractive outcomes they expect from advanced IOLs. We need to be aware of how astigmatism can potentially impact refractive results and recommend options to effectively correct it.

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Pathways for Achieving Premium Refractive Cataract Outcomes:
Ocular Surface as the First Refractive Surface

Managing ocular surface disease for precise preoperative measurements and optimal outcomes

By Douglas K. Devries, OD

Of the three refractive interfaces of the eye—the pre-corneal tear film, cornea, and lens—the pre-corneal tear film is the first and most important.

Tutt et al. reported that tear film irregularities can degrade retinal image quality by as much as 40%.[1] By disrupting the passage of light, tear film irregularities also impact vision quality with multifocal intraocular lenses (IOLs).

Dry eye often decreases contact lens use and leads to dropout. If we intervene when signs and symptoms first appear, patients will be more likely to wear their contact lenses longer and be better prepared by the time they need cataract surgery. If dry eye was not diagnosed

Patient Condition	% with MGD
Dry eye	86%[1]
Perimenopause	79%[2]
Polycystic ovary syndrome	73%[3]
Glaucoma (on prostaglandins)	96%[4]
Glaucoma (non prostaglandin)	58%[4]
Diabetes	58%[5]
VDT users (4+ hrs per day)	85%[6]
Cataract patients	59%[7]
Contact lens wearers	60%[8]

Figure 1. Prevalence of MGD in certain conditions.

1. Cornea 2012; 31:472-478
2. Medicine [Baltimore] 2016; 95:e4268
3. Curr Eye Res 2016; 28:1-5
4. J Glaucoma 2016; 25:770-774
5. Int J Ophthalmol 2016; 9:1740-1744
6. PLoS One 2014; 9:e105575
7. Cornea 2016; 35:731-735
8. Cornea 2015; 34:1098-1104

and treated previously, intervention is necessary before patients are referred for cataract surgery.

IMPACT ON CATARACT SURGERY AND OUTCOMES

Dry eye is particularly common in the patient population with cataracts, who also have co-existing conditions or take medications that may worsen dry eye. *Figure 1* shows the prevalence of meibomian gland disease (MGD) in certain conditions.

The PHACO study by Trattler et al. showed that approximately 77% of patients scheduled for surgery had pos-

"Ocular surface management is a good opportunity for shared care." — *Douglas K. Devries, OD*

itive corneal fluorescein staining.[2] Most had no symptoms, although blurred vision and clinical signs were common.

Lemp et al. reported that 86% of patients with a classified subtype of dry eye had MGD.[3]

Ocular surface disease (OSD) impacts keratometry, corneal topography, refraction, and IOL power calculations. Epitropoulos et al. reported that 17% of hyperosmolar eyes had more than a 1.00D difference in keratometric cylinder and 10% had a greater than 0.50D change in IOL power.[4]

Even if the correct IOL is selected, after surgery patients may have suboptimal visual quality, irritation, and poor healing.

DIAGNOSING AND TREATING OSD

Necessary diagnostics for dry eye include a symptom questionnaire, thorough history, slit lamp exam, evaluation of the tear meniscus, fluorescein staining, tear breakup time, and meibomian gland function assessment.

Point-of-care tests, such as tear osmolarity, inflammation (MMP-9), and lactoferrin protein and/or IgE, are useful in differentiating types of OSD.

Meibography allows us to show patients what their meibomian glands look like in comparison with normal glands. Corneal topography provides placido images where we can see tear film irregularities.

Tear film management strategies depend on the cause and severity of OSD, as well as economic factors and other considerations.

Palliative therapy includes artificial tears, gels, ointments, and environmental changes. We also may recommend omega-6/omega-3 fatty acid supplements.

Prescription medications include cyclosporine and lifitegrast. Lifitegrast was approved for both signs and symptoms and can improve symptoms in as early as 2 weeks.[5]

Short-term corticosteroids also are prescribed for dry eye. In addi-

tion, tetracycline, doxycycline, and azithromycin may be used to treat meibomian gland inflammation.

We also recommend moist heat and lid scrubs, as well as thermal pulsation in cases with significant obstruction, which may be combined with intense pulsed light. Microblepharoexfoliation may be advised to remove the biofilm and debris.

Additional treatments may include punctal plugs and neurostimulation.

CO-MANAGING CARE

Ocular surface management is a good opportunity for shared care. It is important to screen patients for OSD and manage dry eye before referring patients for surgery. We need to explain to patients that dry eye is a chronic condition, so treatment will be ongoing.

Surgical practices prefer to see referred patients with a healthy tear film and ocular surface, and optometrists can be compensated for medical eye care and a greater portion of surgical co-management. Furthermore, patients would rather not return to the surgery center to repeat biometry measurements.

1. Tutt R, Bradey A, Begley C, et al. Optical and visual impact of tear break-up in human eyes. Invest Ophthalmol Vis Sci. 2000; 41:4117-4123.

2. Trattler WB, Majmudar PA, Donnenfeld ED, McDonald MB, Stonecipher KG, Goldberg DF. The Prospective Health Assessment of Cataract Patients' Ocular Surface (PHACO) study: the effect of dry eye. Clin Ophthalmol. 2017; 11:1423-1430.

3. Lemp MA, Crews LA, Bron AJ, Foulks GN, Sullivan BD. Distribution of aqueous-deficient and evaporative dry eye in a clinic-based patient cohort: a retrospective study. Cornea. 2012; 31:472-478.

4. Epitropoulos AT, Matossian C, Berdy GJ, Malhotra RP, Potvin R. Effect of tear osmolarity on repeatability of keratometry for cataract surgery planning. J Cataract Refract Surg. 2015; 41:1672-1677.

5. Holland EJ, Luchs J, Karpecki PM, Nichols KK, Jackson MA, Sall K, Tauber J, Roy M, Raychaudhuri A, Shojaei A. Lifitegrast for the treatment of dry eye disease: results of a phase III, randomized, double-masked, placebo-controlled trial (OPUS-3). Ophthalmology. 2017; 124:53-60.

Dr. Devries is co-founder of Eye Care Associates of Nevada. He is a speaker or consultant for the following: Allergan, Bausch + Lomb, Bio-Tissue, Bruder Healthcare, BVI, Eyes4Lives, Johnson & Johnson Vision/AMO, OcuSoft, Ophthalmic Resources, RPS, RySurg, TearLab, TearScience, ScienceBased Health, Shire, Sun Pharmaceuticals, VMax Vision.

9th Annual Retina Report

Busted Barriers: Triaging Retinal Hemorrhages

While uncommon, this clinical finding can be a harbinger of bigger issues. Here's what you need to know.

By Heather Whyte DeMarco, OD, Diana Mah, OD, Justin Cole, OD, and Jarett Mazzarella, OD

Retinal hemorrhages, often first diagnosed in the primary eye care setting, can be a presenting finding in many ocular and systemic disease states. Because of the blood-retinal barrier, which aids in isolating blood from the retina to avoid retinal toxicity, hemorrhages within the retina are rare unless there is insult to the ocular or systemic vasculature. Such damage can be caused by vascular disease, hematologic disorders and dyscrasias, infections, trauma or hypoxia. In addition, they can be idiopathic (*Table 1*).[1] A look at the clinical presentation of retinal hemorrhages and the pathophysiology that leads to their correct identification and classification can help you work through the differential diagnoses and decide whether to monitor or refer.

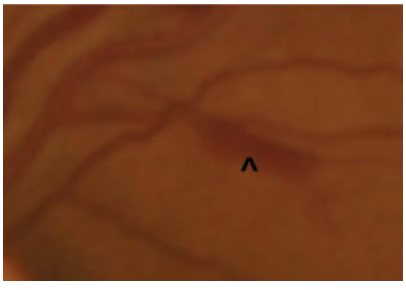

Fig. 1. This preretinal hemorrhage forms a boat shape and blocks underlying retinal vasculature detail.

Classification

Shape, location and size, as well as associated signs and symptoms, are all key to properly classifying a retinal hemorrhage. To begin, clinicians should document the location and depth with respect to the vitreal-retinal-choroidal interfaces. These, along with distribution of the hemorrhage, can give clues to the etiology and help uncover possible systemic comorbidities. Starting anteriorly, the classification of hemorrhages based on depth includes:

1. Vitreous and preretinal. A vitreous hemorrhage represents leakage of blood in and around the vitreous humor. Due to the lack of boundaries, these hemorrhages tend to diffuse throughout the vitreous and, in chronic cases, can settle inferiorly. Upon clinical evaluation, this may lead to a "hazy" appearing fundus due to the collection of red blood cells floating in the vitreous, especially in acute cases. A fresh vitreous hemorrhage often appears bright red, and turns to a more white or cream color due to red blood cell dehemoglobinization over time.

The subhyaloid is located between the posterior limiting layer of the vitreous and the internal limiting membrane (ILM) of the retina. Preretinal anatomical boundaries also exist between the internal limiting membrane and the retinal nerve fiber layer (RNFL). Thus, hemorrhages in both of these locations have more defined structural boundaries and are often indistinguishable (*Figure 1*). Like vitreous hemorrhages, subhyaloid and preretinal hemorrhages also block underlying retinal features and can reduce visual acuity when located near the macula.[2]

Preretinal, subhyaloid and vitreous hemorrhages are largely controlled by gravity. Preretinal and subhyaloid hemorrhages are darker at the bottom of the hemorrhage, where the blood has settled with a distinct hori-

zontal line at the top, known as a "D" or boat shape.[2]

Due to their location, vitreous, subhyaloid and preretinal hemorrhages may be associated with abnormal retinal vessels, specifically in the superficial capillary plexus. These hemorrhages can also be associated with neovascularization of the nerve or retina as these leaky, fragile vessels often proliferate on the surface of the retina and scaffold onto the posterior hyaloid face. Other etiologies include traction from posterior vitreal separation, leading to a breaking or shearing of superficial retinal capillaries, as well as proliferative diabetic retinopathy, trauma, retinal break, vascular occlusion, Terson's syndrome, hypertensive retinopathy and proliferative vitreoretinopathy. Certain cases may have no known etiology and are referred to as idiopathic.[2,3]

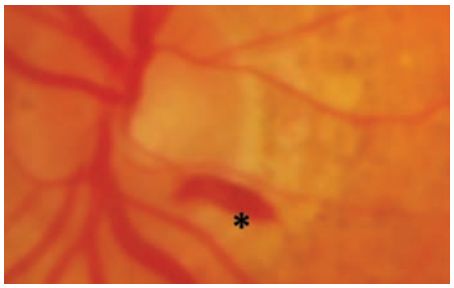

Fig. 2. RNFL hemorrhages can have an elongated shape, such as this one.

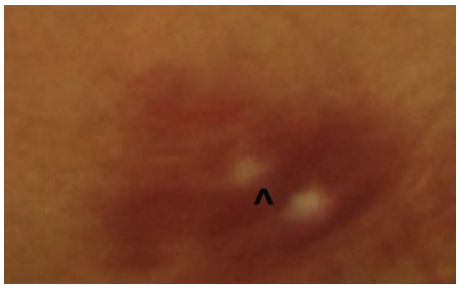

Fig. 3. The white centers of this superficial RNFL hemorrhage are called Roth spots.

2. RNFL. Hemorrhages in this location typically have a thin or elongated shape due to the parallel construction of ganglion cell axons and the distribution of RNFL bundles in the retina. These hemorrhages can be feather, flame, splinter or brush-stroke in appearance (*Figure 2*).[2] They are often found in the posterior pole, last approximately six weeks and typically occur due to pathology of the superficial retinal capillary plexus.[4,5] Researchers postulate flame-shaped hemorrhages are primarily affected by artery-based diseases such as hypertension, blood dyscrasias and anemias.[2]

Occasionally, a superficial hemorrhage may have a white center, in which case it is called a Roth spot,

Table 1. Conditions that Can Present with Retinal Hemorrhages [10,20-24]

Disease	Type of Retinopathy	Typical Laterality
Hypertension	Flame-shaped or intraretinal hemorrhages, microaneurysms/macroaneurysms, cotton wool spots, pre-retinal hemorrhage (rare).	Bilateral, can be asymmetric
Diabetes	Preretinal/vitreous or intraretinal (dot/blot) hemorrhages, microaneurysms, cotton wool spots.	Bilateral, can be asymmetric
Retinal Vein Occlusion	Flame-shaped or intraretinal hemorrhages, cotton wool spots, Bonnet's sign.	Unilateral
Ocular/Head Trauma	Preretinal/vitreous, intraretinal or subretinal hemorrhages, choroidal neovascular membrane in cases of choroidal rupture.	Unilateral, depends on history/type of trauma
Anemia	Flame-shaped or intraretinal hemorrhages, cotton wool spots, Roth spots and vitreous hemorrhage in severe forms.	Bilateral
Leukemia	Preretinal/vitreous, flame-shaped or intraretinal hemorrhages, cotton wool spots, Roth spots, sea-fan neovascularization.	Bilateral
Acute Bacterial Endocarditis	Preretinal/vitreous, intraretinal or flame-shaped hemorrhages in the parapapillary rim, cotton wool spots, Roth spots.	Bilateral
Sickle Cell Retinopathy (SC & S-Thal, most common to have retinopathy)	Arteriole occlusions, intraretinal hemorrhages (pink or salmon patch), cotton wool spots, angioid streaks, black sunburst chorioretinal scars, sea-fan neovascularization with vitreous hemorrhage and tractional retinal detachment.	Bilateral
Connective Tissue Disorders (lupus)	Intraretinal hemorrhage, cotton wool spots, vascular occlusions (severe stages).	Bilateral
Pre-eclampsia	Intraretinal hemorrhages, cotton wool spots, Elschnig spots, arteriolar narrowing, serous detachment.	Bilateral
Ocular Ischemic Syndrome	Intraretinal hemorrhages (located mid-peripherally), cotton wool spots, arteriole narrowing, severe forms can develop retinal neovascularization.	Unilateral/usually asymmetric when bilateral
High Altitude Retinopathy	Vitreous and intraretinal hemorrhages, Roth spots, optic disc edema.	Bilateral
Carbon Monoxide Poisoning/Anoxia	Vitreous and intraretinal hemorrhages, Roth spots, optic disk edema.	Bilateral
Vitamin D Deficiency	Flame-shaped or intraretinal hemorrhage, cotton wool spots.	Bilateral

Case Example

A 59-year-old African-American male presented with a history of vision loss OS, beginning two days earlier with gradual improvement. He reported a dull ache over his left eye but no other ocular symptoms. Systemically, he reported feeling extremely tired for the past few weeks with no other symptoms. He had a history of high blood pressure, arthritis and borderline hyperglycemia. He was taking atenolol 50mg daily for his blood pressure. He had an ocular history of an old blow-out fracture OD, without any ocular sequelae. Visual acuities were 20/25 OD and 20/40^{-2} pinhole to 20/30 OS. Pupils were equal, round and reactive to light with no afferent pupillary defect OU. Intraocular pressures were 13mm Hg OD and 15mm Hg OS. Slit lamp exam was unremarkable.

Dilated exam showed a cup-to-disc ratio of 0.2 vertical OD and 0.25 vertical OS with mild peripapillary edema OS. The macula was clear OD, but OS showed retinal thickening nasal to the fovea. Scattered RNFL and intraretinal hemorrhages and cotton wool spots (CWS) were noted in the posterior pole OS greater than OD. Presumed Roth spots were noted along the inferior temporal arcade OD (*Figure 1*). A peripapillary RNFL OCT confirmed peripapillary thickening OS greater than OD and a macular cube OCT confirmed mild inner retinal thickening nasal to the fovea OS (*Figures 2 and 3*). This thickening on OCT OU correlated to CWS OU in the RNFL of the retina.

The patient was diagnosed with bilateral retinopathy of unknown etiology. Lab work ordered included complete blood count, BUN, HgA1c, sed rate, CRP, sickle cell screen, ACE, ANA, Lyme titer and RPR. The patient was scheduled in four days to review the lab work and order any additional testing if needed. Later that day, the lab notified the provider of critically low white blood cell count and low platelet and RBC counts.

After coordinating with the on-call physician, the patient was transferred to the emergency room due to pancytopenia with concern for leukemia. A hematologist reviewed the case and recommended a bone marrow biopsy. The patient was discharged and a bone marrow biopsy was performed several days later, indicating acute myeloid leukemia. The patient was subsequently admitted for chemotherapy.

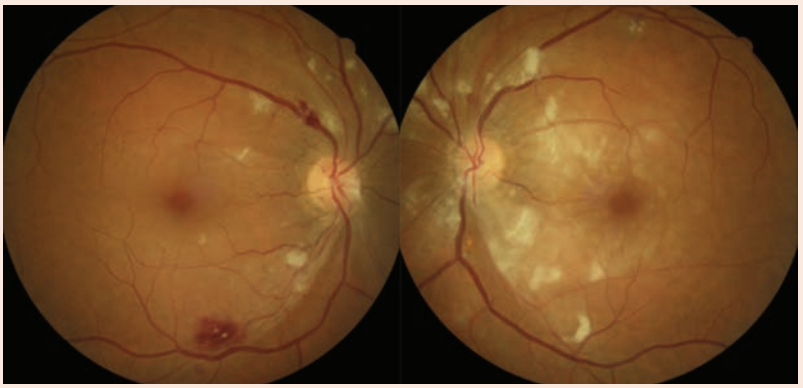

Fig.1. The patient's posterior pole fundus photos OU show scattered CWS with RNFL and intraretinal hemorrhages.

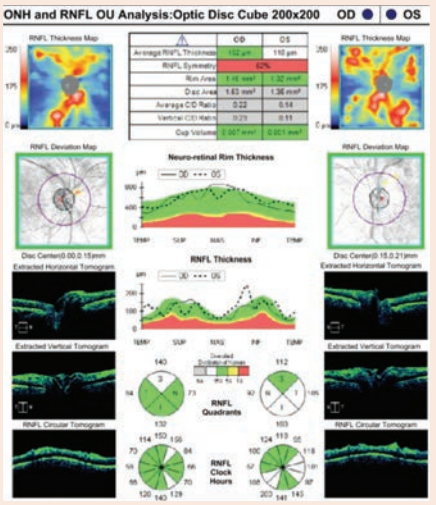

Fig. 2. In the ppRNFL OCT, OS demonstrates increased average thickness outside the normative database. Reduced symmetry is noted OD to OS in the ppRNFL. The TSNIT graph shows significant thickening of the superior ppRNFL OD and the nasal/temporal and inferior RNFL OS. Horizontal and vertical tomographs show no lazy 'V' sign indicative of papillitis; however, the RNFL thickness maps indicate focal peripapillary thickening, whose location corresponds to that of the numerous CWS noted on the fundus images.

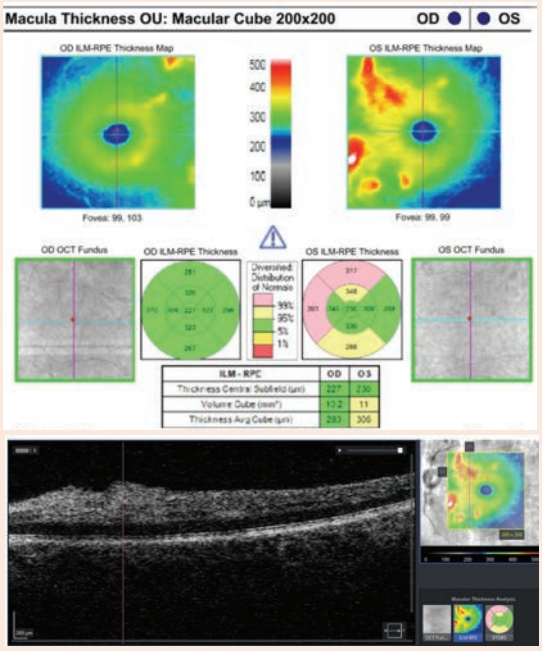

Fig. 3. In the macular cube OCT, the right eye shows normal thickness values compared with normative values. The superior nasal edge of the thickness map OD shows significant elevation correlating to CWS. The OS shows paramacular focal thickening corresponding to CWS. The OS horizontal B-scan shows mild inner retinal hyper-reflectivity/ thickening associated with CWS localized to the RNFL.

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which can be caused by ischemia, fibrin accumulation or white blood cell accumulation (*Figure 3*).[2,6] Roth spots are categorized as a non-specific sign of blood dyscrasia.[7] Examples of associated etiologies include: anemia, anoxia, arteriovenous malformations, collagen vascular disease, diabetic retinopathy, hypertensive retinopathy, HIV, leukemia, multiple myeloma, trauma and radiation.

3. Intraretinal. Commonly known as "dot or blot," these are found within the inner nuclear or outer plexiform layers of the retina. They fill the entirety of the retinal layers, occupying and displacing the normal retinal architecture, therefore forming round, uniform hemorrhages.[2,7] The compressive force from the surrounding layers leads to the typical dot or blot shapes (*Figure 4*). Intraretinal hemorrhages appear slightly darker red than other hemorrhages. They typically have a pre-venular deeper capillary layer origin and therefore are associated with vein-based conditions or congestive disease. Conditions such as diabetes, venous occlusive disease, ocular ischemic syndrome, sickle cell retinopathy and juxtafoveal telangiectasia can lead to evidence of pathology in the deeper retinal layers.[2]

Fig. 4. Intraretinal hemorrhages are round and uniform, often forming dot or blot shapes.

Table 2. Possible Laboratory Blood Workup for Retinal Hemorrhages[25-31]

Test	Indication
Complete blood count; Platelet count	• Rule out blood dyscrasias (anemia/leukemia), infection and hematologic etiologies. • Low hemoglobin or a low hematocrit may indicate anemia due to excessive bleeding, chronic disease.
Erythrocyte sedimentation rate; C-reactive protein	• Represent nonspecific markers of inflammation within the body.
Fasting glucose; Hemoglobin A1c	• For metabolic syndrome; fasting plasma glucose provides current blood sugar; HgbA1c provides insight over a three-month period.
Lipid profile	• Evaluate for hyperlipidemia and its association with metabolic syndrome, increased risk of vascular disease.
Prothrombin time/ partial thromboplastin time	• Assess the time it takes a clot to stop bleeding; clotting time can be slowed by blood thinners and can increase in females taking estrogen-based birth control medications.
Protein C and protein S	• Decreased levels can effectively limit the ability to stop clotting, putting those individuals at risk for thrombotic events. This can present in the retina in young patients with venous occlusions.
Factor V leiden	• Defects in clotting should also be ruled out in patients that have no other systemic risk factors. • These patients are at increased risk for deep vein thrombosis and pulmonary embolisms.
Prothrombin gene mutation	• Puts patients at risk for retinal vascular occlusions and should help diagnose those at risk for stroke secondary to undiagnosed thrombophilia.
Homocysteine	• In the presence of hemorrhages with retinal venous occlusion, especially in younger patients without other risk factors. Elevated homocysteine levels can be associated with blood clotting and atherosclerosis.
Antithrombin III	• A deficiency can be associated with thrombotic events which can cause vascular retinal occlusions.
Antiphospholipid antibodies	• Rule out antiphospholipid syndrome, which can cause vascular thrombosis.
ELISA	• Rule out infectious etiology, such as HIV testing in at-risk individuals.
Lyme titers; Herpes simplex titers; Syphilis testing FTA-ABS/RPR	• Should always be considered in cases where the diagnosis is unclear, as syphilis is known as the great masquerader.
ANA	• Positive in 95% of cases of systemic lupus erythematosus.
Anti-dsDNA	• Found in roughly half of people with lupus; Anti-Sm affects the cell nucleus and is usually positive in patients with lupus.
Anti-RNP antibody testing	• Is present in many autoimmune diseases, including lupus.
Anti-histone antibodies	• Likely found in patients with lupus secondary to medications.
Anti-Ro/SS-A, anti-La/SS-B antibodies	• Found in patients with Sjögren's syndrome and are important for women with lupus, as these antibodies can affect the neonatal fetus.
HLA-B5/B12	• For conditions that present with anterior and posterior segment findings; Rule out Bechet's disease.
HLA-B27	• Rule out Reiter's syndrome, inflammatory bowel disease, psoriatic arthritis and ankylosing spondylitis.

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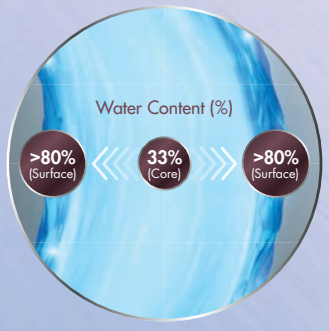

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References: 1. Angelini TE, Nixon RM, Dunn AC, et al. Viscoelasticity and mesh-size at the surface of hydrogels characterized with microrheology. *Invest Ophthalmol Vis Sci.* 2013;54:E-abstract 500. **2.** Pitt WG, Jack DR, Zhao Y, et al. Transport of phospholipid in silicone hydrogel contact lenses. *J Biomater Sci Polym Ed.* 2012;23(1-4):527-541. **3.** Greiner JV, Glonek T, Korb DR, et al. Phospholipids in meibomian gland secretion. *Ophthalmic Res.* 1996;28(1):44-49. **4.** Shine WE, McCulley JP. Polar lipids in human meibomian gland secretions. *Curr Eyes.* 2003;26(2):89-94. **5.** Michaud L, Forcier P. Comparing two different daily disposable lenses for improving discomfort related to contact lens wear. *Cont Lens Anterior Eye.* 2016;39(3):203-209.

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Hemorrhage

4. Subretinal. These hemorrhages, located underneath the photoreceptor layer of the retina and above the retinal pigment epithelium (RPE), are not confined laterally, which tends to make them appear broader with indistinct borders. They are confined anteriorly by the overlying retina and posteriorly by the tight junctions of the RPE. The hemorrhage is red, but not as bright a red as more superficial inner retinal hemorrhages due to their depth (*Figure 5*). Subretinal hemorrhages can be caused by choroidal neovascularization membranes (CNVM) types 1 through 4.

5. Sub-RPE. Located between the RPE and Bruch's membrane, these appear dark red with defined borders (*Figure 6*). Based on the depth, the overlying retinal vessels can be visualized.[2,8] A common etiology of these hemorrhages is a CNVM. However, clinicians must also rule out less common causes such as acute trauma, as in a choroidal rupture, or choroidal tumor.

Demographics

While trauma can cause retinal hemorrhages in any age group, bilateral hemorrhages in patients younger than three warrant suspicion of child abuse.[1,9] Other differentials include: retinopathy of prematurity, persistent hyperplastic primary vitreous, Coat's disease (acquired) and familial exudative vitreoretinopathy (inherited). Here, a thorough case history in regards to birth, trauma and any family history of ocular conditions is key.

Retinal hemorrhages in young adults are most commonly caused by Eales disease (male predilection), high altitude, Valsalva maneuvers or hematologic conditions caused by genetic disorders, anemias, blood cancers, sickle cell disease (higher frequency in people of African descent) or autoimmune conditions such as systemic lupus erythematous.[10] In particular, almost half of patients with acute leukemia have retinal hemorrhages that are commonly intraretinal within the posterior pole and may be white-centered.[11,12]

Retinal vascular changes in young female patients may be associated with oral contraceptive use, as they can cause an increase in blood viscosity.[13,14]

Retinal hemorrhages in middle-aged and older patients are most commonly a result of systemic vascular conditions such as hypertension, diabetes and carotid occlusive disease, as well as vascular occlusions, posterior vitreous detachment and retinal tears.[1,5,15]

Patients older than 65 are at a greater risk of developing macular degeneration, and 30% of patients older than 75 have signs of maculopathy.[16,17] This could result in the presence of retinal hemorrhages secondary to conversion to exudative macular degeneration. Also,

peripheral exudative hemorrhagic chorioretinopathy, which may be a variant of age-related macular degeneration, has a mean onset of age 80 to 85.[18] Patients within this age group are also at risk for retinal hemorrhaging secondary to manifestations of systemic conditions such as diabetes and hypertension, although one study shows hypertension explained less than half of retinal hemorrhages found in patients older than 60.[19]

Diagnostics

Although a solitary retinal hemorrhage in one eye may not raise alarm, large interocular asymmetries in retinopathy should increase your suspicion and lead you to consider etiologies other than hypertension and diabetes.

One of the most crucial components of any ocular examination is a thorough case history, including social history. Medication use, history of trauma, history of recent Valsalva maneuvers, flashes or floaters and dimming/blacking out of vision are all pertinent when examining a patient with retinal hemorrhages. If no systemic history is available, begin differentiating based on the initial clinical assessment, which should include blood pressure, body mass index and blood sugar.

Optical coherence tomography, fluorescein angiography and fundus photography are all useful tools for determining the location and depth of the hemorrhage. Bloodwork also can be instrumental in determining the correct etiology, as well as any systemic associations (*Table 2*). Comanagement and care coordination with the patient's primary care provider is essential, as is providing detailed education to ensure the patient is an active part of the team.

Ocular manifestations of systemic conditions can present asymmetrically, but most show some laterality. When an asymmetry in retinal hemorrhaging is noted absent ocular trauma, the carotid arteries should be evaluated via a carotid duplex ultrasound, computed tomography angiogram or magnetic resonance angiogram of the carotid arteries to rule out clinically significant stenosis or plaque formation. In some cases of carotid occlusive disease, chronic hypoperfusion to the retina on the ipsilateral side of the blockage of the internal carotid or ophthalmic arteries can lead to an asymmetry in retinopathy.

Monitor or Treat?

Optometrists are tasked with determining whether the ocular findings are isolated or indicative of more widespread systemic disease. That decision will guide the clinician for proper treatment or even a potential referral for subspecialty care. ODs can follow these basic steps when triaging retinal hemorrhage patients:

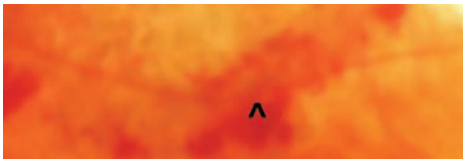

Fig. 5. The depth of this subretinal hemorrhage makes it less bright red than more superficial inner retinal hemorrhages.

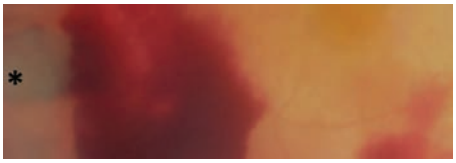

Fig. 6. Sub-RPE hemorrhages are often noted to be dark with well-defined borders, as seen here.

Drs. DeMarco, Mah, Cole and Mazzarella are optometrists in North Carolina.

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1. Take a detailed medical and social history to determine potential correlations to the patient's ocular findings. The patients' known systemic comorbidities, current medications and activities of daily living may give the astute clinician valuable clues to an underlying etiology.

2. Determine whether the retinal hemorrhages are vision threatening based on the location to the fovea and other associated symptoms and ocular findings.

3. Generate a list of differential diagnoses. This will help to determine the need for referral to a retina specialist for further monitoring or treatment. The timing of the referral will depend on the potential for progression of the condition and any potential for visual loss. The differential diagnoses will also help comanagement with the primary care physician for continued coordination of ocular and systemic care with consideration for further testing, such as bloodwork.

4. Educate the patient about their ocular findings and any potential associations to systemic comorbidities. Patient education can be accomplished by means of drawings, software programs, reviewing of ancillary imaging results or other means that allow the patient to appreciate their eye findings in an easily interpreted manner. Convey to the patient the current risk the ocular findings have on their vision, as well as define the risks of progression and potential visual consequences.

5. Discuss a follow-up or referral time and schedule the patient to return to clinic with any new ocular symptoms or changes in vision.

Retinal hemorrhages are often first noted on comprehensive eye examinations during routine care. Often, these are correlated to known systemic or ocular comorbidities. In cases where no known associations can be elicited from the history or determined from clinical evaluation, further workup and comanagement with the primary care doctor may be necessary to determine the underlying etiology. A firm grasp of the classification of retinal hemorrhages, and knowing how to properly triage these patients, will ensure proper patient management. ■

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9th Annual Retina Report

The Bugs Behind Infectious Retinitis

Be prepared to find the etiology behind a patient's inflamed retina.

By Jim Williamson, OD, and Jessica Haynes, OD

Infectious retinitis, while rare, can have dire visual consequences. Many causes exist, including viral, bacterial, tick-borne, parasitic and fungal. Successful management of these patients requires prompt recognition to quickly initiate treatment and refer, though the outcome may still be unfavorable. This discussion of the various etiologies can help you hone your differentials and properly diagnose these patients in a timely manner.

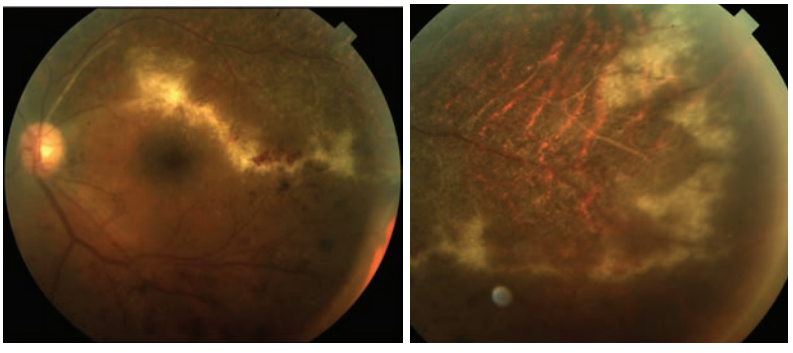

Fig. 1. Along with the "ketchup and cottage cheese" appearance, CMV may advance like a "brush fire" or the tip of an ocean wave, leaving a necrotic retina.

Invasion of the Virus

Necrotizing herpetic retinitis describes a spectrum of infections caused by herpes viruses, mainly varicella zoster virus (VZV), herpes simplex virus (HSV) and cytomegalovirus (CMV).[1,2] These conditions include acute retinal necrosis (ARN), progressive outer retinal necrosis (PORN) and CMV retinopathy.[1,2] While separate conditions, they may represent a continuum whose clinical presentation depends on the patient's immune status.[1] These conditions are quickly progressive and can lead to devastating vision loss, even bilateral blindness.[1,2,5]

ARN. Patients with this condition may present with symptoms of blurry vision, floaters or ocular pain.[4] It often presents with patches of retinal whitening and necrosis in the peripheral retina that extend posteriorly with time.[5] Vitritis, anterior chamber cell, vasculitis and optic nerve involvement are common. Larger areas of necrotic retina increase the risk of retinal detachment (RD).[5-7] Major causes of vision loss include RD, optic atrophy and retinal ischemia.[6]

ARN commonly presents in immunocompetent patients, but may also present in immunocompromised patients such as those with HIV/AIDS.[8] Preceding signs of viral infection, such as cold sores, herpetic corneal disease, meningitis, encephalitis and disseminated shingles are common.[6]

Diagnosis is traditionally made based on clinical examination alone, often using the American Uveitis Society's criteria: (1) One or more foci of discrete necrosis in the peripheral retina, (2) rapid progression without antiviral therapy, (3) arteritis and occlusive vasculitis, and (4) robust vitritis and anterior chamber reaction.[9] Clinicians can also use polymerase chain reaction (PCR)

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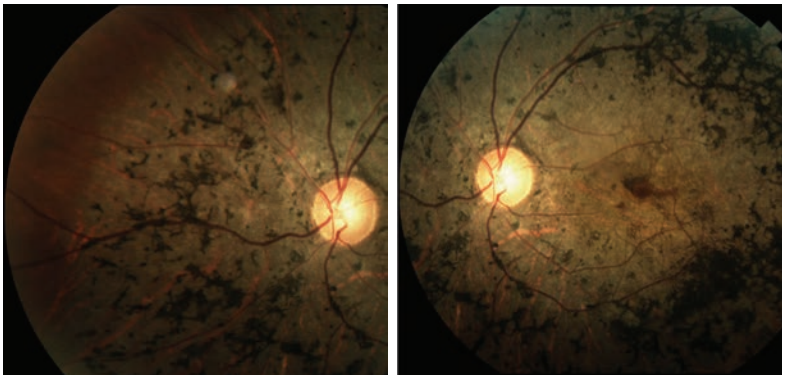


Fig. 2. These images depict the classic presentation of syphilitic retinopathy.

to detect viral DNA and confirm infection—79% to 100% of ARN cases undergoing PCR testing revealed positive results for either VZV or HSV.[8]

PORN. This typically presents in immunocompromised patients, usually in those with HIV/AIDS whose CD4 count is less than 50cells/µl. Additional forms of immunosuppression, such as chemotherapeutic agents, are also risk factors.[4] Patients with PORN often present with complaints of decreased peripheral vision or sudden vision loss. They may have floaters, but typically do not have pain.[4]

The condition presents with multiple regions of yellow-white retinal necrosis in either the posterior pole or peripheral retina that progress rapidly and coalesce. These regions initially affect the outer retina but eventually lead to full-thickness involvement. Hemorrhages may be present along with papillitis; however, a robust inflammatory response with dense vitritis and anterior chamber cell is not common. The necrotic retina becomes atrophic with a propensity to develop multiples holes, ultimately leading to RD in many patients.[4]

CMV. Present in its latent form in about 50% of the general population, this can lead to a severe necrotizing retinitis in patients who are immunocompromised.[12,13] The condition usually presents in patients with CD4 counts of 50cells/L or less.[13] CMV retinitis is the most common opportunistic ocular infection in patients with HIV/AIDS, although highly active antiretroviral therapy has significantly decreased the incidence of CMV retinitis in this population.[13] However, more widespread use of systemic immunosuppressive agents for conditions such as autoimmune disease has also led to an increase in those without HIV/AIDS.[12]

Patients with CMV retinitis often present with blurred vision and floaters but may also be asymptomatic.[14,15] The disease produces lesions of full-thickness retinal necrosis with a granular appearance (*Figure 1*). These lesions often occur along blood vessels and may be associated with significant retinal hemorrhaging, leading to descriptions such as "ketchup and cottage cheese."[14,15] Retinal vasculitis, optic nerve edema, vitritis and anterior uveitis may also be present. RD is common and is a leading cause of vision loss in patients with CMV retinitis.[14]

Diagnosis is chiefly based on clinical presentation.[14,15] Most patients have a previous diagnosis of HIV/AIDS or history of immunosuppression. If not, testing is necessary. Serologic testing for CMV is available, but due to its high presence in the general population, it has little use. PCR testing may help to identify the virus.[14,15]

Using OCT, researchers describe CMV lesions as either full-thickness or cavernous necrosis.[16] In full-thickness necrosis, there was thickening and hyperreflectivity of all retinal layers. The retinal pigment epithelium (RPE) was also thickened with a ruffling of its more anterior border, and the Bruch's membrane was easily visible. In cavernous necrosis, hyperreflectivity in the inner retina with large empty spaces exists within the outer nuclear layer, leaving the RPE intact. OCT also revealed vitreous cell and thickening of the posterior hyaloid.[16]

Beware the Bugs

A bacterial infection can also cause retinal inflammation.

Syphilis. This is caused by *Treponema pallidum*.[17] While ocular complications are uncommon, panuveitis is the most usual finding when they do occur.[17,18] Others include interstitial keratitis, anterior and intermediate uveitis, scleritis, vasculitis, chorioretinitis, serous RDs, optic neuritis and optic nerve gumma, which are solitary inflammatory lesions (*Figure 2*).[17-19]

A few findings are considered distinctive, such as multiple, creamy-white preretinal precipitates in those with panuveitis.[17,19] Involved retina tends to be mildly opacified compared with the dense retinal whitening seen in necrotizing herpetic retinitis.[19] Necrotizing retinitis is a rare complication but can mimic ARN.[19] An infrequent complication, acute syphilitic posterior placoid chorioretinitis, presents with deep oval plaques of retinal whitening.[17,19] When imaged with OCT, these regions have outer retinal alterations including disruption of the ellipsoid zone, external limiting membrane and RPE as well as subretinal fluid.[20] Congenital syphilis can lead to chorioretinitis with a salt and pepper appearance.

Serological testing is necessary for diagnosis and

is divided into treponemal and non-treponemal tests. Treponemal tests include *T. pallidum* hemagglutination assay and fluorescent treponemal antiboday-absorption. These labs remain positive for life and cannot be used to determine disease activity and treatment efficacy, but they have a higher specificity than non-treponemal tests. The latter include venereal disease research laboratory test and rapid plasmin reagin test, both helpful for monitoring disease activity and treatment efficacy.[17,19]

Cat scratch disease (CSD). Caused by *Bartonella henselea*, this is the most common cause of neuroretinitis and is typically benign and self-limited.[21,22] The bacteria is harbored in cats and kittens, and can be acquired in humans through a scratch or bite. However, the bacteria can also be transmitted by dogs, fleas and may even have unknown vectors of transmission.[22]

Systemic symptoms such as lymphadenopathy, fever and malaise typically occur prior to ocular findings, but can be coincident as well.[21,22] Not all patients with ocular complications will report systemic symptoms.[22]

Posterior segment findings, either bilateral or unilateral, are variable.[22] Many reports cite optic disc edema with macular star (neuroretinitis) as the most common finding, while others describe focal areas of either superficial or deep retinal whitening (*Figure 3*).[22,23] Additional findings include optic nerve edema without macular star, vitritis, macular edema, serous detachments, hemorrhage, optic nerve granuloma and angiomatous lesions.[21-25] Presence of macular star may initially be absent and form post-resolution of macular edema.[22]

Fluorescein angiography may reveal optic nerve leakage and hyperfluorescence of retinal lesions.[24,25] Clinicians should demonstrate the extent of vascular nonperfusion in occlusive events to help determine visual prognosis and the likelihood of secondary complications such as neovascularization.[24] Peripapillary telangiectasia and angiomatous lesions may be present.[23,24,26] OCT can be a helpful tool to document and monitor macular edema, serous detachments and optic nerve edema.[26]

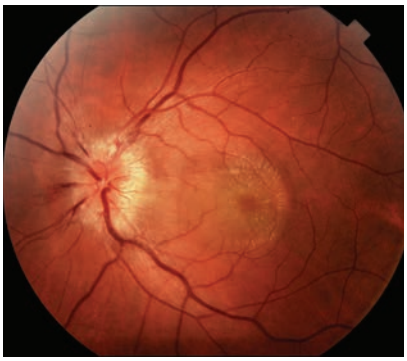


Fig. 3. Neuroretinitis can present with a macular star formation, as seen here.

Ocular diagnosis is based on fundus findings, but readily available serological testing for *Bartonella henselea*

should be obtained to help confirm the diagnosis.

Tuberculosis. This is caused by *Mycobacterium turberculosis* and can have ocular involvement, even in isolation without pulmonary manifestations.[32] All forms of uveitis are possible as well.[32]

The most common posterior finding is multiple choroidal tubercles, which look like deep, ill-defined, round, gray or yellowish lesions, possibly having overlying serous detachments.[32,33] Larger choroidal granulomas may be present, often mimicking choroidal tumors.[32,33] Choroidal involvement can also occur in the form of serpiginous-like choroiditis. This appears as multifocal plaque-like lesions in the peripapillary region that spread circumferentially. Additional findings can include retinal vasculitis (typically affecting veins more than arteries), neuroretinitis and endophthalmitis.[32,33]

Significant challenges exist in the diagnosis of ocular tuberculosis. Cultures and PCR testing have low yields, and biopsy is often impossible.[34] Frequently, diagnosis relies on the clinical picture, tuberculin skin test, chest X-ray and, sometimes, response to treatment.[34] The utility of interferon-γ release assays, such as the Qauntiferon-TB Gold (Qiagen) test, is still uncertain.[35]

Out of the Woods

Tick-borne diseases (TBDs) are often underdiagnosed, for several reasons. First, more than 75% of patients don't remember having a tick bite.[36] Second, signs such as arthralgia, skin involvement and neurological changes may be consistent with those of autoimmune diseases.[37] Patients with TBDs may first present to their eye care provider due to ocular symptoms.

Lyme disease. The most common TBD in the United States, this disease had 27,000 confirmed cases in 2013.[38] *Borrelia burgdorferi* spirochete is the causative agent in the United States, while *B. garinii* and *B. afzelii* prevail in Europe.[40] Transmission occurs via the deer tick in the east and the Western black-legged tick in the west.[41] Fortunately, the risk of contracting *Borrelia* from an infected tick is between 1% and 3%.[39]

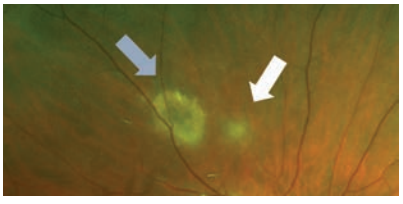
In the early stage of Lyme disease, up to 80% of patients will have erythema migrans, or a bulls-eye rash roughly eight days post-infection.[37,41] A flu-like illness and lymphadenopathy may accompany the rash, and ocular involvement, if any, is confined to follicular conjunctivitis and mild photophobia.[40]

Stage two (three to 10 weeks after inoculation) has the most ophthalmic changes. Ocular manifestations include optic neuritis, uveitis, choroiditis, venous occlusion, cotton wool spot and neurological pupil abnormalities.[36,40] Keratitis is the most common finding in stage three disease, which occurs months to years later.

In patients suspected of exposure, an enzyme immunoassay should be ordered, and if positive, an immunoblot test. Because Lyme testing may be negative within the first few weeks of the disease, serological testing can be deferred if the clinical picture correlates with the disease and the patient portrays the bulls-eye rash.[36]

Rocky Mountain spotted fever (RMSF). This is a life-threatening disease caused by *Rickettsia*. The primary vector in most of the United States is the American dog tick. However, in the Rocky Mountain area and Canada, it is the Rocky Mountain wood tick. The brown dog tick, which mainly affects Mexico, has been implicated in eastern Arizona.[42] The gram-negative bacteria targets the lining of small and medium-sized blood vessels, which leads to the triad of "Rickettsial vasculitis"—vascular inflammation, blood vessel damage and altered vascular permeability.[37] Ocular findings include retinal hemorrhage (about 50% of RMSF have thrombocytopenia), retinal artery or vein occlusion, vascular sheathing, optic neuritis and neuroretinitis.[36,40]

Signs and symptoms include fever, nausea, headache, vomiting and possibly severe abdominal pain—especially in children—which may lead to an incorrect gastroenteritis diagnosis.[43] The hallmark sign is the spotty rash, which starts around the wrist and ankles. Diagnosis relies more on clinical findings, as laboratory testing, such as a skin biopsy, is difficult to yield positive results.[37] Also, given the severity of the disease, treatment should be initiated regardless of a negative test.



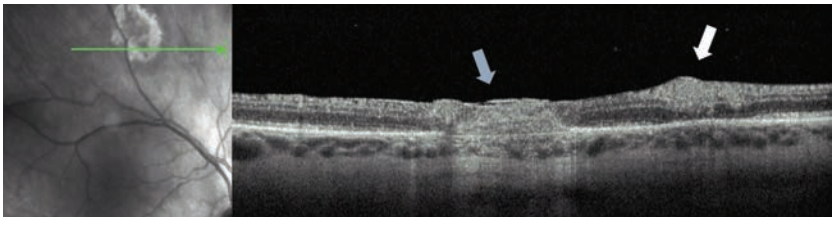


Fig. 4. This resolving toxoplasmosis lesion (blue arrow) has a new region of active infection (white arrow). Note that the older lesions involve all retinal layers, whereas the early ones only affect the inner retina.

Unwilling Hosts

Other retinitis cases can be linked to

any number of parasites:

Toxoplasmosis is the leading cause of posterior uveitis, and is due to *Toxoplasma gondii*—a ubiquitous human parasite.[44] Infection occurs by ingestion of contaminated soil or tissue cysts in undercooked meat (pig and sheep).[45,46] Ocular findings include retinochoroiditis, retinal vasculitis, branch retinal vein occlusion and anterior and intermediate uveitis.[45] The complaint of "headlights in the fog" is the common presenting sign. A complication of infection is increased intraocular pressure, which may differentiate this process from other possible causes of retinochoroiditis.[47]

Acute infection leads to a superficial retinitis and vitritis. OCT imaging may show a thickened and hyperreflective inner retina, edema, subretinal fluid, vitreous cell and thickening of the posterior hyaloid. The condition can progress into deep layers, leading to chorioretinal scarring (*Figure 4*).

Diffuse unilateral subacute neuroretinitis (DUSN) results from nematode infestation, presumably in the subretinal space, and mostly afflicts healthy children and young adults.[48] DUSN should always be suspected when a patient of this demographic presents with unilateral vitritis, retinal vasculitis, optic neuritis and outer retinal/inner choroid multifocal lesions—the early stage of the disease.[49] Late-stage changes include atrophy of the retinal nerve fiber layer, inner and outer retina and optic nerve.[48] Unfortunately, clinical visualization of the worm occurs in only 25% to 40% of patients.[50] However, researchers have seen the worm's location using OCT angiography.[50]

Toxocariasis, caused by ingesting *Toxocara* larvae from foods soiled with dog or cat feces, may lead to a unilateral ocular presentation of peripheral or posterior granuloma or panuveitis with no distinct nodular lesion.[51] Children are especially at risk for exposure given their hygiene habits and the contamination rates (as high as 40%) of play areas.[51] Only a few adult cases have been reported.[52]

Fear the Fungus

Given the choroid's high blood flow, most fungal lesions begin as a choroiditis.[54] Fortunately, research of adult and pediatric patients with fungemia shows the eye has less than 1% of fungal dissemination, possibly due to prompt initiation of antifungal treatment in clinically suspicious cases, even before culture results.[55]

Candida. This is the most common healthcare-associated bloodstream infection, with at least 50% of cases occurring in the intensive care setting.[56] Community-acquired risk factors include an immunocompromised

state, intravenous (IV) drug use, diabetes, catheter placement and broad-spectrum antibiotic use.[56] Direct inoculation from ocular trauma or surgery can occur.[57] Ocular manifestations include white fluffy chorioretinal lesions, focal vitritis and iridocyclitis.

Researchers described two patterns of OCT findings in *Candida* endophthalmitis—chorioretinal and intraretinal—which they believe represent the site of ocular invasion. The chorioretinal pattern includes the raincloud sign (preretinal hyperreflective dense masses with a strong shadowing effect) and the sawtooth-shaped sign (vertically oriented hyperreflectivity bands adjacent to the lesion). The less indolent intraretinal pattern (hyperreflective sphere) spared structures beyond the inner nuclear layer.[58]

Aspergillus. The vast majority of exogenous cases are molds (87%), and the most common agent is *Aspergillus*.[59] It grows in soil and decaying vegetation with airborne spores, which place the immunocompromised, those with pulmonary disease and IV drug use at risk. Macular infiltration is common, and patients often present with a painful decrease in visual acuity. Other signs include vascular occlusion, retinal necrosis and retinal hemorrhages.[57]

Ocular histoplasmosis syndrome (OHS). A mild and transient pulmonary infection in otherwise healthy people, this is caused by *Histoplasma capsulatum* or *H. duboisii*.[60] The Mississippi and Ohio river valleys form the "histo belt." Clinically, OHS appears as peripapillary atrophy (PPA), "histo spots" or "punched out" chorioretinal scars in the macula and midperiphery and choroidal neovascularization (CNV) or its disciform scar sequelae (*Figure 5*).[61]

Diagnosis of OHS is based on observation, as skin testing may induce flare-ups. Two of the three ocular findings listed above should be met.[61] Smoking is a significant risk factor for CNV development.[62] In addition, one study found that the presence of PPA and macular histoplasmosis spots invoked a respective 4% and 25% risk of vision loss to the fellow eye.[62]

Retinitis, although a rare condition, highlights the importance of a thorough funduscopic examination, even in asymptomatic patients. With myriad etiologies on the differential, knowing the classic presentation of each is crucial to narrowing down the diagnosis and initiating treatment as early as possible. Management depends on the entity and can range from in-office monitoring to an infectious disease or retina specialist referral. Despite prompt diagnosis, positive outcomes are not always possible. For those cases, ODs should employ low vision tactics to enhance patients' remaining vision and help them maintain functional independence. ■

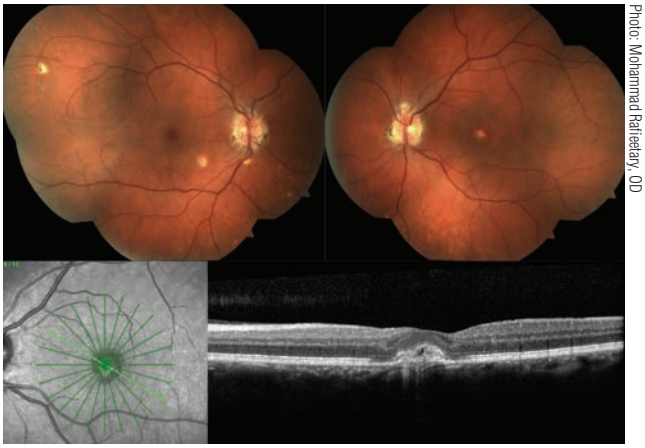


Photo: Mohammad Rafieetary, OD

Fig. 5. This patient presented with with the OHS triad: PPA, multiple punched out lesions and early development of CNV.

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Dr. Haynes is a clinical optometric fellow at Charles Retina Institute in Germantown, Tenn.

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PEER-REVIEWED PRA ARTICLE

The Larry Alexander Resident Case Report Contest:

Cloudy with a Chance of Retinopathy

Clinical findings and management of endophthalmitis.

By Varun Khedekar, OD, and Candace Cordero, OD

In 2017, the Optometric Retina Society (ORS) chose to honor the legacy of Larry Alexander, OD, by launching the Larry Alexander Case Report Contest for optometric residents. The contest was made possible by support from Optovue. Submissions were reviewed independently by the ORS board. The case below is this year's winner.

A patient who presents to the eye clinic with an acute red eye, decreased vision and concurrent eye pain can represent a vast array of pathologies. Our diagnostic challenge as clinicians is to distinguish the everyday dry eye syndrome from the rare ophthalmic emergencies that can have sight-threatening complications such as endophthalmitis.

Endophthalmitis is a rapidly progressing purulent inflammation of the aqueous and vitreous that usually stems from an infection.[1]

This report outlines a rare case of endogenous endophthalmitis, in an immunocompromised male patient, that was secondary to endocarditis with methicillin-resistant *Staphylococcus aureus* (MRSA) sepsis.

It also details common clinical findings, disease process, microorganism profile and proper treatment strategies for the different forms of endophthalmitis.

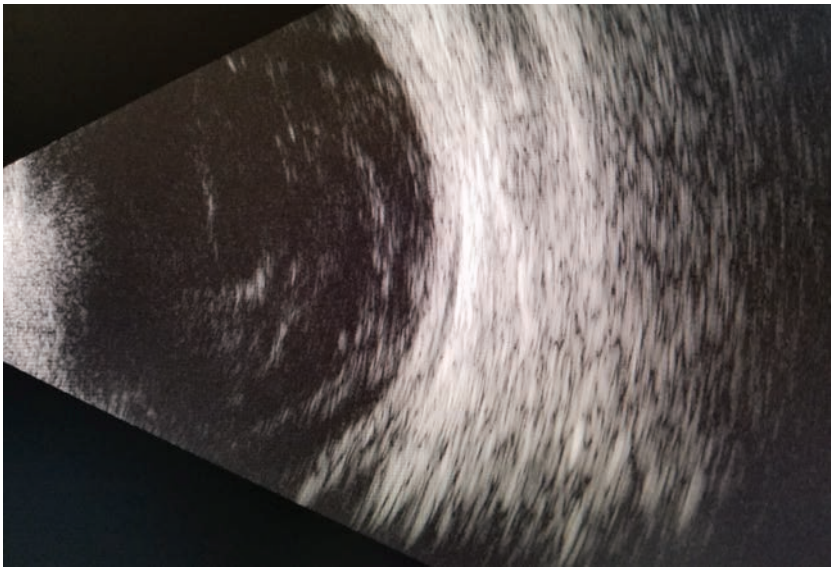

Fig. 1. A B-scan of the right eye reveals vitritis with no retinal detachment or mass.

Presentation

A 61-year-old Caucasian male presented emergently for examination complaining of sudden vision loss in his right eye over the last two days. He rated his pain as a "five" on a scale of one to 10 and reported seeing "white swiggly lines" that had worsened since onset. He denied other visual symptoms. His last examination five months prior was unremarkable. His ocular history, however, was remarkable for partial lower left excision for basal cell carcinoma and previous alpha-2a interferon treatment for corneal intra-epithelial neoplasm of the right eye. The patient's medical history was positive for Type II diabetes mellitus and a history of alcohol, cocaine and intravenous drug use. The patient's family history was non-contributory. He had no known drug allergies and was oriented to time, place and person.

Uncorrected visual acuity (VA) was counting fingers at one foot in his right eye and 20/25-2 in the left. The right pupil was minimally reactive with a trace afferent pupillary

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defect. The left pupil was round and reactive to light. Extraocular muscles were unrestricted in all gazes without pain or diplopia. Confrontation fields were constricted in all inferior quadrants in the right eye and were full in the left eye. Cover test was orthophoric at distance and near. On red-cap desaturation testing, the patient reported "blue" cap in the right eye, 100% red in the left. Pseudo-isochromatic color plates were attempted but not completed due to poor patient cooperation and fatigue.

Slit lamp biomicroscopy revealed normal external adnexae in both eyes. The left anterior ocular structures were normal unless otherwise stated. The right bulbar conjunctiva had trace diffuse injection. The right cornea was remarkable for grade 1 Descemet's folds centrally with diffuse microcystic edema. The right anterior chamber had grade 3-4 cells with grade 1+ flare and a 1.5mm hypopyon inferiorly. Both irides were flat and intact. The anterior chamber depth was normal. Goldman applanation tonometry measured 12mm Hg OU at 3:10pm.

The patient was dilated using one drop of 1% mydriacyl and 2.5% phenylephrine OU. Evaluation of the posterior segment with a 90D lens revealed grade 2+ nuclear sclerosis of the lens of both eyes. The vitreous media of the right eye had a dense vitritis that prevented further posterior views of the retina. The B-scan ultrasound revealed a dense vitritis and an absence of a retinal detachment or mass (*Figure 1*).

The left eye fundus findings included: clear vitreal media, a healthy optic nerve with a 0.30 H/V cup-to-disc ratio and distinct margins. There were dot hemorrhages with elevated patches of choroidal whitening along the superior and inferior temporal arcades. The

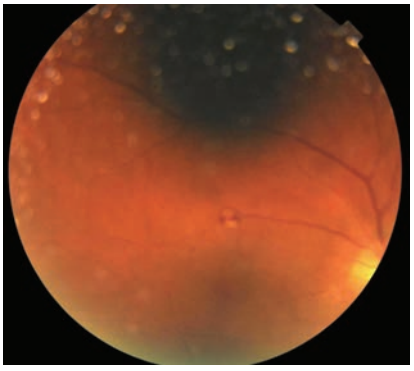

Fig. 2. Presence of Roth spot along superior temporal arcade of the right eye.

macula was flat and even with two Roth spots inferior to the fovea. The peripheral retina was flat without pathology.

We considered differential diagnoses of panuveitis, chorioretinitis (infectious/inflammatory), malignant neoplasms, intraocular foreign bodies (IOFBs) and endogenous endophthalmitis. After a retinal consultation, the patient was diagnosed with endogenous endophthalmitis.

Intravitreal injections of 0.1ml of 2.25mg/0.1ml ceftazidime and 0.1ml of 5ug/ml amphotericin B were performed without complications. Bacterial and fungal blood cultures were ordered along with an infectious disease consultation to determine the primary source of infection. The patient was started on intravenous vancomycin, Zosyn (piperacillin and tazobactam, Pfizer) fluconazole and clindamycin and was scheduled to return to the eye clinic the following morning for re-evaluation.

Day one follow-up. The patient presented without new visual complaints, stable acuity and stable exam findings in both eyes. The patient received an intravitreal injection of 0.1ml of 1mg/0.1ml vancomycin in his right eye. He was also scheduled to undergo pars plana vitrectomy (PPV) in the right eye in four days if no improvement was found at the follow-up in two days. Human immunodeficiency virus (HIV) labs were also ordered by the infectious disease department.

Day three follow-up. The patient returned with significant improvement in his right eye's vision. The nurse present at the exam revealed the patient had fever spikes over the previous 48 hours. Uncorrected distance VA was 20/200 OD and 20/25-1 OS. The right bulbar conjunctiva still had trace diffuse injection. The right cornea showed trace Descemet's folds centrally and temporally with no sodium fluorescein staining. The right anterior chamber had grade 3 cells with no flare and no hypopyon.

Posterior exam in his right eye revealed grade 1 vitritis with a flat and even macula and 0.45 H/V cup-to-disc ratio of the optic nerve with distinct margins. His right eye's posterior pole had scattered intraretinal hemorrhages with scattered white choroidal lesions and Roth spots (*Figure 2*). The posterior exam of the left eye was stable from the initial visit. The blood cultures returned positive for MRSA, and bloodwork results showed a newly diagnosed HIV positive status. Given improvement in his right eye acuity and decreased inflammatory reaction, the vitrectomy was not performed.

Day five follow-up. The patient reported progressive improvement in the right eye's vision. Further work-up by the infectious disease department and primary care physician revealed a positive diagnosis for endocarditis with MRSA sepsis. Uncorrected distance VA was 20/100 OD and 20/30 OS. The bulbar conjunctiva was white and quiet in both eyes. The right cornea showed trace non-granulomatous keratic precipitates inferior with no sodium fluorescein staining. The right anterior chamber had grade 3 cells with no flare and no hypopyon.

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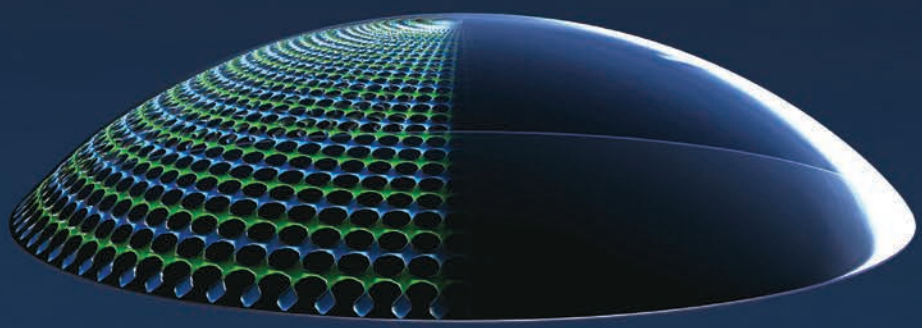

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Dilated fundus examination of the right eye revealed grade 1 vitritis with otherwise stable findings to the previous dilated fundus exam. Given improvement but persistent inflammatory reaction in the right eye, the patient was started on one drop prednisolone acetate 1% TID OD.

Day eight follow-up. The patient returned to clinic after another three days with improvement in his right eye's vision. Uncorrected distance VA was 20/80 OD and 20/25 OS. The bulbar conjunctiva was white and quiet in both eyes. The right cornea showed resolving trace non-granulomatous keratic precipitates inferior with no sodium fluorescein staining. The right anterior chamber had grade 2 cells with no flare and no hypopyon.

Dilated fundus exam in the right eye revealed trace vitritis with resolving intraretinal hemorrhages, white choroidal lesions and Roth spots. The patient continued one drop prednisolone acetate TID OD. The patient was monitored closely with subsequent follow-up. At last exam his best-corrected VA at distance was 20/25+2 OD and 20/20-1 OS, the anterior chamber was deep and quiet with no cells or flare, no vitritis and rare intraretinal hemorrhages with Roth spots. The patient is currently being treated as in-patient by cardiology for endocarditis.

Discussion

Endophthalmitis, a rapidly progressing inflammatory disorder secondary to infection of the vitreal cavity, can present from an exogenous or endogenous source. Exogenous endophthalmitis can arise from acute or chronic postoperative blebitis, trauma or intravitreal injection.

Postoperative endophthalmitis accounts for approximately 70% of exogenous cases.[1] Acute cases generally present within the first week. Patients present with severely decreased visual acuity, eye pain, eyelid swelling, mucoid discharge and hyperemia. Examination with biomicroscopy may reveal corneal edema, diffuse bulbar conjunctival injection, hypopyon, dense vitritis and possible retinitis. Of note, approximately 25% of patients will not experience eye pain.[1] Acute postoperative cases are most commonly caused by gram-positive, coagulase negative *micrococci*. Chronic cases, which are differentiated from acute cases by an onset of greater than a six-week duration postoperatively, are commonly caused by slower growing gram-negative bacteria or fungi. One study looked at the microbiologic spectrum of acute postoperative bacterial endophthalmitis and revealed that 94.2% of patients showed a gram-positive bacteria stain while only 6.5% showed a gram-negative bacteria stain.[2,3] Of the gram-positive strains, *Staphylococcus aureus* presented with the highest prevalence (9%), followed by *Streptococcus* species at 2.2%.[2]

The presence of a bleb after trabeculectomy surgery presents a continuing risk for infection. Breakdown of the bleb wall allows for microbe infiltration from the tear film and surrounding ocular structures. Early leakage (within three months of surgery) is often secondary to incomplete conjunctival closure or trauma. Investigators suspect that late onset leakage (leakage after three months of surgery) is associated with anti-metabolite use (i.e., mitomycin-C or 5-fluorouracil) during trabeculectomy.[4]

A 2007 retrospective study found a 15% risk of bleb leakage within five years of surgery associated with the use of anti-metabolite.[4] Blebitis presents with sudden onset pain, photophobia, mucoid discharge, hyperemia and possible hypopyon. With its extension into the vitreous, bleb-associated endophthalmitis (BAE) has similar clinical findings to blebitis but with exaggerated signs and symptoms. Due to bleb leakage being the primary source for infection, the most common microbe associated with BAE is *Staphylococcus epidermidis*, followed closely by the *Streptococcus* family.[6]

Even with aggressive early management, BAE cases have a poor visual prognosis. Retrospective studies show 94% of cases result in 20/200 or less visual acuity with approximately 35% resulting in no light perception.[6,7]

A penetrating globe injury can operate as an access point for opportunistic microbes. The incidence of endophthalmitis after a history of penetrating injury without an IOFB is approximately 12% and jumps to 25% with the presence of an IOFB.[8,9] Patients will present with the typical endophthalmitis signs and symptoms: conjunctival injection, purulent discharge, moderate anterior chamber reaction, lid edema, light sensitivity, vitritis and possible hypopyon. Eye pain can range from mild photophobia to severe pain, depending on the type of microbe, making the final diagnosis challenging. Most cases of post-traumatic endophthalmitis are caused by gram-positive bacterial organisms, most often the *Bacillus* species, especially in the presence of an IOFB or a non-sterile environment.[10]

While the advent of intravitreal injections, specifically anti-vascular endothelial growth factor (VEGF) injections, has reformed treatment for proliferative retinal diseases such as diabetic retinopathy and macular degeneration, it also presents an avenue for microbial infiltration. A seven-year retrospective study examined 199 cases of endophthalmitis and found that anti-VEGF injections

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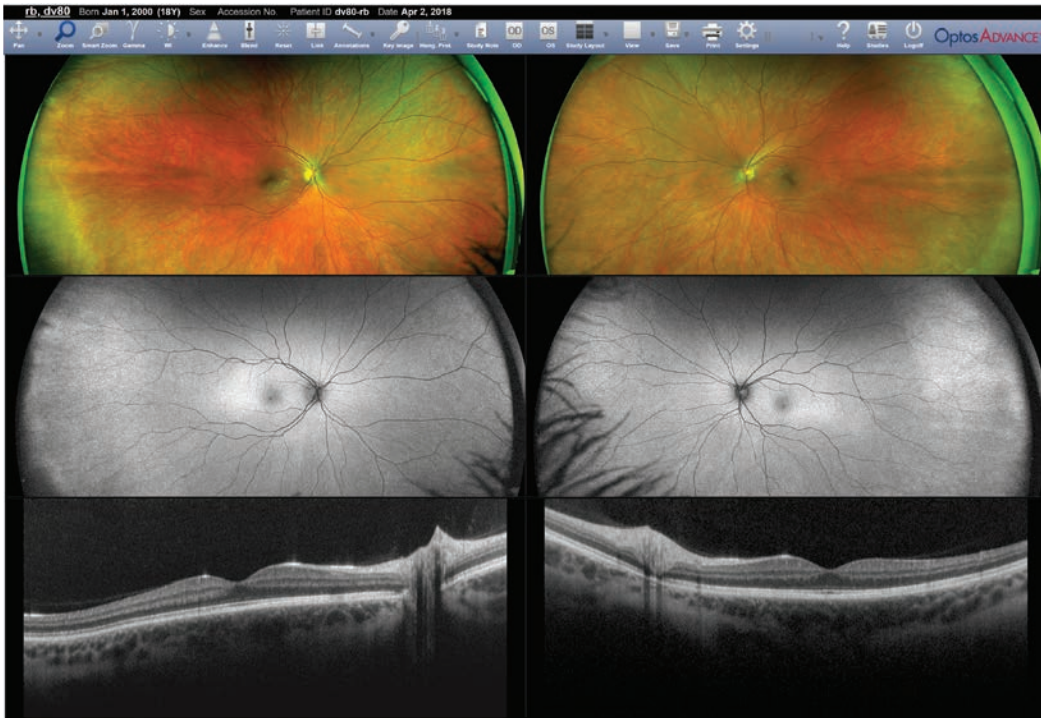

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represented 8.5% of all cases.[11] Furthermore, analysis of vitreal tap from these cases found the most common culprit to be of the coagulase-negative *Staphylococcus* family (50% of cases), similar to postoperative endophthalmitis.[2,11]

Endogenous endophthalmitis accounts for only 2% to 8% of endophthalmitis cases, but is still sight-threatening and must be promptly diagnosed and swiftly managed.[12] In endogenous endophthalmitis, the eye is the secondary site of infection, and a full systemic work-up is warranted to find the source of infection. In 44% of cases, no causative source is identified.[13] Risk factors include immunocompromised states, recent surgery, pregnancy, endocarditis, intravenous drug use, indwelling catheters and dental procedures.[14] The infectious agent travels from its original source via the bloodstream where it then invades the vasculature of the posterior segment and eventually spreads to the vitreous through rupture of a septic embolus.[15]

Endogenous endophthalmitis signs and symptoms include: red eye, eye pain, reduced visual acuity, photophobia, corneal edema, conjunctival injection, cells and flare, hypopyon and vitritis. Prospective studies from the Americas, Europe, Asia and Australia that performed microbiological analysis found an interesting variation in results. Anterior chamber and vitreal samples that were analyzed through gram staining and culture positivity found gram-positive organisms, most often *Staphylococci* and *Streptococci*, to be culprits in the Americas and Europe. In Asia and Europe, gram negative microbes and fungi tended to dominate.[16,17]

Endogenous endophthalmitis requires rapid management. Necessary ancillary workup should include B-scan ultrasound—especially with the presence of significant media opacification secondary to vitritis—to rule out retinal or choroidal detachments, dislocated lens and the presence of IOFBs. Determining the specific etiology is accomplished with vitreal aspiration or diagnostic PPV, followed by culturing and histology. Vitrectomy has a 92% diagnostic rate of culturing compared with vitreal aspiration (44%).[18]

Immediate PPV is often performed for several reasons: direct reduction of infectious agents and toxins, clearing of media opacities, better culturing sample and faster clearance of intravitreal antibiotics.[19,20] Another emerging technique for identification of endogenous endophthalmitis after procuring vitreous or aqueous samples is real-time polymerase chain reaction (RT-PCR). This procedure has increased sensitivity and specificity compared with culturing, rapid test time (90 minutes) and less worry about contaminated cultures.[21,22] However, culturing is still recommended as part of the management plan because RT-PCR does not highlight antibiotic susceptibility.[23] Given that endogenous endophthalmitis is a result of systemic infection, it is imperative to perform a blood culture with sterile precautions to find the source.

After obtaining cultures through PPV or vitreal aspiration, empirical intravitreal antibiotic and antifungals are typically initiated. Vancomycin (1.0mg/0.1ml) and ceftazidime (2.25mg/0.1ml) are often the antibiotics of choice for broad-spectrum coverage (gram + and gram -); however, amikacin (400ug/0.1ml) may also be used in penicillin allergic patients.[1,23] The use of intravitreal antibiotics may come with complications such as corneal opacification and retinal toxicity. Aminoglycosides, most notably gentamicin, may cause a macular infarct.[18,24] In addition to antibiotics, intravitreal amphotericin-B (5-10ug/0.1ml) or voriconazole (100-200ug/0.1ml) are the recommended antifungals of choice due to their broad-spectrum coverage. They carry the same risk of retinal toxicity as antibiotics.[18,24]

Systemic antibiotics are usually not the treatment of choice for exogenous endophthalmitis due to their poor penetration through the blood-retinal barrier and time needed to be processed systemically. However, in the case of endogenous endophthalmitis, systemic antibiotics and antifungals must be initiated to treat the underlying source of infection and continued for four to six weeks, depending on the patient's individual health status.

The use of corticosteroids to help reduce intraocular inflammation is controversial. A 2003 study found better visual outcomes with additional treatment with intraocular steroids.[25] However, conflicting studies found no improvement in visual outcome with the use of steroids.[26] Current corticosteroid use is left to the clinician's judgment.

This case demonstrates the importance of patient history, awareness of clinical findings and an inter-disciplinary approach for timely intervention of endophthalmitis. Regardless of its source, endophthalmitis can progress rapidly with longstanding ocular and systemic ramifications. However, with proper examination, ancillary testing and treatment the prognosis for patients is favorable. ■

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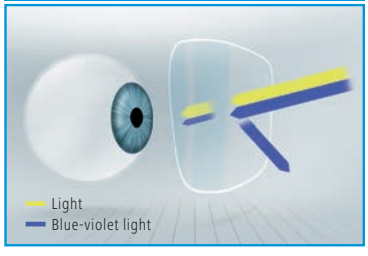

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9th Annual Retina Report

WARDING OFF THE BLUES

Blue light has its pros and cons. Here's how to help your patients manage it for the best systemic and ocular outcomes. **By Bill Hefner, OD, MEd**

There is no arguing that today's patients are inundated with more screen time than ever before. According to a 2015 survey, 60% of American adults are on their digital devices more than six hours per day, with 28% of those individuals exceeding 10 hours per day.[1]

Given the increasing accessibility of cell phones, tablets, computers and more for both education and recreation, we as healthcare providers have to be prepared for a rising tide of significant unintended consequences we don't yet fully understand.[2] In fact, there is growing concern that our vision and health are adversely affected by our inability to unplug.[3-5] One particular area of increasing interest is the effects of short wavelength blue light, or high energy visible blue light (HEV). As researchers and vision scientists continue their efforts to identify and quantify the effects of prolonged exposure to HEV emissions from digital devices, including whether or not smart phones, tablets and computers actually give off enough HEV to trigger damage to our visual systems and a decline in our general health, we owe it to our patients to become knowledgable about HEV.

A Light Primer

Before evaluating the effects of HEV, ODs must first understand what it is and where it comes from. Found in the 380nm to 500nm range, HEV comprises the violet to violet blue end of the light spectrum—and it's everywhere. And while much of the focus is on HEV emissions from digital devices, we can't forget about the more energy efficient, higher-output ambient lighting systems that, unlike their incandescent predecessors, pump out significantly more HEV. For example, nearly 35% of cool white light-emitting diode (LED) emissions are in the

Release Date: May 16, 2018
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Goal Statement: With growing concern that our vision and health are adversely affected by our inability to unplug, it's more important than ever to understand the effects of blue light. This article describes what blue light is, where it comes from and its effects on patients' systemic and ocular health. It also shares ideas on how optometrists can help patients incorporate risk mitigation strategies to ward off its negative effects.

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HEV range, and roughly 26% of compact fluorescent light. In comparison, incandescent bulbs put out less than 12% of their light in the HEV range.[6] Artificial light notwithstanding, we should remember that the majority of our HEV exposure comes from the sun, with 25% to 30% of its spectral emissions falling within the HEV range.[1]

While our understanding of the effects of ultraviolet (UV, 380nm and below) and infrared (IR, 780nm and above) light is relatively well developed, we don't possess a similar grasp of HEV. Luckily, HEV studies continue to yield additional insights into everything from the modulation of the circadian rhythm and improving cognition and memory to HEV's role in countering myopia progression, as a causative factor in the development of symptoms associated with digital vision syndrome (DVS) and in retinal pigment epithelial (RPE) and photoreceptor cell death.[7-13]

Based on current research, we can divide the effects of HEV into three broad categories: (1) circadian rhythm modulation with associated impacts on systemic health, (2) ocular effects and (3) medical effects leveraged in the treatment of certain disease processes such as jaundice and dermatological conditions.

This last category is perhaps most well known and understood, as HEV is a mainstay in the treatment of many dermatological conditions such as acne, psoriasis and atopic dermatitis. It also helps to promote wound healing through its antimicrobial and anti-inflammatory side effects.[14]

What follows is a brief synopsis on the effects of HEV on our patients' systemic and ocular health and how we can help patients incorporate risk mitigation strategies to ward off the negative effects of blue light while maintaining its benefits.

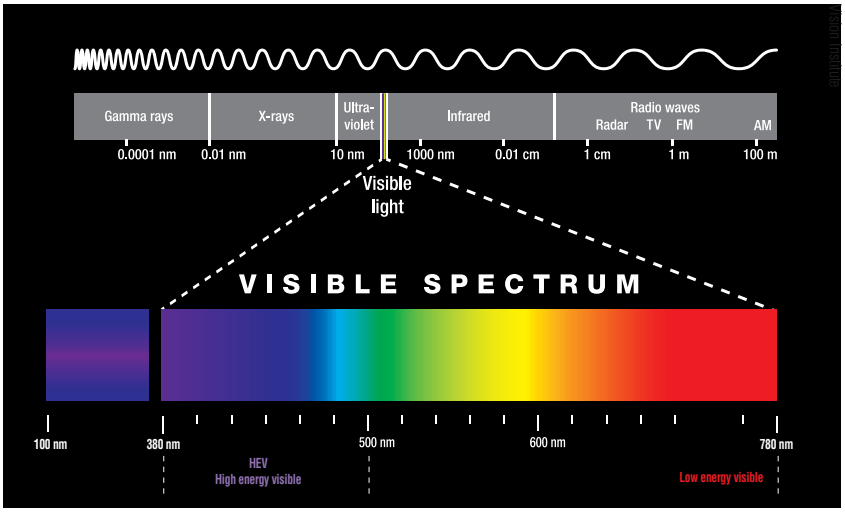


Image: Adapted with permission from Essilor and Paris Vision Institute

As part of the visible spectrum of light, HEV exposure is inevitable.

Get Into the Rhythm

Many factors affect the human sleep cycle. HEV exposure is a crucial component that has direct relevance to us because of its influence through the visual system and the specialized cells that regulate our circadian rhythm. These intrinsically photosensitive retinal ganglion cells (ipRGCs) have a peak spectral sensitivity between 444nm and 486nm—which, coincidentally enough, is right at the upper end of the HEV spectral range.[7] Unlike its photoreceptor siblings, the rods and the cones that undergo hyperpolarization of their cell membranes when stimulated by the wavelength of light corresponding to their chromophores, ipRGCs will *depolarize*, which ultimately inhibits the release of melatonin from the pineal gland.[7]

Researchers have studied the possible role of ipRGCs on photoentrainment for decades, but with the exponential rise of digital devices in everyday life, interest has spiked. Digital devices aren't inherently bad; the problem is their LED light sources have higher outputs in the HEV range (roughly 35%) and white light is actually a blue light (peak emission near 450nm) with a yellow phosphor (peak emission around 580nm)

Table 1. Popular e-Readers and Their Peak Spectral Emissions[15]

Device	Size (in)	Spectral Peak (nm)
Book	n/a	612
iPad	9.7	452
iPhone	3.5	452
Kindle	6	612
Kindle Fire	7	448
Nook Color	7	448

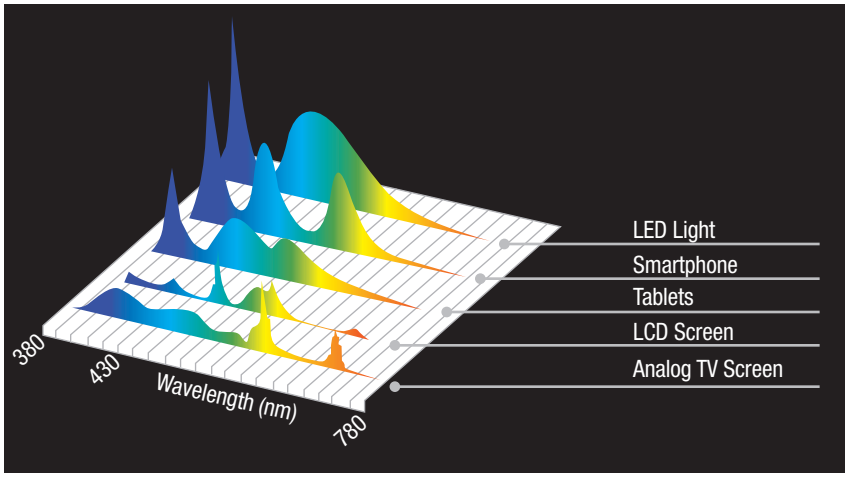
Kids These Days

More alarming than the number of adults on digital devices is the impact on today's youth.

In a 2014 survey, 85% of parents reported their children are using a digital device more than four hours per day—a number expected to skyrocket with the expansion of "electronic classrooms" (at-home learning) and the incorporation of laptops and tablets into traditional learning environments.[2]

Educational activities that historically employed chalkboards and overhead projectors are now being supplanted by lesson plans delivered in the asynchronous learning world of BlackBoard, Schoology and similar online learing platforms. While these highlight slick animations and graphics with easy-to-manipulate "plug and play" topical instructions that leverage school-provided tablets and computers, they also expose students to more blue light than ever before.

Image: Adapted with permission from PFO Global



Newer technologies such as LEDs and smartphones emit far more blue light than older technologies such as analog TV screens.

that becomes less effective over time.[15] In essence, HEV emissions from digital devices that use white LED will actually increase over time.

In 2015, research into the impact of e-readers (e.g., the Apple iPad with a peak wavelength emission of 452nm) on circadian disruption compared with traditional books (peak of 612nm) provided telling results (*Table 1*). Subjects read from either an e-reader or a book for approximately four hours immediately prior to going to bed, five nights in a row. The authors found the e-reader group had a lag in the onset of sleep by about 10 minutes, a REM sleep period that was shorter by approximately 10 minutes and was subjectively more tired the next day. The most significant finding, however, was the delayed increase of melatonin by nearly two hours.[16]

While this study could not say whether the findings were due to the light's wavelength or the intensity of the source itself, a 2017 study confirmed wavelength as the culprit. In this study, subjects were exposed to light sources at either 80lux or 350lux and the light spectrum of 460nm or 620nm for two hours before bed. Each night, researchers collected multiple urine and oral temperature samples and evaluated melatonin levels. While the intensity of light had a negligible effect on sleep quality and melatonin production, the shorter wavelength light had a statistically significant impact on both quality of sleep and melatonin secretion.[17]

In addition to documenting delayed sleep and increased next-day fatigue, several studies implicate melatonin suppression via HEV-related device exposure in increased levels of insulin resistance, elevated blood pressure, seasonal affective disorder and even certain types of cancers.[18-21] One study found subjects exposed to light boxes with a peak wavelength of 468 +/-8nm for 1.5 hours upon waking and 1.5 hours before bed had increased levels of insulin resistance.[18] The evening HEV exposure, in particular, led to higher peak glucose production and diminished sleepiness compared with the morning exposure or no exposure to HEV.[18]

While melatonin plays a vital role in our sleep regulation, blood glucose levels and blood pressure, it's also a powerful antioxidant with more than just a passing association with certain types of cancer, specifically those related to hormone production such as breast and ovarian. Epidemiological studies demonstrate the association between exposure to light at night (LAN)/decreased melatonin levels and cancer.[19] The prospective, longitudinal Nurses' Health Study II (NHSII), which began in 1989 and now boasts more than 116,000 subjects, has been surveying participants every two years in an effort to gather various health data points as the cohort ages. According to one of their recent releases, women living in areas with higher levels of ambient LAN suffer a higher incidence of breast

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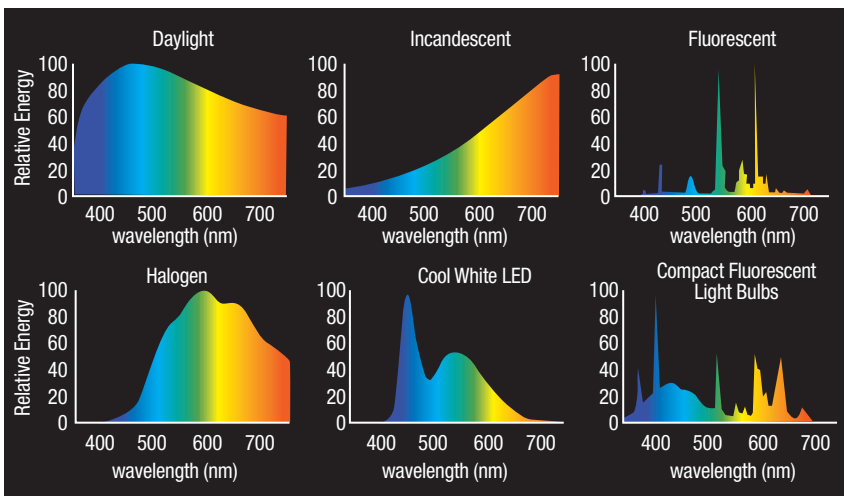


Every light source emits some amount of HEV, and choosing the right lighting can make a big difference in a patient's daily exposure.

cancer, particularly in premenopausal women with a history of smoking—even after accounting for other risk factors.[20] Previous NHSII research also demonstrates creatine-corrected melatonin concentrations were lower in women with >15 pack-years of smoking than in individuals who had never smoked—a significant finding, considering lower melatonin levels are associated with an increased incidence of breast cancer.[21] All of this data has led researchers to believe LAN exposure and smoking share similar melatonin-mediated pathways.[22]

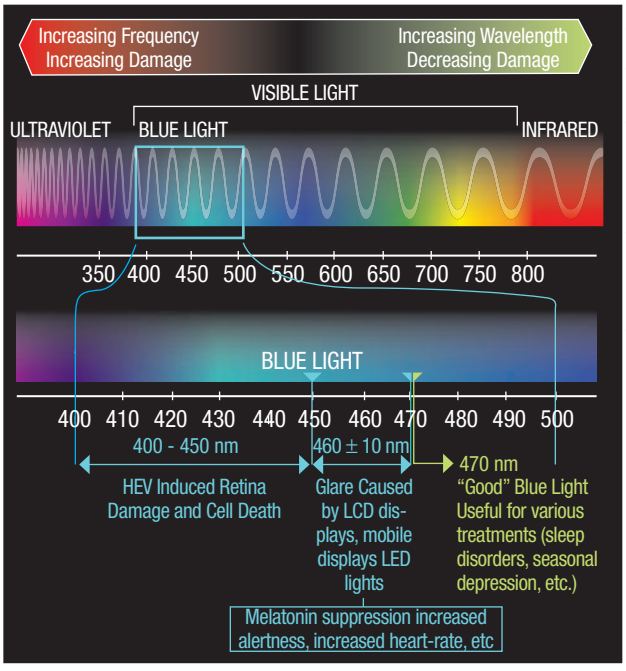


Image: Adapted with permission from High Performance Optics

While many consider blue light harmful, it can have some positive effects, particularly HEV wavelengths above 470nm. For example, the baby in the image below is receiving blue light therapy to treat jaundice.

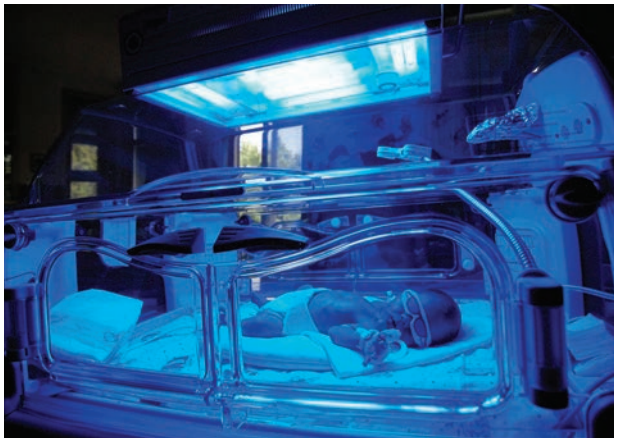


Image: Getty Images

On the Bright Side

HEV-mediated circadian rhythm modulation has some positive attributes as well, perhaps the most encouraging being improved memory and cognition.

Light therapy to treat Alzheimer's disease is not new, but the applications to other neurodegenerative diseases such as Huntington's and Parkinson's are also beginning to show promise.[7] In one study, researchers investigated the effects of white LED light on a strain of mice susceptible to developing Alzheimer's and reached some promising conclusions. In the first of two studies, they implanted a fiber optic probe into the rodents' hippocampus and flashed white LED light at 40Hz for one hour. The mice had a decrease in the degraded form of the protein tau and a decrease of beta amyloid.[9] In the follow-up study, the same strain of mice were placed in a blackout room and exposed to the same white LED light flickering at 40Hz for one hour—with similar results.[9] While researchers have yet to determine whether the results are due to the flicker frequency or the HEV emission, the findings continue to advance our understanding of the potential effects of light therapy.

Other studies have evaluated the use of blue light in promoting mental alertness.[23] In one investigation, researchers evaluated the effects of daytime and nighttime blue (460nm) and green (555nm) light exposure. Subjects were exposed to either blue light or green light for 6.5 hours in the middle of a 16-hour wake cycle during the biological day. Compared with the green light, exposure to blue light (either at night or during the day) improved auditory reaction times, with electroencephalography readings showing greater activity associated with heightened alertness. The trade-off, however, was that blue light exposure at night increased sleepiness during the day.[23]

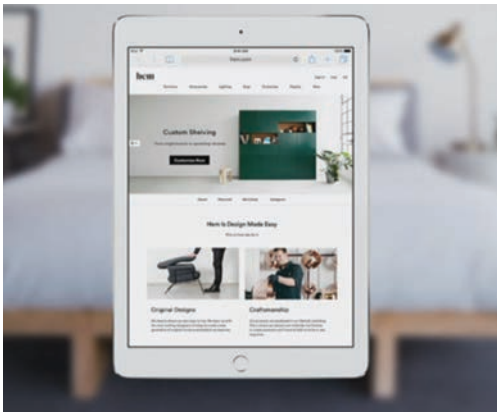
Eyes in the Spotlight

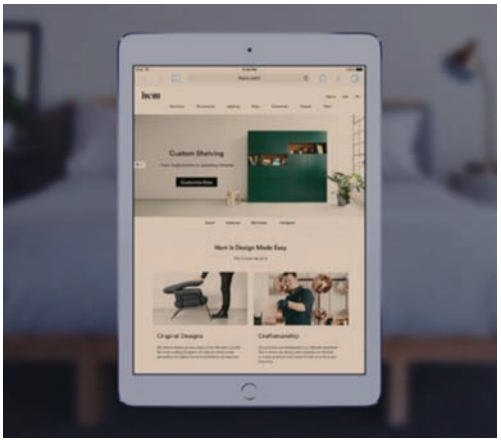
With all the attention on HEV and its effect on our systemic well-being, it's easy to lose sight of the fact that it also has an impact on our ocular health—issues addressed every day in our clinical care of patients.

Researchers have long known that HEV has a deleterious effect on retinal health.[24] It's been tied to visual changes such as the development of drusen and photoreceptor apoptosis, and DVS.[24]

As blue light enters the eye, it is absorbed by the photoreceptor outer segment, triggering the conversion of the opsin retinal to all-trans-retinal. Subsequent oxidation of the all-trans-retinal leads to the production of reactive oxygen species (ROS) such as singlet oxygen, hydrogen peroxide and other free radicals that accumulate in the photoreceptor outer segment.[24] These ROS have an affinity for breaking down cell membranes, causing

Images: Apple





Selective blue light filtering technologies, such as Apple's Night Shift feature, seen here, can help patients limit HEV exposure at night.

incomplete phagocytosis of expended photoreceptor outer segments and a build-up of lipofuscin in RPE cells.

Lipofuscin levels are present early in life and gradually increase over time, becoming measurable around age 10 and peaking around age 70.[12] However, too much can result in drusenoid changes that can lead to age-related macular degeneration (AMD).[12] When lipofuscin and its hydrophobic fluorophore A2E begin amassing in the RPE, the risk of RPE and photoreceptor damage with subsequent death secondary to repeated HEV exposure is at its greatest.

To highlight the significance of HEV in this process, consider the results of this study: after saturating human RPE cells with A2E and exposing them to blue and green light, researchers found RPE apoptosis only occurred in those exposed to the blue light.[13] Other researchers using rodent models found exposure to blue LEDs and full-spectrum white LEDs for a period as short as nine days (in 12-hour cycles of light and dark) cause obvious damage to the outer nuclear layer. The researchers attribute the damage to the generation of the ROS during excitation of the photoreceptor cells.[25]

One missing component in much of this research, however, is the role of the xanthophyll carotenoids lutein and zeaxanthin. These pigments are not only crucial in filtering out short wavelength light before it reaches the photoreceptors and RPE, but also serve as powerful free-radical scavengers.

A fortunate part of the aging process is the natural yellowing of the human lens that increases over time (effectively reducing the amount of blue light reaching the retina); unfortunately, the density of the macular pigments decrease, leaving the eye increasingly vulnerable to HEV's phototoxic effect. This inverse relationship is profoundly important when considering risk mitigation strategies because it serves as a natural segue into a conversation with your patients about how the loss of macular pigmentation, a family history of AMD and the ever-increasing exposure to HEV (natural or man-made) can fuel the breakdown of the RPE and eventually lead to permanent retinal damage.

Caution should be exercised, however, because in spite of all the available research into the relationship between HEV and AMD, the evidence is still inconclusive. The challenges in establishing a definitive link are wide ranging and include such things as differences in pupil sizes and inter-palpebral fissure width, duration and intensity of the light, the distance from the light source and the actual spectral composition of the light source itself.

Often overshadowed in the HEV-ocular complications research, DVS is another effect of HEV exposure—and one that may affect many more patients than the HEV-AMD relationship. According to a 2015 meta-analysis of the literature, 64% to

Light Among the Stars

In another powerful display of the importance of HEV photo-entrainment research, NASA recently announced that it will be replacing the fluorescent lighting on the International Space Station with an LED lighting system they can manipulate. This new system will allow researchers to evaluate the efficacy of modulating the human circadian rhythm by changing the wavelength and intensity of the light source. Using a dynamic lighting schedule, they will employ three pre-determined settings to optimize lighting for: (1) circadian rhythm adaption using a general illumination setting of 4,500K white light at 210 candela; (2) improved alertness and performance with a phase shifting/alertness setting of 6,500K, blue-enriched white light at 420 candela; and (3) sleep enhancement using a pre-sleep setting of 2,700K, blue-depleted white light at 90 candela. They hope this type of lighting system can reduce astronauts' dependence on pharmacological aids such as caffeine and sedatives to regulate their sleep-wake-alertness patterns. For those in space, and perhaps someday those of us on Earth, we will be able to more effectively harness the power of light to improve our sleep patterns and our health.

National Aeronautics and Space Administration. Testing solid state lighting countermeasures to improve circadian adaptation, sleep, and performance during high fidelity analog and flight studies for the International Space Station (lighting effects). www.nasa.gov/mission_pages/station/research/experiments/2279.html. Accessed April 2, 2018.

90% of computer users experience DVS with symptoms ranging from eye strain and headache/eye ache to blurry vision, diplopia and dry, burning, watering eyes.[10] HEV is now implicated in the sequelae associated with DVS because its short wavelength creates a greater propensity for it to scatter as it moves through the ocular tissues, resulting in glare and decreased contrast sensitivity. In addition, this same scatter triggers micro-accommodative changes and, consequently, changes in the phoric posture—a constant "auto-focus" and "auto-depth" that never achieves stability.[11]

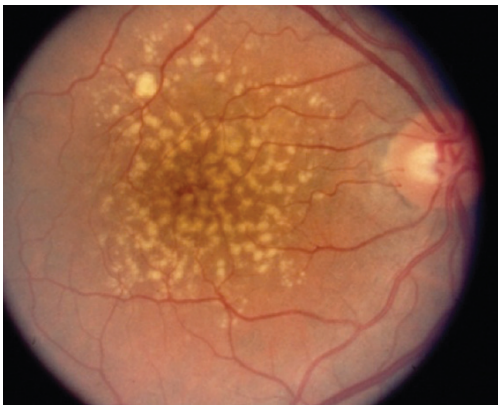
Blue Light Just Right

Rather than completely blocking blue light, patients should take a two-pronged approach to ensure the blue light they are exposed to doesn't become detrimental to their health. First, patients should eat naturally occurring carotenoids, foods high in omega-3 fatty acids and phytoflavenoids while decreasing risky behaviors such as diets high in saturated and trans fats, smoking and unprotected sun exposure. By changing a diet to consume foods higher in lutein and zeaxanthin, the body becomes better able to form additional macular pigmentation that can protect against macular degeneration due to blue light absorption, reduce photo-oxidative stress and subsequently stabilize RPE cell membranes.[26] Second, patients should try to better manage their blue light exposure by incorporating spectacle lenses, smart in-home lighting options and other technologies. While each strategy is equally important, we as optometrists can be particularly helpful with in employing the latter strategy.

Some of the blue-filter lens coatings available include: Essilor's Prevencia (peak absorption between 415nm and 455nm), Kodak's Total Blue (blocks up to 80% of the HEV between 380nm and 440nm), Hoya's BlueControl and Zeiss's BlueProtect (peak absorption between 380nm and 455nm). Gunnar Optiks are specialized computer spectacles designed with peak absorption between 380nm and 470nm. All of these technologies are designed to selectively block harmful HEV, allowing the longer wavelength blue light through and are easy for patients to incorporate into their daily regimens. While they are not the panacea for everything HEV, research shows these coatings decrease harmful effect by between 10.6% and 23.6% while reducing symptoms associated with DVS in roughly 30% of patients.[6]

Coupling these lens coatings with technologies such as f.lux, Apple's Night Shift or any blue light-reducing apps for the Android platform will provide patients with even more comprehensive benefits in the areas of phototoxic protection and circadian rhythm stabilization—ultimately reducing the ocular and systemic risks associated with HEV exposure.

HEV exposure is inevitable and has many negative, and a few positive, systemic and ocular ramifications. Each source—the sun, artificial lighting and digital devices—affects us in a different yet predictable manner depending on the distance, intensity, duration and timing of exposure. Our job is to provide patients with recommendations to balance the good and bad effects of HEV, for both their systemic and ocular health. Reducing risky behaviors such as smoking, unprotected sun exposure and poor diet, while increasing healthy behaviors such as dietary intake of naturally occurring carotenoids and phytoflavinoids and incorporating blue light-filtering spectacle lenses and other technologies are all crucial to protecting our patients. ■



HEV's known effects on retinal health have led researchers to explore it's effects on AMD.

Dr. Hefner spent 20 years in private practice in Topeka, Kan., specializing in pediatrics and family practice optometry before re-joining the faculty at Pacific University College of Optometry in 2017.

He continues to serve in the United States Air Force as the Medical Group Commander for the 190th Air Refueling Wing in Topeka.

Dr. Hefner has provided humanitarian health care in El Salvador, Armenia and Trindad and Tobago. He is a past president of the Kansas Optometric Association, Heart of America Contact Lens Society, and a Fellow of the American Academy of Optometry.

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1. Which of the following is a source of HEV blue light?
a. Sun.
b. LED TV.
c. Digital devices.
d. All of the above.

2. HEV blue light is defined as which wavelength of light?
a. Below 380nm.
b. Above 720nm.
c. 380nm to 500nm.
d. 555nm to 720nm.

3. Which of the following has the greatest percentage of its output in the HEV range?
a. Compact fluorescent lights.
b. LEDs.
c. Incandescent bulbs.
d. Sun.

4. What is the greatest source of HEV blue light?
a. Sun.
b. Indoor lighting.
c. Smartphones.
d. Computers.

5. Which of the following conditions can be treated with HEV blue light?
a. Acne.
b. Psoriasis.
c. Jaundice.
d. All of the above.

6. Which of the following cells play the leading role in circadian rhythm regulation?
a. ipRGCs.
b. Rods.
c. Cones.
d. None of the above.

7. What is the peak spectral sensitivity of the ipRGCs?
a. 380nm to 410nm.
b. 444nm to 486nm.
c. 570nm to 620nm.
d. 640nm to 684nm.

8. ipRGC depolarization results in a neurotransmission that ultimately leads to the pineal gland releasing what hormone?
a. Melatonin.
b. Oxytocin.
c. Insulin.
d. Melanopsin.

9. In a 2015 study, researchers discovered that use of an e-reader in the period immediately preceding bed caused:
a. An increase in the release of melatonin.
b. Drowsiness.
c. Digital vision syndrome sequelae.
d. Delayed melatonin secretion.

10. HEV-related melatonin suppression has been associated with:
a. Elevated blood pressure.
b. Seasonal affective disorder.
c. Peak blood glucose production.
d. All of the above.

11. According to the Nurses' Health Study II:
a. Ambient LAN is associated with an increased incidence of hormone-related cancers such as breast and ovarian.
b. Ambient LAN is tied to poorer patient care outcomes.
c. Ambient LAN and smoking share similar melatonin-mediated pathways.
d. a and c.

12. Using mice predisposed to developing Alzheimer's, researchers found:
a. There was an increase in the protein tau and beta amyloid.
b. There was no change in their protein tau.
c. There was a decrease in the protein tau and beta amyloid.
d. The mice showed no behavioral changes.

13. HEV blue light has been shown to:
a. Increase mental alertness.
b. Decrease mental alertness.
c. Degrade auditory reaction times.
d. Diminish electroencephalography readings.

14. NASA is deploying a new LED lighting system to the International Space Station in an effort to modulate astronauts' circadian rhythms by:
a. Changing the intensity of the light.

OSC QUIZ

b. Changing the wavelength of light.
c. Adjusting exposure durations.
d. All of the above.

15. Lipofuscin is the result of:
a. Increased stores of all-trans-retinal.
b. Incomplete phagocytosis of the expended photoreceptor outer segments.
c. Excessive amounts of carotenoids.
d. None of the above.

16. Lipofuscin builds up in the:
a. RPE.
b. bipolar cells.
c. Amacrine cells.
d. ipRGCs.

17. Which of the following fluorophores have been associated with HEV-related apoptosis of the RPE?
a. A4F.
b. A2E.
c. C3PO.
d. RA2.

18. Which of the following are true regarding carotenoids:
a. They filter out short wavelength HEV light.
b. They are free-radical scavengers.
c. They naturally increase in density with age.
d. a and b.

19. HEV-related digital vision syndrome can present with which of the following symptoms?
a. Eye strain.
b. Headache.
c. Blurry vision.
d. All of the above.

20. A comprehensive strategy to managing HEV exposure should include which of the following?
a. Spectacle lens coatings.
b. Blue light reducing apps.
c. Dietary modifications to include more carotenoids and phytoflavinoids.
d. All of the above.

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19. Ⓐ Ⓑ Ⓒ Ⓓ
20. Ⓐ Ⓑ Ⓒ Ⓓ

Post-activity evaluation questions:

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

21. Improve my understanding of blue light and its ocular and systemic effects. ① ② ③ ④ ⑤
22. Add to my knowledge of blue light's role in circadian rhythm modulation. ① ② ③ ④ ⑤
23. Recognize the positive effects of blue light and how to filter exposure to improve health. ① ② ③ ④ ⑤
24. Better understand the deleterious effect of blue light on retinal health. ① ② ③ ④ ⑤
25. Improve my knowledge of dietary changes that can affect blue light-associated side effects. ① ② ③ ④ ⑤
26. Improve my knowledge of blue light filtering lenses and digital device technologies. ① ② ③ ④ ⑤

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

27. The content was evidence-based. ① ② ③ ④ ⑤
28. The content was balanced and free of bias. ① ② ③ ④ ⑤
29. The presentation was clear and effective. ① ② ③ ④ ⑤
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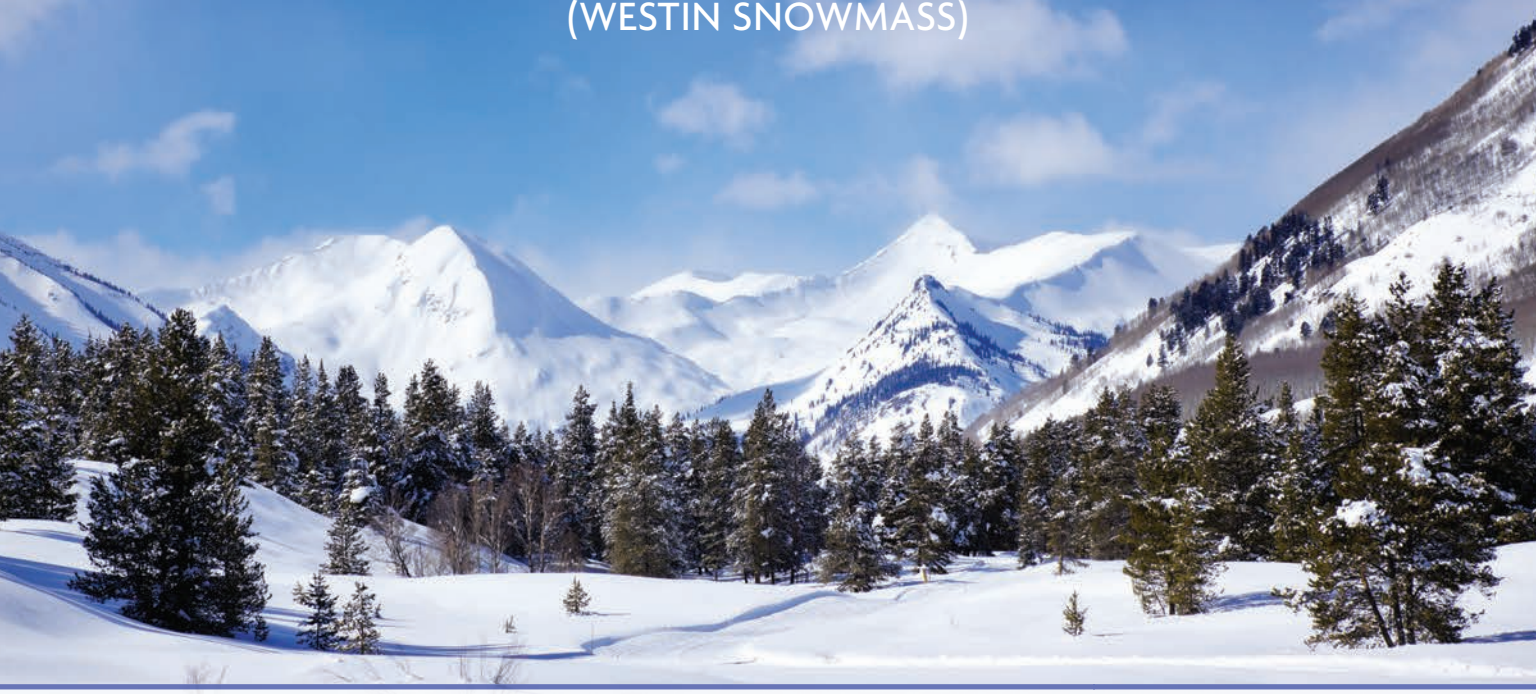

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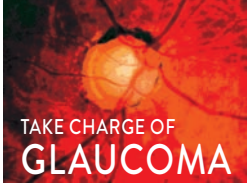

Part 2: Diagnostics

PEER • REVIEWED • PRA • ARTICLE

Prepping Your Diagnostic Toolbox

Caring for this population takes more than an IOP check these days. Here are some must-have diagnostic strategies.

By Brian Fisher, OD, April Stursma, OD, Austin Lifferth, OD, and Stephanie Carter, OD

Today's diagnostic technology is growing rapidly; not long ago, time-domain optical coherence tomography (TD-OCT) revolutionized the way we looked at certain ocular conditions such as glaucoma. Now, affordable high-resolution spectral-domain OCT (SD-OCT) performs everything from retinal nerve fiber (RNFL) to ganglion cell analysis (GCA), and even non-contact pachymetry and anterior segment angle evaluation, depending on the instrument. Here's a look at traditional and innovative glaucoma diagnostics and how to incorporate them into daily clinical practice.

A Timeless Triad

Visual field testing, Goldmann applanation tonometry (GAT) and systematic optic nerve evaluation are the three classic tests that synergistically help diagnose primary open-angle glaucoma (POAG) early and detect glaucomatous progression.

Pressure

POAG care continues to be based on the concept that higher intraocular pressures (IOPs) are a significant risk factor for both the development and the progression of the disease. Several population-based studies show the prevalence of POAG increases as IOP increases and reducing IOP lowers the rate and incidence of glaucomatous progression.[1-6] Nonetheless, an IOP greater than 21mm Hg is a relatively arbitrary measure and is a poor screener for diagnosing glaucoma on its own.

Clinicians should take several IOP measurements, especially in patients with normal tension glaucoma, as each measurement is a brief snapshot of the overall diurnal IOP range and likely does not fully represent the peak pressures often found during the nighttime hours and while in the supine position.[7] Repeat IOP measurements will help the clinician to better understand the patient's potential risk for progression.[8-10]

Despite IOP's utility in glaucoma management, as many as 50% of patients within the "normal" 10mm Hg to 21mm Hg range have glaucoma.[11] One study more aptly describes glaucoma as "a group of progressive optic neuropathies characterized by degeneration of retinal ganglion cells and resulting changes in the optic nerve head."[12] This broader understanding calls for an equally broader approach to glaucoma evaluation that includes not only IOP, but also optic nerve head (ONH) and visual field testing.[13]

Structure

Research has identified several key quantitative and qualitative optic nerve features that clinicians should systematically evaluate with dilated fundus photography.[14-20] Some of the most significant of these morphological features include:

Optic disc and cup. The size of the optic disc correlates directly with the size of the optic cup and the

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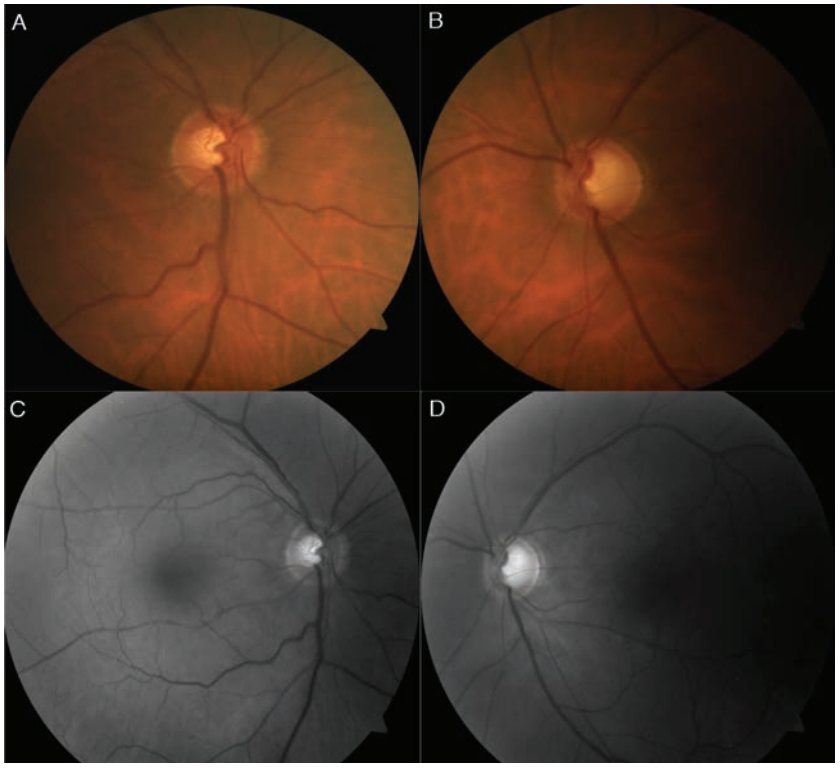


Fig. 1. Color fundus photo (A) of the right optic nerve shows mild superior thinning, while the left (B) shows noticeable superior, temporal and inferior thinning. Red-free photos show loss of the bright RNFL pattern superiorly in the right (C) and both superiorly and inferiorly, extending temporal, in the left (D).

neuroretinal area—thus, a large cup in a large disc may be normal, while an average cup in a small disc may suggest glaucomatous damage.[21,22] Optic disc size is also affected by racial differences: people of African descent are known to have larger nerves, followed by Asians and Hispanics, with Caucasians having relatively smaller nerve sizes.[21,22]

The normal shape of the optic disc is a slightly vertical oval (the vertical disc diameter is about 7% to 10% larger than the horizontal diameter) while the normal shape of the optic cup is horizontally oval, making the normal neuroretinal rim usually the broadest in the inferior and superior disc regions. When considered as a single variable, glaucoma susceptibility is mostly independent of the optic disc shape, but the ISNT rule (optic nerves typically show a larger rim width inferior, superior, nasal and then temporal) can help differentiate pseudo glaucomatous nerves from early glaucoma.[22,23]

Neuroretinal rim. There is preferential loss of neuroretinal rim in the inferior and superior optic disc regions in early glaucoma, temporal rim loss in moderate glaucoma and nasal inferior then nasal superior in advanced glaucoma.[22] The sequence of this preferential neuroretinal sector rim loss correlates with the progression of visual field defects. In addition, neuroretinal rim pallor is a common clinical endpoint of several different non-glaucomatous optic neuropathies and neurological diseases. In cases where the pallor extends beyond the cupping, additional bloodwork, imaging or both may be necessary.[24-26]

Optic disc hemorrhages. These subtle, transient and easily missed findings are an important risk factor for the development of glaucoma in patients with ocular hypertension, are a risk factor for glaucoma progression and are a significant predictor of visual field loss, with a faster rate of visual field progression.[5,27-30]

Retinal nerve fiber layer (RNFL). Defects in this layer are some of the earliest signs of glaucoma and are most commonly found in the inferior temporal and superior temporal sectors of the optic nerve.[22,31] Research shows a seven- to eight-fold greater risk of future visual field loss if glaucomatous RNFL defects are noted at baseline.[32]

Function

Reliable baseline visual field testing, traditionally captured with standard automated perimetry (SAP), is essential to monitor for any functional glaucomatous progression, and clinicians should obtain at least two reliable visual fields in the first six months, or three if the patient exhibits a high lifetime risk of visual disability.[33,34] One study suggests six visual fields in the first two years can help to rule out rapid progression and establish baseline data.[35] The American Optometric Association clinical guidelines and the American Academy of Ophthalmology Preferred Practice Patterns are both consistent with these recommendations, while also highlighting the importance of customized visual field testing frequency for each patient.[36,37]

Follow-up should be based on the risk of progression and, if progression is noted, the estimated rate of progression, in addition to risk factors for further progression, stage of disease and life expectancy.[34]

Clinicians should also consider 10-2 visual field testing in all stages

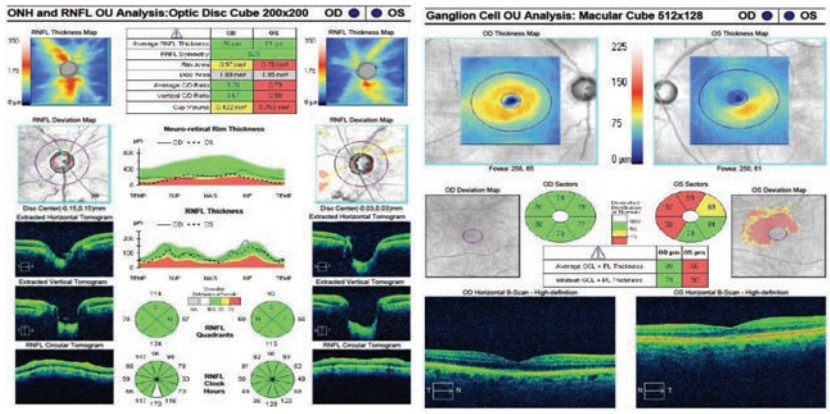


Fig. 2. At left, the RNFL thickness map shows mild superior thinning in the right eye, and notable superior and inferior thinning in the left. The deviation map shows focal superior and temporal thinning in the right. The left is significantly thinner compared with the right average thickness and the superior, inferior and temporal quadrants and wedges. Despite the RNFL showing green in these sections, structural thinning is evident. In the GCC at right, the right eye is within normal limits, whereas the left shows notable thinning to the average GCL thickness, minimum GCL thickness and to the inferior nasal, superior nasal and superior sectors. There is also mild thinning in the superior temporal sector.

of the disease—especially with normal tension glaucoma—to more frequently detect glaucomatous macular involvement that is often undiagnosed (or underestimated) with standard 24-2 or 30-2 testing.[38-41]

The 24-2 threshold visual field pattern tests a total of 54 points, each of which are each six degrees apart. Twelve of the points tested are within the central 10 degrees, and only four of these points are tested within the macular region, which accounts for more than 30% of the total retinal ganglion cells (RGCs) and more than 60% of the visual cortex area.[42] By comparison, the 10-2 threshold visual field pattern tests 68 points that are all two degrees apart and one degree from either side of the horizontal and vertical meridians.[38]

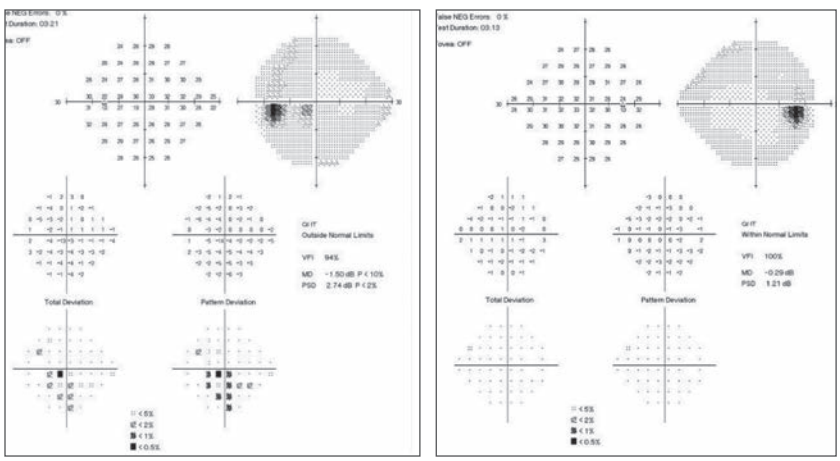
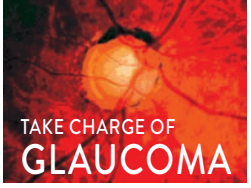


Fig. 3. In the baseline 24-2 SAP, the left eye shows an inferior arcuate close to fixation with central involvement. The right eye shows a normal baseline SAP 24-2.



Technologies on the Horizon

Many new tools finding their way into research are providing novel metrics for understanding glaucomatous damage and progression:

OCT angiography (OCT-A). This noninvasive imaging technology analyzes retinal, macular and ONH microcirculation. Evidence suggests reduced blood supply in the optic nerve and peripapillary region of glaucoma patients, and a correlation may exist between the severity of the disease and the degree of the reduced blood flow.[1-3] The most common test parameters used in glaucoma patients include vascular density within the ONH, peripapillary retina and macula, as well as flow index of the optic disc.[1,4-6] Currently, most platforms currently in use do not have a normative database to compare these parameters. One of the largest OCT-A studies found peripapillary and disc vascular densities were significantly lower in open-angle glaucoma than suspects and healthy eyes.[7] Other researchers show lower total and temporal ONH vessel densities in glaucomatous eyes. These reduced densities strongly correlated with OCT, visual field mean deviation (MD) and visual field index (VFI).[3,8]

Corneal hysteresis (CH). This represents the cornea's ability to bend, flex and absorb biomechanical stress from applied pressure. Several studies show that increased CH (>9mm Hg), measured with the Ocular Response Analyzer (Reichert), allowed the cornea to absorb intraocular stresses caused by high-low diurnal IOP fluctuations.[9,10] At times when CH is >9mm Hg, mechanical stress is reduced at the level of the lamina cribrosa and optic nerve, and less glaucomatous neuropathy was seen in these patients.[9,10] In contrast, study participants who possessed low CH (<9mm Hg) were at increased risk for developing glaucomatous optic neuropathy.[9,11-13] Factors that contribute to low CH include thin CCT, age, elevated IOP and increased HbA1C.[9,14] Although research has a long way to go in better understanding CH's role in glaucoma, clinicians may one day measure CH as part of routine glaucoma care and follow up.[15]

Electroretinogram (ERG). Three types of ERG exist: full-field (ffERG), multifocal (mfERG) and pattern (pERG). ffERG tests the entire retina, including all rod and cone function; mfERG provides a topographic map of cone function; and pERG evaluates RGC function, as does the photopic negative response (PhNR).[16-18] Studies agree that PhNR correlates with GCC thickness, but has lower diagnostic ability than that of GCC OCT.[18-22] PhNR and pERG are the most common ERG techniques used in glaucoma diagnostics, but pERG has been more established in clinical research.[19,20] Studies investigating pERG in glaucoma suspects agree that it was beneficial in early stage disease, but less in moderate-to-advanced stages due to amplitude saturation. Other research shows a severe reduction in amplitude before structural RNFL thinning occurred. They also detected glaucomatous changes four years before it was confirmed on SAP.[20-21] Before implementing ERG into clinical practice, clinicians must weigh the cost and benefit of the test, especially considering current literature suggests OCT still overtakes ERG in diagnostics.[16-24]

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Overlooked Techniques

Gonioscopy, an oft-ignored yet vital skill, helps clinicians determine the type of glaucoma and individualize the treatment plan, particularly for the one-in-four patients with secondary glaucoma.[43]

In addition, measuring a patient's central corneal thickness (CCT) can help inform the treatment plan as well, considering the Ocular Hypertension Treatment Study revealed, independent of IOP, a strong correlation between CCT in ocular hypertensive patients and conversion to open-angle glaucoma. The researchers found subjects in the thinnest CCT subgroup (<558µm) were three times more likely to convert to glaucoma than those within the thickest CCT subgroup (≥588µm).[44] Therefore, patients with CCT <558µm are at the greatest risk of conversion and should be monitored closely.

The Advanced Toolbox

As our understanding of glaucoma grows, so do the technologies we can use to detect and manage it. While not a part of the traditional triad, OCT can help assess for longitudinal structural change in all stages, especially in early and pre-perimetric glaucoma. It also provides quantitative and noninvasive objective measurements of the macular ganglion cell complex (GCC), peripapillary RNFL and ONH parameters. One study found RNFL OCT detected damage in approximately one-third of glaucoma patients up to five years before the appearance of the earliest visual field defects.[45]

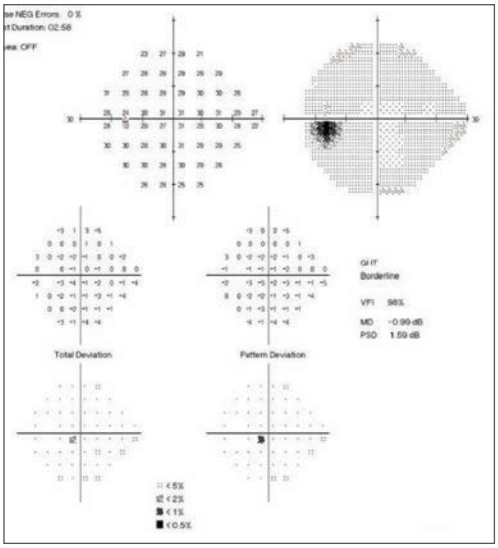

Fig. 4. Repeat SAP 24-2 of the left eye show a deep central defect close to fixation within the central 10 degrees.

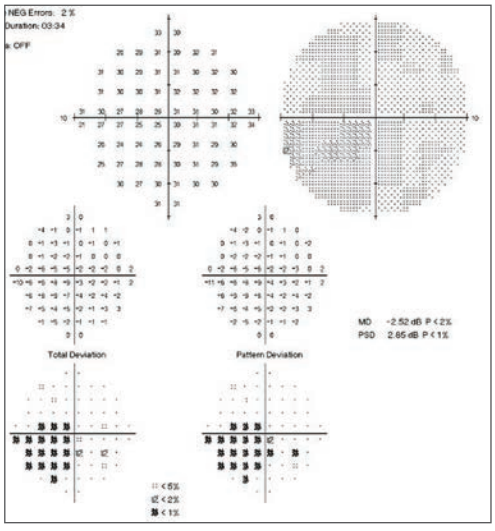

Fig. 5. Baseline 10-2 SAP of the left eye shows a deep central scotoma spatially correlating to the focal defect seen on 24-2.

RNFL OCT. Studies suggest the age-related rate of change for average RNFL thickness ranges between -0.16µm/year and -0.44µm/year.[46] One particular study showed an average RNFL loss of -0.52µm/year: -1.35µm/year in the superior quadrant and -1.25µm/year in the inferior quadrant.[46] When greater than these values, or when noticeable changes are seen on the RNFL thickness map, clinicians must determine if the change is due to inter-visit glaucomatous structural loss or intrasession noise or test-retest variability.[46,47] The most reliable approach to address RNFL loss is to examine serial changes and rate of change.[46] The RNFL OCT has a guided progression analysis (GPA) option, providing a longitudinal analysis of average, inferior and superior RNFL thickness measurements. Furthermore, a rate of change option is measured in µm/year. For baseline images, we recommend taking two RNFL OCTs on the initial visit to complete the GPA and rate of change analysis on follow up.

RNFL results should supplement the evaluation of the optic nerve and is not meant to replace functional SAP testing. OCT technology has its

limitations, including each machine's limited normative database, intrasession noise causing reduced signal strength, inaccurate test-retest variability and artifacts.[47] OCT longitudinal monitoring and serial testing is the best way to monitor these patients.

GCC OCT. The macular region is another confirmed site for initial glaucomatous damage.[48-51] RGCs and axons constitute 30% to 35% of retinal macular thickness and almost 50% of RGCs are within the macula.[42,46,48] This indicates using GCC OCT for monitoring all stages of glaucoma, ocular hypertension and suspects. GCC becomes essential in moderate-advanced glaucoma, when RNFL thickness reaches its floor effect (around 50µm), as it can't detect structural change in these stages. Likewise, GCC is useful in cases of anomalous ONH, high myopia or both.[52] Studies show GCC OCT's diagnostic ability in glaucoma is comparable with that of RNFL thickness.[46,48-51] Furthermore, other studies show GCC OCT of glaucoma suspects and pre-perimetric glaucoma participants detected thinning before functional loss.[53-55]

Putting it All Together: a Teaching Case

A 70-year-old white male presented for a routine new patient eye exam with no vision or health complaints. He provided little detail of any ocular history, but he denied a family history of glaucoma. His medical history was normal and he denied taking medications. His best-corrected visual acuity was 20/20 OD and OS, pupils were equally round and reactive to light with no afferent pupillary defect, confrontation visual fields were full to finger count and extraocular muscles were unrestricted in all gazes.

IOPs were 19mm Hg OD and 20mm Hg OS. Anterior segment evaluation was unremarkable. The anterior chamber was deep and quiet, and the anterior chamber angles were 4/4 by Van Herick. On dilated exam, the media of the lens showed trace nuclear sclerosis and no pseudoexfoliation.

Fundus examination revealed a clear vitreous, macula that was flat with even pigment, an arterial-venous ratio of 2/3 and a peripheral retina that was flat and intact with no pathology noted.

The optic nerves had a cup-to-disc ratio of 0.55 OD and 0.75 with peripapillary atrophy OS (*Figure 1*). There was mild superior neuroretinal rim thinning OD and notable superior, temporal and inferior thinning OS. No Drance hemorrhage, pallor, spontaneous venous pulsation (SVP) or loss of perfusion was seen. A higher risk for glaucoma exists when no SVP is observed.[56] The optic nerves were 1.9mm OD and 2.0mm OS, and the optic disc and cup had normal shape. The central retinal vessel trunk was centered OD and decentered superior nasal OS, which has a strong correlation to central visual field loss for all glaucoma severities.[57]

RNFL and GCC OCT findings (*Figure 2*) supplemented and agreed with the clinical findings of superior thinning OD and advanced superior and inferior thinning OS.

We chose to obtain additional IOP measurements and baseline visual fields before initiating treatment. Upon follow up one month later for 24-2 SAP testing, gonioscopy, IOP and pachymetry, the patient brought his old eye exam records, which showed an untreated maximum IOP of 25mm Hg OD and 27mm Hg OS. The patient stated he hadn't returned for follow-up care. At this visit, IOP was 22mm Hg OU, and gonioscopy was open to ciliary body 360 degrees, had normal pigment and no angle recession, peripheral anterior synechiae or neovascularization of the angle. Pachymetry was 520µm OD and 510µm OS. With these clinical findings, the patient was diagnosed with POAG.

On baseline SAP 24-2 baseline, the glaucoma hemifield test (GHT) was within normal limits OD and outside normal limits OS (*Figure 3*). All findings had a strong structure-function relationship with previous evaluations and testing. Based on these findings, treatment was initiated with latanoprost QHS OS, and the patient was scheduled to return in six weeks for repeat visual field testing and IOP check.

The patient returned with untreated IOP of 19mm Hg OD and treated IOP of 14mm Hg OS. On repeat 24-2 testing, OS showed improvement from baseline and the GHT was borderline (*Figure 4*). Due to a depressed central defect, a 10-2 SAP of the OS was acquired (*Figure 5*), which show a deep central scotoma involving fixation. Here, 24-2 SAP greatly underestimated the depth of this central scotoma, and performing 10-2 SAP exposed the depth of this functional deficit.

The patient was followed closely, and we initiated treatment in the right eye six months later due to a repeatable inferior nasal step, which correlated to the superior thinning on baseline. ■

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Fend Off the Supervillain

Here's how to protect a vulnerable cornea from opportunistic threats that might make a bad situation even worse. **Edited by Joseph P. Shovlin, OD**

Q I recently attended a lecture where the presenter mentioned the potential benefit of using a fluoroquinolone when prophylaxis is needed to ward off bacterial infection in a patient with epithelial herpes simplex virus (HSV). I'm not sure what the rationale is here. Is there any research available on this?

A "While I'm not familiar with any specific research tying fluoroquinolone prophylaxis into the treatment of HSV keratitis, microbial superinfection is an infrequently encountered, but well-known, consequence of herpetic disease and falls under the clinical spectrum of 'metaherpetic disease,'" says Aaron Bronner, OD, of Pacific Cataract and Laser Institute.

The terminology used to describe this scenario might be in need of an update, however, notes Dr. Bronner. "Metaherpetic disease is a somewhat dated descriptor of things that aren't directly HSV-related corneal infections or inflammations, but are still consequences of the previous viral activity." For these patients, prophylactic antimicrobial treatment could be helpful, he says.

Why Prophylaxis?

According to Dr. Bronner, the most common metaherpetic finding is neurotrophic keratitis. Metaherpetic microbial superinfection, on the other hand, is "less commonly seen but more acutely sight threatening."

Here, the mechanism is much like that of any opportunistic infection: "A breach in the corneal epithelium allows opportunistic microbial infection of the cornea, most typically with a member of the normal periocular flora," says Dr. Bronner. This means that the herpetic infection is only important in two ways in the setting of superinfection:

1. It provides the break in the epithelium that opens the door to microbial infection, either via a dendritic ulceration or through creation of a neurotrophic lesion.
2. An early microbial lesion in an eye with previous herpetic activity can be easily misdiagnosed as being herpetic early in its course, which allows the actual etiology to remain untreated, leading to worse outcomes.

"Therefore, prophylaxis against microbial infection has good clinical rationale," says Dr. Bronner. "That said, for most dendritic ulcers, I don't routinely add prophylaxis unless the lid margins look unhealthy or the epithelium is slow to heal." Dr. Bronner does always add prophylaxis in cases of neurotrophic ulcers, however, since these lesions can epithelialize much more slowly, increasing the likelihood of superinfection.

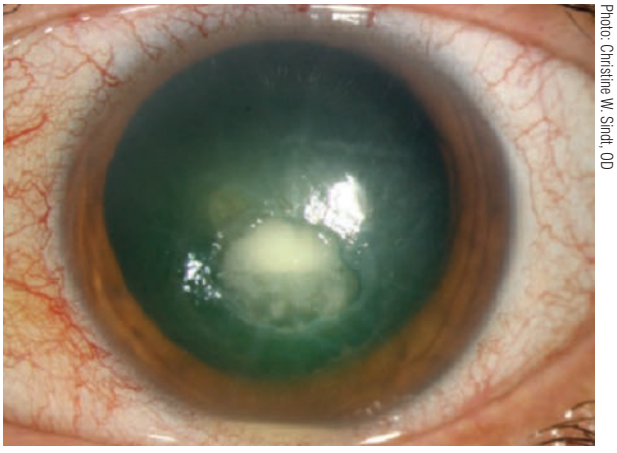

Photo: Christine W. Sindt, OD

By implementing prophylactic antimicrobial treatment, practitioners may be able to protect patients from bacterial superinfection, shown here.

Cover Your Bases

If you are considering prophylactic antimicrobial treatment, be sure to check your patient's history first. "Any time you are adding antimicrobial prophylaxis, I think it's wise to ask the patient about a history of methicillin-resistant *Staphylococcus aureus* (MRSA)," Dr. Bronner says. "If they are carriers or have a previous history of MRSA infection, you may need to adjust the prophylactic agent to cover for this." ■

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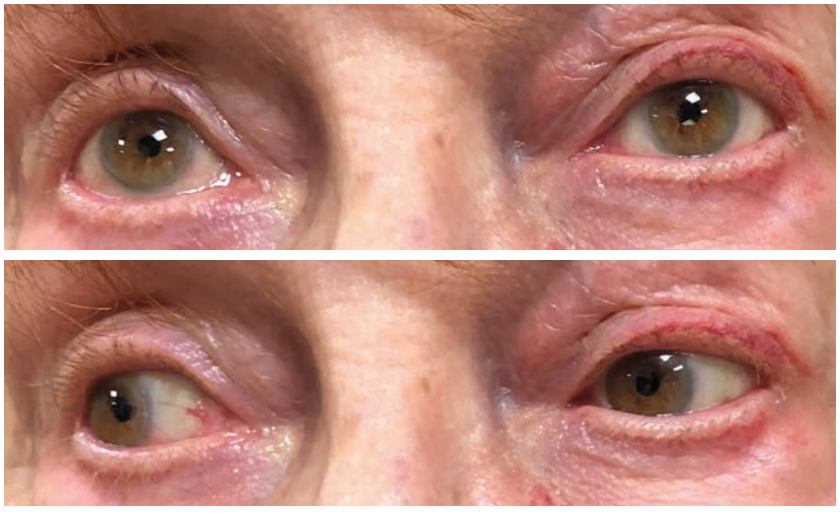
Missed Connections

A carotid cavernous fistula can present with a number of ocular findings—be ready to refer and follow. **By Michael Trottini, OD, and Michael DelGiodice, OD.** ***Case by Dr. Trottini.***

An 88-year-old Caucasian female presented to the clinic with acute onset of pain and swelling around her left eye. She stated that about a week prior she had an upper respiratory infection, and after blowing her nose, she noted her left eye had become red and swollen. She also had a headache localized behind the left eye that persisted after the illness passed. She mentioned the onset of a "whooshing" sound in her left ear and double vision that gradually worsened in intensity over the past week. Her extensive medical history included hypertension, high cholesterol, congestive heart failure, chronic obstructive pulmonary disease, deep vein thrombosis, hypothyroidism, anxiety/depression and a history of lung cancer about 30 years ago. Her current medications included Diovan (valsartan, Novartis), bumetanide, Lasix (furosemide, Sanofi-aventis), Eliquis (apixaban, Pfizer), Pravachol (pravastatin, Bristol-Myers Squibb), levothyroxine, albuterol and alprazolam.



Figs. 1 and 2. Upon initial exam, the patient showed upgaze restriction, above, and adduction deficit when looking to the right, below. Periorbital fluid and hemorrhage are noted in both images as well.

The Stats

On examination her best-corrected visual acuity (BCVA) was 20/30 in the right and left eyes. Intraocular pressures were 12mm Hg in the right eye and 14mm Hg in the left eye. Extraocular motilities were full on the right but showed upgaze and adduction restriction on the left (*Figures 1 and 2*). Cranial nerves three, four and six were intact. Exophthalmometry was 12mm OD and 16mm OS. Her pupils were normal and equal with no afferent pupillary defect. There was periorbital fluid and some hemorrhage around the left eye and some ptosis on the left eyelid. She had had cataract surgery OU, and her IOLs were in good position. Her retinal exam was normal except for minimal epiretinal membranes and posterior vitreous detachments in both eyes, which were noted in her past history and were stable.

Imaging ASAP

Given her symptoms and exam findings, my main concern was an orbital mass or process behind the left eye. Because she was taking Eliquis and the concern for any risk of orbital or intracranial hemorrhage, I decided to admit her to the hospital rather than send for outpatient testing. Prompt computed tomography (CT) of the orbits and CT-angiography (CTA) of the head and neck showed an enlarged left superior orbital vein with contrast filling on arterial phase imaging, which was highly suspicious for a left carotid cavernous fistula (CCF) (*Figure 3*).

I consulted neurology and neurosurgery, and they scheduled the patient for a diagnostic cerebral angiography to confirm the CCF with probable treatment the following day. Her Eliquis was stopped and she was switched to an aspirin and Plavix (clopidogrel, Bristol-Myers Squibb). The cerebral angiography showed a subtype A direct, high-flow CCF, which is high risk for intracranial hemorrhage.

Aftermath

Treatment was indicated, and a transarterial embolization was successful, with near complete resolution of the fistula.

The patient remained in the hospital for a number of days due to dyspnea, anxiety and decreased renal function; once these issues were managed and resolved, the patient was discharged. One month later upon re-examination, I noted all of her symptoms, including the proptosis and extraocular motility restriction, had resolved.

Discussion

CCFs are abnormal connections between the carotid artery and the cavernous sinus.[1] Various classifications of CCFs exist, based on etiology (traumatic or spontaneous), hemodynamics (high-flow or low-flow lesions) or anatomy or angiographically (direct when the fistula originates directly in the internal carotid artery, or indirect when the fistula originates in the dural branches of the carotid artery).[1,2] Further characterization of CCFs are divided into four subtypes according to the arterial supply: subtype A is a direct, high-flow CCF between the internal carotid artery (ICA) and cavernous sinus; subtypes B, C and D are indirect, low-flow CCFs where the fistula originates in the dural branches of the carotid artery.[1]

The classification system is important from a neurosurgical standpoint because the treatment is specific for each entity.[2]

Ocular symptoms and clinical findings vary. Headache, retro-orbital pain, decreased vision and diplopia are typical patient complaints.

A direct CCF, as seen in this patient, is a high-flow lesion where blood flows from a high-pressure compartment (the ICA) to a low-pressure compartment (the cavernous sinus).[1] When this occurs, it causes an increase in pressure in the cavernous sinus, which can result in cranial nerve three, four, five and six palsies.[1,2] In this patient's case, her motility restriction was not due to a nerve palsy, but to the enlarged ophthalmic vein.

Additionally, this increase in pressure leads to engorgement of the ophthalmic veins, causing symptoms such as ocular bruit, chemosis and pulsatile proptosis.[1] Direct, high-flow CCFs are most commonly a result of trauma. When spontaneous or outside the setting of trauma, they are generally associated with a ruptured intracavernous aneurysm, Ehlers-Danlos syndrome or fibromuscular dysplasia.[1,2] In this patient's case, she denied any prior trauma and did not have any risk factors for this direct CCF. While blowing her nose likely did not cause the CCF, it is possible the pressure it created exacerbated the pre-existing condition.

Indirect, low-flow CCFs do not pose a significant risk for hemorrhage and can be monitored. If the patient cannot tolerate the symptoms or if there are ocular morbidities stemming from a low-flow fistula, endovascular treatment is recommended.

Because direct, high-flow CCFs have an 8.4% risk of hemorrhage, treatment is always recommended.[3] The main goal for treatment is exclusion of the fistula while maintaining patency of the ICA.[1] This is typically achieved with endovascular embolization with a combination of detachable balloons, coils, stents or liquid embolic agents.[4]

Once the diagnosis has been made, the OD's role is to continue to monitor all findings that were present on initial exam and to follow for resolution after neurosurgical intervention. Symptoms from a CCF will significantly improve following treatment. Resolution of symptoms and clinical findings can occur hours or days after treatment but can sometimes take weeks to months.[1] ■

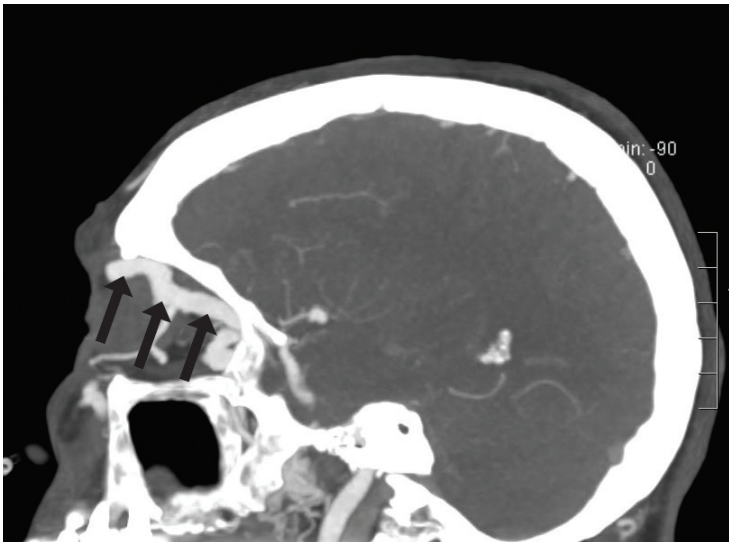


Fig. 3. The patient's CTA imaging demonstrates an enlarged left superior ophthalmic vein.

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Pressure Watcher

Worried about glaucoma, a patient presents with complaints of visual disturbances.

By James L. Fanelli, OD

A 32-year-old African-American female presented to the office in January with complaints of fluctuating vision and blurriness in both eyes that tended to wax and wane. The symptoms started approximately two weeks earlier, and had a slightly progressive nature to the intensity and duration of the visual disturbances, prompting her urgent visit.

Four years earlier we diagnosed her with ocular hypertension. Her intraocular pressures (IOPs) were in the mid 20s and she had a positive family history of glaucoma. She was myopic, correctable to 20/20 OD, OS and OU, with otherwise normal ophthalmic findings. She was lost to follow up until the most recent presentation.

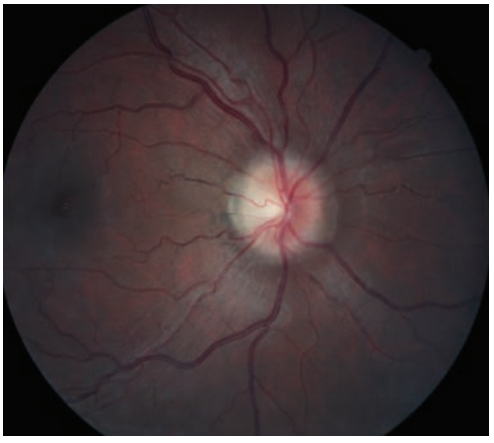
When she presented in January, she was somewhat worried about glaucoma, given our previous visit when the condition was discussed, her lack of interim follow-up and the fact that several family members have glaucoma—one of which apparently has suffered significant vision loss. Her medications included: Lasix (furosemide, Sanofi Aventis), losartan, Sprintec (norgestimate and ethinyl estradiol, Teva), Orencia (abatacept, Bristol-Myers Squibb) and Plaquenil (hydroxychloroquine, Sanofi). She reported no known allergies to medications. She started taking Plaquenil only a year earlier and her rheumatologist had told her that she needed to have a baseline ophthalmic examination, which she didn't schedule.

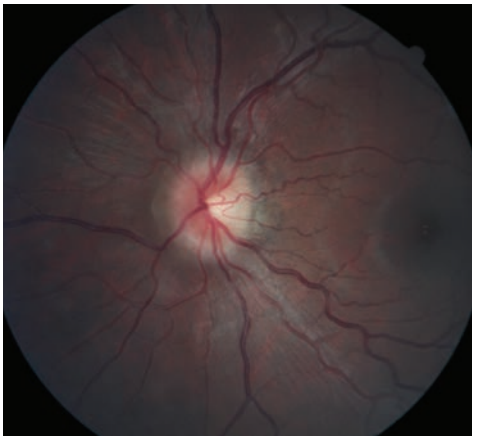
Her complaints were difficult to verbalize, other than transient blur in both eyes that lasted anywhere from 10 minutes to as long as three hours, with no particular pattern to their onset. She experienced mild headaches temporally on the right side more than the left that sometimes accompanied the blurry episodes. These symptoms were new and her medication regimen was stable for approximately one year, meaning that there were no new medications introduced that may have precipitated the complaints.

Examination

Entering and best-corrected visual acuities were 20/25- OD and 20/20-2 OS. Refractively, she was in the -7.50D range OU, similar to her last refraction several years earlier.

A slit lamp examination of her anterior segments was essentially unremarkable. Her corneas were clear, her anterior chambers were deep and quiet, and anterior chamber angles were wide open at the slit lamp. Her irides and crystalline lenses were clear in both eyes. Applanation tensions at 3:30pm were 25mm Hg OD and 26mm Hg OS. Pachymetry readings were 525µm OD and 533µm OS, which were similar to previous measurements. Prior to dilation, threshold





Figs. 1 and 2. At left, the patient's right fundus as it appeared on her initial, urgent presentation. At right, her left eye, also at initial presentation. Note the "C" shaped halo shadowing in both the right and left eyes, with the temporal neuroretinal rim essentially intact.

standard automated perimetry was performed, and there were scattered areas of missed points in both eyes, with reasonable reliability indices. However, the field defects were not identifiable other than scattered generalized depression.

The patient was then dilated in the usual fashion with 1% tropicamide and 2.5% phenylephrine. Through dilated pupils her anterior vitreous was unremarkable. The macular evaluations of both eyes were also unremarkable, as were the retinal vascular appearances. Her optic nerves were characterized by small cups, approximately 0.25x0.25 OD and 0.30x0.35 OS, but her neuroretinal rims were elevated, especially nasally. There was slight asymmetry in the neuroretinal rim elevation, but both the right and the left nasal neuroretinal rims were elevated and markedly different from images taken several years earlier. The clinical appearances of the discs were characteristic of disc edema modified Frisén scale stage 1 (*Figures 1 and 2*).[1] Spontaneous venous pulsations were absent in both eyes, and there was no record of their presence or absence at the earlier visit.

Her temporal neuroretinal rims were intact bilaterally, with no evidence of edema or swelling. Optical coherence tomography (OCT) optic nerve scans, as well as macular OCT scans, were obtained (*Figure 3*). The macular scans, and in particular the ganglion cell layer scans of both maculae, were entirely normal. The perioptic retinal nerve fiber layer (RNFL) circle scans were also essentially normal, though the innermost RNFL circle scan did demonstrate slight increased thickness to the superior, nasal and inferior RNFL sectors, as compared with the reference data base. The 4.1 and the 4.7 circle scans were entirely normal, indicating that the apparent swelling of the neuroretinal rim did not extend from the center of the optic nerve too far outward. However, the radial optic nerve scans, as well as the optic nerve raster scans, did in fact demonstrate bilateral disc edema with anterior bowing of the Bruch's membrane and retinal pigment epithelium (RPE) complex, especially nasally.

Discussion

This patient presented with worries associated with visual disturbances she attributed to the possibility of glaucoma. In fact, the clinical picture was just the opposite; rather than elevated IOP being the cause of her complaint, the picture was more suggestive of elevated intracranial pressure (ICP).[2]

Certainly, she does carry risk factors for developing glaucoma, but we are not looking at that condition

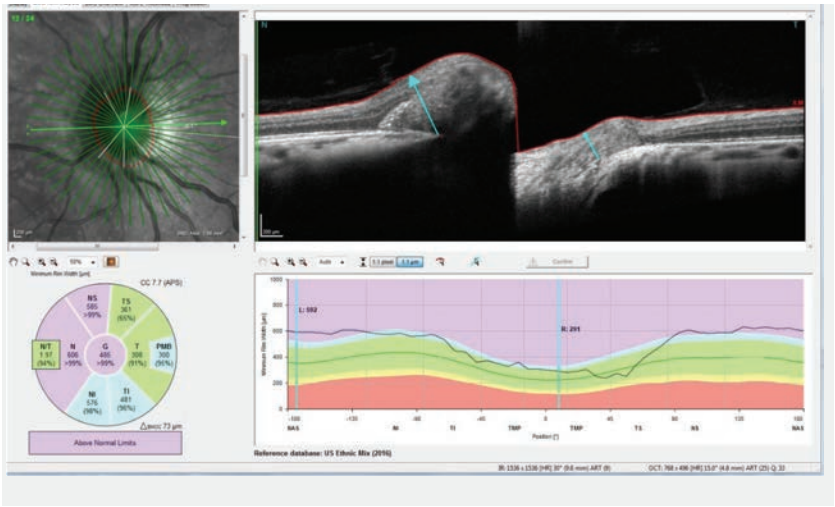


Fig. 3. Note the anterior deflection of the Bruch's membrane/RPE complex nasally in the patient's right optic nerve, consistent with elevated retrolaminar pressure, and disc edema due to elevated intracranial pressure.

here. In reviewing her medications list, as mentioned, there were no new medications introduced in the past year, and disc edema is not generally associated with the medications she was currently taking. Her macular OCT scans, as well as her general slit lamp macular evaluations, indicated that there were no visible effects from the Plaquenil use. A review of her headache history revealed nothing that raised any red flags. She did admit that she had gained about 25 pounds since our last visit. She was approximately 5'6" and weighed 165lbs.

Given the presentation, she was worked up in the usual fashion of a patient with bilateral disc edema, with emphasis given to several possible etiologies, with idiopathic intracranial hypertension (IIH) and dural venous sinus thrombosis at the top of the list. I ordered magnetic resonance imaging (MRI) and magnetic resonance venography (MRV), and she was scanned about four days after the initial presentation. The MRV was normal, as was the MRI. There was no displacement of the cerebral structures, and given no trauma history, cerebral bleeds were not very likely.

Interestingly, though a lumbar puncture (LP) is in order for this patient, in the area in which I practice, most neurologists have ceased performing LPs, and neurology consults are exceedingly difficult to obtain short of an emergency consult. Ultimately, I decided that the LP was not critical to the case at this point, and I began a regimen of 500mg Diamox sequels (acetazolamide, Duramed Pharms Barr) BID along with a dietician consult. She was scheduled for follow-up and has complied with the medication regimen and follow-up schedule. Thirty days after initiating the carbonic anhydrase inhibitor, repeat scans of her optic nerves show reduction in the disc edema (*Figure 4*).

So why is a case like this discussed in a *Glaucoma Grand Rounds* column? What relevance does it have to glaucoma, other than the patients risk for developing glaucoma? Well, the answer lies in the anatomy of the optic nerve. Traditionally, we think of glaucoma as a disease where the IOP exceeds a healthy level to the point that optic nerve perfusion is compromised, resulting in compromise to the individual ganglion cells. This can happen when IOP is significantly elevated, or conversely when optic nerve perfusion pressure is greatly reduced, or anywhere in between. Which makes for a sliding scale for what is considered an IOP that is 'too high.'

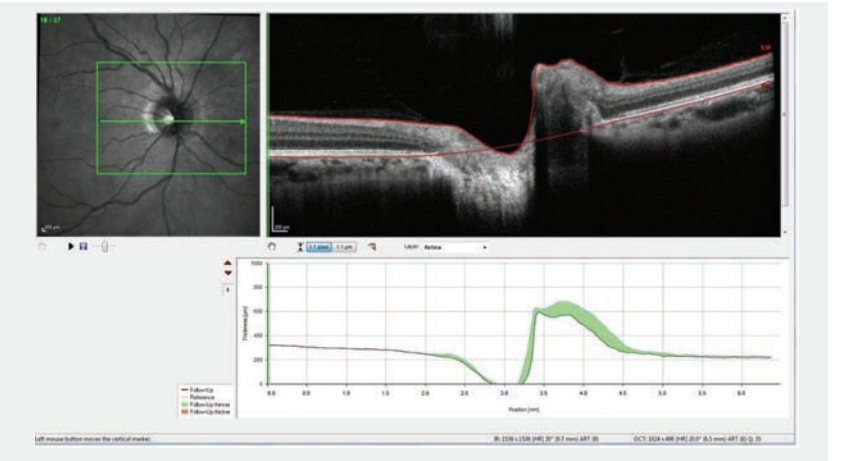


Fig. 4. This optic nerve raster OCT scan is a follow-up showing significant reduction in the nasal neuroretinal rim swelling, as evidenced by the green areas in the change analysis.

Under Pressures

But that is only half the picture: what is happening anterior to the lamina cribrosa, to IOP and to the blood supply to the individual ganglion cells, or axomplasmic flow through the individual ganglion cells. That is but one consideration. The other consideration is what is happening behind the lamina cribrosa. Specifically, in the subarachnoid space around the optic nerve, anteriorly to the lamina cribrosa. And this is the area of cerebrospinal fluid dynamics.

Cerebrospinal fluid pressure is exerting pressure on the lamina anteriorly, whereas intraocular pressure is exerting pressure on the lamina posteriorly. And trapped in the middle between these two pressure gradients are the ganglion cells we exhaustively examine in the context of glaucoma. This is the basis of the translaminar pressure gradient that needs to be considered in all patients with glaucoma and optic nerve disease.

Many factors can affect ICP, aside from the obvious things like space-occupying lesions, cerebral hemorrhages and IIH.[3] Sleep apnea, cardiovascular diseases and body weight can all affect ICP in one way or another. And when you alter ICP, you are exerting influence on the retrolaminar optic nerve. Furthermore, just as IOP tends to have diurnal variations, so too does ICP. Sometimes those diurnal fluctuations can work in tandem and somewhat "protect" the optic nerve, whereas other times, they can work antagonistically and negatively affect the optic nerve health (for example, when IOP is high and ICP is low).

In the NASA space station program, research shows individuals who remain in a zero gravity environment for extended periods of time, develop disc edema.[4] Researchers believe this is directly tied to the effects of zero gravity to ICP, and the asymmetry between the ICP and the IOP.[4]

As for our patient, weight reduction and temporary use of Diamox is resulting in normalization of her optic nerve appearance. Once this is behind us, then we continue moving forward concentrating on the anterior side of the lamina cribrosa, and her risk of developing glaucoma. ■

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MIGS: Getting Better All the Time

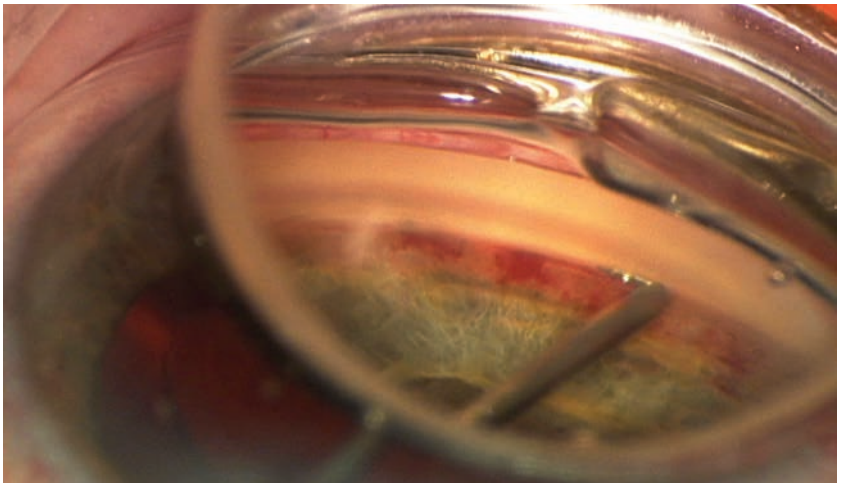
Studies show these techniques are evolving and leading to improved outcomes.

By Joseph W. Sowka, OD, and Alan G. Kabat, OD

A 55-year-old woman under management for primary open angle glaucoma for the past 15 years presented for a checkup. Her peak intraocular pressures (IOPs) were 31mm Hg OD and 26mm Hg OS. She had moderate glaucomatous damage to each optic disc.

For the majority of her care, her treated IOP was in the mid-teens with stability in her visual field, optic disc photography and optical coherence tomographic retinal nerve fiber layer. However, over the past two and a half years, her IOP slowly and consistently rose to the mid 20s, despite escalating her therapy to include latanoprost and dorzolamide-timolol FC (she was allergic to brimonidine). Selective laser trabeculoplasty (SLT) lowered her IOP to the upper teens for about a year before she rose again to the mid 20s in her right eye with her left remaining in the high teens.

In this case, medical therapy was maximized and trabeculoplasty already employed. Though apparently stable, the right eye maintaining mid-20s pressure was disconcerting. Repeat trabeculoplasty was not embraced and trabeculectomy and tube implantation were likewise discounted as excessively risky in an eye maintaining an elevated IOP but not yet showing signs of progression. In that she had a mildly symptomatic cataract in her right eye (with 20/30- acuity), it was felt that lens extraction with implantation of an iStent (Glaukos) would be her next best, safest route of pressure reduction. She successfully underwent combined cataract extraction with iStent implantation with a subsequent sustained, medicated IOP of 18mm Hg in that eye.



Under magnification you can see an iStent being inserted through the trabecular meshwork during cataract surgery.

Two For One

Research shows that modern cataract surgery with phacoemulsification substantially reduces IOP and need for glaucoma medications in many patients.[1-3] The exact mechanism of IOP reduction is unknown, though some have speculated that removal of a cataractous lens removes any possible phacomorphic component of IOP rise.

Minimally invasive glaucoma surgeries (MIGS) have gained popularity in cases of glaucoma and co-existent cataract due to its ease of implantation during cataract surgery, favorable safety profile and the possibility for effective and long-lasting IOP reduction. Additionally, patients may also experience a reduced need for IOP-lowering medications.[4] This can be especially popular among patients who are non-adherent.

Most MIGS procedures are performed *ab interno* and microincisional through a clear cornea during cataract surgery. The procedures are minimally traumatic and can be performed coincident with lens extraction. The iStent, specifically, is implanted in conjunction with cataract surgery through the trabecular meshwork and into the Schlemm's canal.[5]

Some researchers speculate that increased outflow facility following phacoemulsification solely accounted for the IOP reduction and that the MIGS devices added little in additional IOP reduction.[6-8]

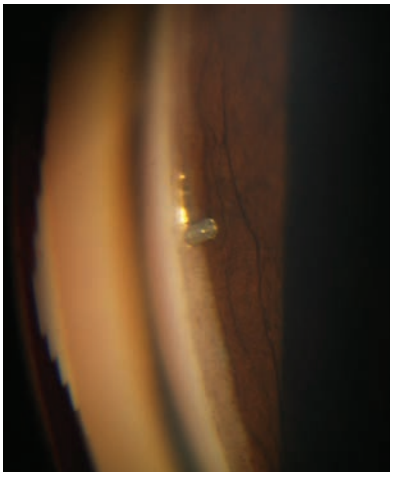
Indeed, many early reports may have substantiated only a minimal extra IOP reduction through a MIGS procedure.[7] However, since these procedures have a substantial learning curve, later results should be considered in judging efficacy.

In the Literature

A recent meta-analysis of 37 studies examining a combined 2,495 patients, iStent with phacoemulsification as compared with phacoemulsification alone, noted 4% IOP reduction from baseline following phacoemulsification alone compared with 9% reduction in IOP following an iStent implant with phacoemulsification.[9] This IOP reduction rose to 27% following two iStent implants with phacoemulsification. Compared with cataract extraction alone, iStent with phacoemulsification resulted in significant reduction in the postoperative IOP and a significant decrease in the number of glaucoma medications needed postoperatively. The overall assessment was that iStent with phacoemulsification significantly outperforms phacoemulsification alone in terms of reduction in IOP and postoperative medications being used.[9]

Other non-comparative studies show positive outcomes, safety and decreased medication burden. A study involving a mainly a Hispanic population with predominantly moderate or severe glaucoma shows a substantial reduction of IOP and medication need and favorable safety for a year following stent implantation during cataract surgery. At one year, 94% of eyes in that study achieved their predefined treatment goal of reduced IOP and/or medications. Two eyes had filtering surgery; the remaining 95% avoided such treatment.[10]

Another prospective, open-label, non-randomized study evaluating



When implanted, as seen here, the iStent and other MIGS devices are designed to help lower IOP. New research shows how they're fairing.

long-term safety and efficacy of iStent trabecular microbypass stent implantation during cataract surgery in patients with primary open-angle, pseudoexfoliation glaucoma, ocular hypertension or secondary or post-traumatic glaucoma saw that the procedure was safe and effective, as measured by a sustained reduction in IOP and medication use and an excellent safety profile through three years after surgery.[11]

Looking to the Future

While results have been improving as surgeons work through learning curves, future advancements in the iStent trabecular bypass are being developed. Glaukos is currently pursuing FDA approval for a second-generation product called iStent Inject, a new trabecular bypass system that implants two devices, rather than one as with the current iStent. Results of a prospective, randomized, multicenter clinical trial of the iStent Inject were presented at the 2018 American Society of Cataract and Refractive Surgery annual meeting. The device met the study's primary and secondary effectiveness endpoints. At 24 months, 75.3% of the iStent Inject cohort achieved a 20% or greater reduction in unmedicated IOP, compared with 61.9% for the cataract-only cohort and a mean unmedicated IOP reduction of 6.9mm Hg for the iStent Inject cohort, compared with 5.4mm Hg for the cataract-only cohort.

MIGS procedures are statistically better at reducing medication load and IOP when combined with cataract surgery as opposed to the effects of cataract surgery alone. With surgical experience and advancements in the devices implanted, better IOP outcomes can be expected. Much the same way early glaucoma imaging devices improved diagnosis, but paled in comparison to current technology, we can likely expect that later generation MIGS procedures and devices will exceed those being used today. ■

Drs. Sowka and Kabat have no financial interest in any products mentioned. Dr. Sowka has previously served in an advisory manner for Glaukos.

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

Snip and Squeeze: Canaliculitis

Surgery may be the best option for patients with this condition.

By Leonid Skorin, Jr., DO, OD, MS, Emmalee Toldo, OD, and Sebastian Baker, CNP

Although primary lacrimal canaliculitis accounts for only 2% to 4% of lid pathologies, it is important to recognize, as misdiagnosis may result in delayed treatment and worsening infection.[1]

Patients commonly present with localized pain, persistent unilateral epiphora, recurrent or nonresolving conjunctivitis and chronic discharge.[1,2] The condition has a 5:1 predilection for females, and the inferior canaliculus is affected in two-thirds of cases.[1,3] Classic signs consist of a "pouting" punctum, yellowing of the surrounding skin and localized hyperemia.[1,2] *Actinomycetaceae* organisms are the common infectious agent, although other bacterial, fungal or viral entities can be culprits.[2] With time, they clump together in yellowish clusters of sulfur granules or concretions.

Topical and oral antibiotics, warm compresses and digital manipulation are common initial therapy, although the time between presentation and accurate diagnosis often leads to concretions deep within the canaliculus.[3] Complete removal of the infection and any concretions requires surgical intervention.[1]

Under the Knife

Prior to surgery, a topical anesthetic is applied to the affected eye and an injection of lidocaine 1% with 1:100,000 epinephrine is administered to the pericanalicular tissues. The area is prepped with povidone iodine and draped. After confirming anesthetic effect, a plastic corneal shield is placed to protect the globe.

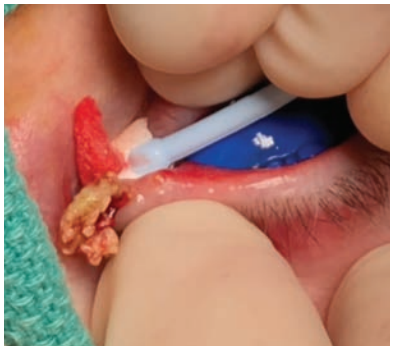

Photo: Emmalee Toldo, OD

After making an incision along the eyelid canaliculus, the surgeon expresses the sulfer granules.

The surgeon makes an incision along the longitudinal aspect of the eyelid canaliculi, sparing the puncta, and expresses the canaliculi contents. Any expressed concretions should be sent to pathology. The surgeon then passes a dilating probe through the puncta to check for patency. Curettage on the inner lining of the canaliculi and the adjacent areas ensures complete removal of concretions and ensures no pyogenic granuloma is present. If the surgeon finds a pyogenic granuloma, it is excised and also sent to pathology.

An antibiotic (e.g., moxifloxacin ophthalmic solution 0.5%) is then injected through the canalicular system and into the lacrimal sac using a tuberculin syringe and lacrimal catheter. After control of hemostasis with digital pressure, the plastic corneal shield is removed and an additional drop of topical antibiotic is applied to the corneal surface.

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

Recovery Road

The patient uses topical antibiotics QID in the affected eye for one week and should continue any previously prescribed oral antibiotics and return for follow-up in one week. Ice packs on the eyelids and over-the-counter pain medications are appropriate as needed. Patients experience minimal postoperative pain and achieve good cosmetic appearance once the edema resolves.

Comanaging clinicians should review the pathology and microbiology results to determine if the causative organism is susceptible to the prescribed antibiotics. If so, the patient should continue the treatment for an additional week. If not, switch the patient to a treatment therapy based on the results of the culture and continue the regimen for one to two weeks.[1] ■

Dr. Skorin is an ophthalmologist at the Mayo Clinic Health System in Albert Lea, Minn.

Dr. Toldo is a recent graduate of Pacific University College of Optometry.

Mr. Baker is a nurse practitioner participating in the Mayo Clinic Emergency Medicine Fellowship program.

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RESPONSIBILITIES: CANDIDATES ARE EXPECTED TO BE HIGHLY KNOWLEDGEABLE IN THE FIELD OF PRIMARY CARE OR VISION SCIENCE, AND CAN DEVELOP AND TEACH COURSES AND/OR LABORATORIES IN THE SUBJECT AREA. THE CANDIDATE MUST ALSO BE ABLE TO PROVIDE DIRECT PATIENT CARE AND CLINICAL INSTRUCTION TO PROFESSIONAL STUDENTS AS WELL AS RESIDENTS, AND BE INVOLVED IN INTERDISCIPLINARY PRACTICE WITH OTHER EDUCATIONAL PROFESSIONALS.

Candidates must be willing to actively participate in curricular assessment, professional development, student counseling and service activities within the college, university and the scientific community. Successful candidates are also expected to be involved in research and scholarly activities, and have a sincere commitment to optometric education, community service and patient care. Primary duties include, but are not limited to:

a) **Teaching**
- Developing and delivering lectures and/or laboratories for related areas, as assigned;
- Embracing and enhancing the didactic
- philosophies in the O.D. program;
- Maintaining and expanding the high quality clinical practice environment for optometry students on rotation;
- Precepting students on clinical rotation at the Midwestern University Eye Institute where applicable;

b) **Service**
- Helping to maintain and grow the state of the art optometry program with a strong interdisciplinary focus that meets the needs of patients in the surrounding community; is efficient, patient friendly, and cost-effective;
- Working closely together with all optometry and ophthalmology faculty to provide a complete range of eye and vision care services;
- Participating in leadership roles in state, regional, and national optometry organizations;
- Participating on College and University committees, as assigned;
- Participating in College and University service activities.

c) **Scholarly activity**

Engaging in research and scholarly activity, including presentations at scientific meetings, research, and publication in peer reviewed journals sufficient to qualify for academic advancement in a non-tenure or tenure track position. advancement in a non-tenure track position.

QUALIFICATIONS: Primary care candidates must possess a Doctor of Optometry degree from an ACOE-accredited institution, must have completed an ACOE-accredited residency, and must be eligible for an optometric state license in the state in which the college is located. Primary eye care clinical expertise is also required. For the vision science position, a strong preference will be given to candidates with a PhD in Vision Science with established research and publication activities, although candidates with promising research programs are also encouraged to apply. The University provides start-up funds for new faculty researchers.

CONTACT INFORMATION: Interested applicants should apply online at www.midwestern.edu and include curriculum vitae and letter of interest specifying the position and college that he/she wishes to be considered for. Inquiries may be directed to Dr. Melissa Suckow, Dean; Midwestern University: msucko@midwestern.edu

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UMSL Optometry — UMSL College of Optometry Cornea and Contact Lens Faculty Position Available
Assistant, Associate or Clinical Professor (non-tenure-track)

The College of Optometry at the University of Missouri-Saint Louis announces an opportunity to join a dynamic and progressive academic community. The successful applicant will receive a nine-month faculty appointment. Initial rank will be commensurate with prior experience, qualifications and individual interests. There is the possibility for a summer instructional assignment if mutually agreeable. Desired area of emphasis is cornea, external disease, refractive surgery and contact lenses.

Responsibilities: Successful candidates will be expected to provide didactic, laboratory and clinical instruction and serve as a mentor for student research. The positions require a commitment to working with diverse student and patient populations within college operated and affiliated clinics. Candidates should be willing to explore alternative teaching styles in the classrooms such as learner-centered and case-based approaches.

Qualifications: The position requires a Doctor of Optometry (OD) degree and license to practice optometry in Missouri. A license to practice in Illinois is desirable. Candidates with a Masters or Doctoral Degree or who have completed an ACOE-accredited residency with an emphasis in Cornea and Contact Lenses are preferred. Successful candidates will demonstrate a sincere commitment to evidence based optometric education, research/scholarship, community service and patient care in a variety of environments.

The University of Missouri-St. Louis is a public, metropolitan land-grant institution committed to basic and applied research, teaching and service with 17,000 students and 1,325 full and part-time faculty members. UMSL is the largest university in the St. Louis region and the 3rd largest in Missouri with 131 degree and associate programs. For additional information about UMSL see www.umsl.edu

The College of Optometry includes a 4-year professional degree (O.D.) program and post-professional residency programs.

For additional information about the College see optometry.umsl.edu

Those who wish to be considered a candidate for a position must provide an application that includes a letter of interest, curriculum vitae and a list of four professional references. Formal submissions must be done via the University website www.umsl.jobs.

Applications will be accepted and reviewed immediately. The positions will remain open until filled.

Questions may be directed to:
Julie DeKinder, OD, Director, Academic Programs
dekinderj@umsl.edu

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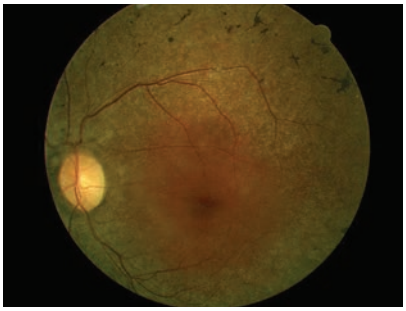
Diagnostic **Quiz**

Night Spots

By Andrew S. Gurwood, OD

History

A 44-year-old Caucasian male reported to the office with a chief complaint of poor night vision. He explained that he'd seen other eye doctors and that they had told him he had some "freckles" in his left eye. His systemic and ocular histories were unremarkable, and he denied allergies of any kind.



The findings in this fundus photograph portray a patient who presented with self-reported poor night vision. Does the imaging show any signs of disease?

Diagnostic Data

His best-corrected entering visual acuities were 20/20 OU at distance and near. His external examination was normal, with evidence of sluggish pupil on the left side. His peripheral confrontation visual field was distorted and constricted in the left eye. The biomicroscopic examination of the anterior segments found normal structures with Goldmann applanation pressures measuring 15mm Hg OU. The pertinent dilated fundus findings are demonstrated in the photograph.

Your Diagnosis

Does the patient's case require any additional tests, history or information? What steps would you take to manage this patient? Based on the information provided, what diagnosis would you make? What is the patient's most likely prognosis? To find the answers and read about how this case was managed, please visit *Review of Optometry* online at www.reviewofoptometry.com. ■

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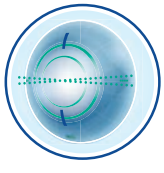

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References: **1.** Alcon data on file, 2012. **2.** Wolffsohn JS, Hunt OA, Chowdhury A. Objective clinical performance of 'comfort-enhanced' daily disposable soft contact lenses. *Contact Lens & Anterior Eye.* 2010;33(2):88-92. **3.** Laboratory study release profile; Alcon data on file, 2007. **4.** Winterton L, Lally J, Sentell K, Chapoy L. The elution of poly (vinyl alcohol) from a contact lens: The realization of a time release moisturizing agent/artificial tear. *J Biomed Mater Res B Appl Biomater.* 2007;80B:424-32. **5.** Laboratory study release profile; Alcon data on file, 2007.

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