Renal Disease, *P. 64* • Crystalline Keratopathy, *P. 74* • Managing Ocular Pain, *P. 84*

REVIEW of OPTOMETRY®

44TH ANNUAL TECHNOLOGY REPORT

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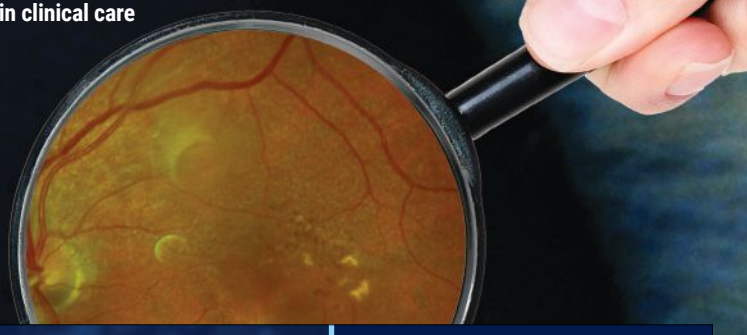
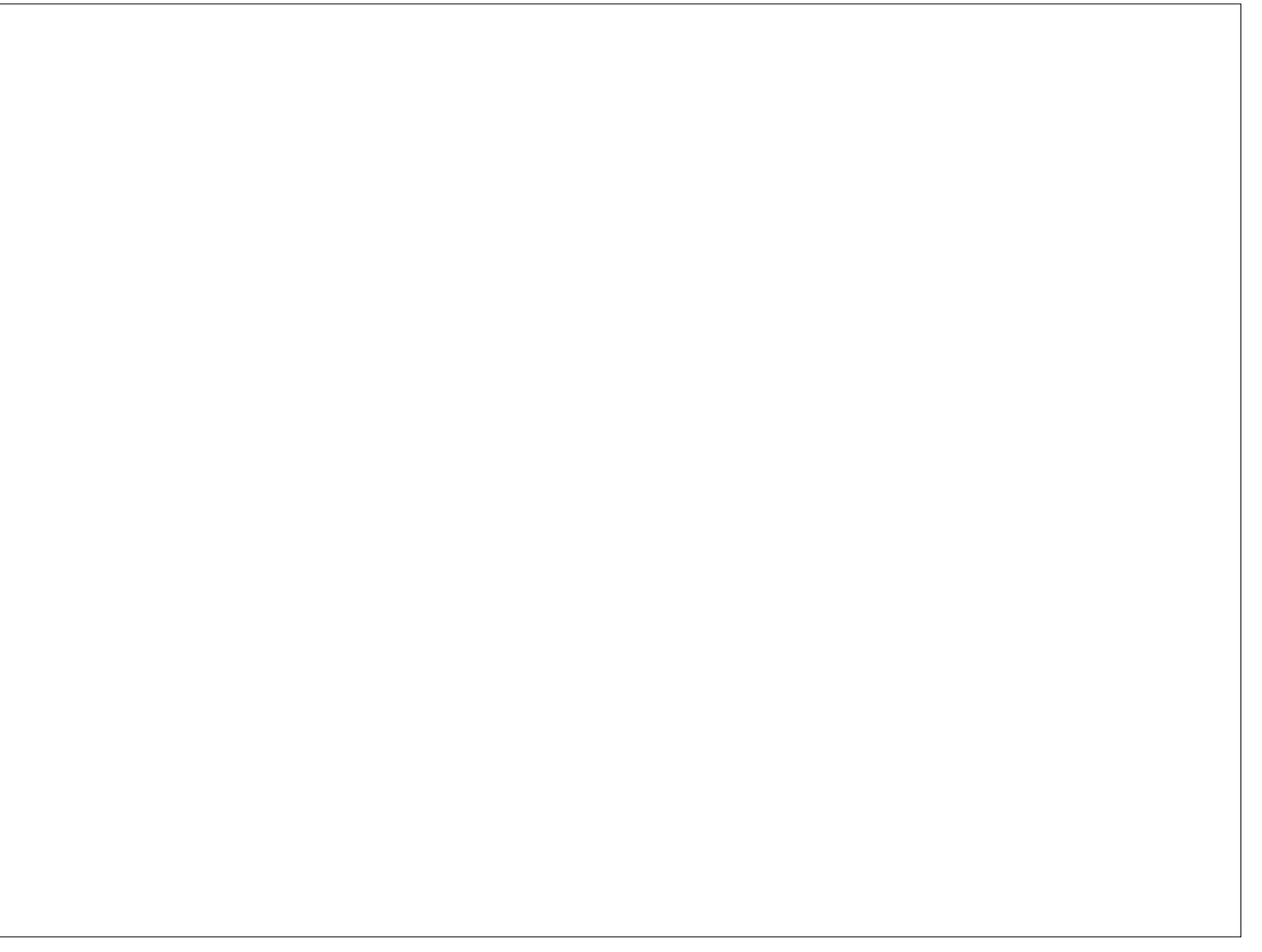


RETINAL IMAGING: SEE MORE THAN EVER BEFORE



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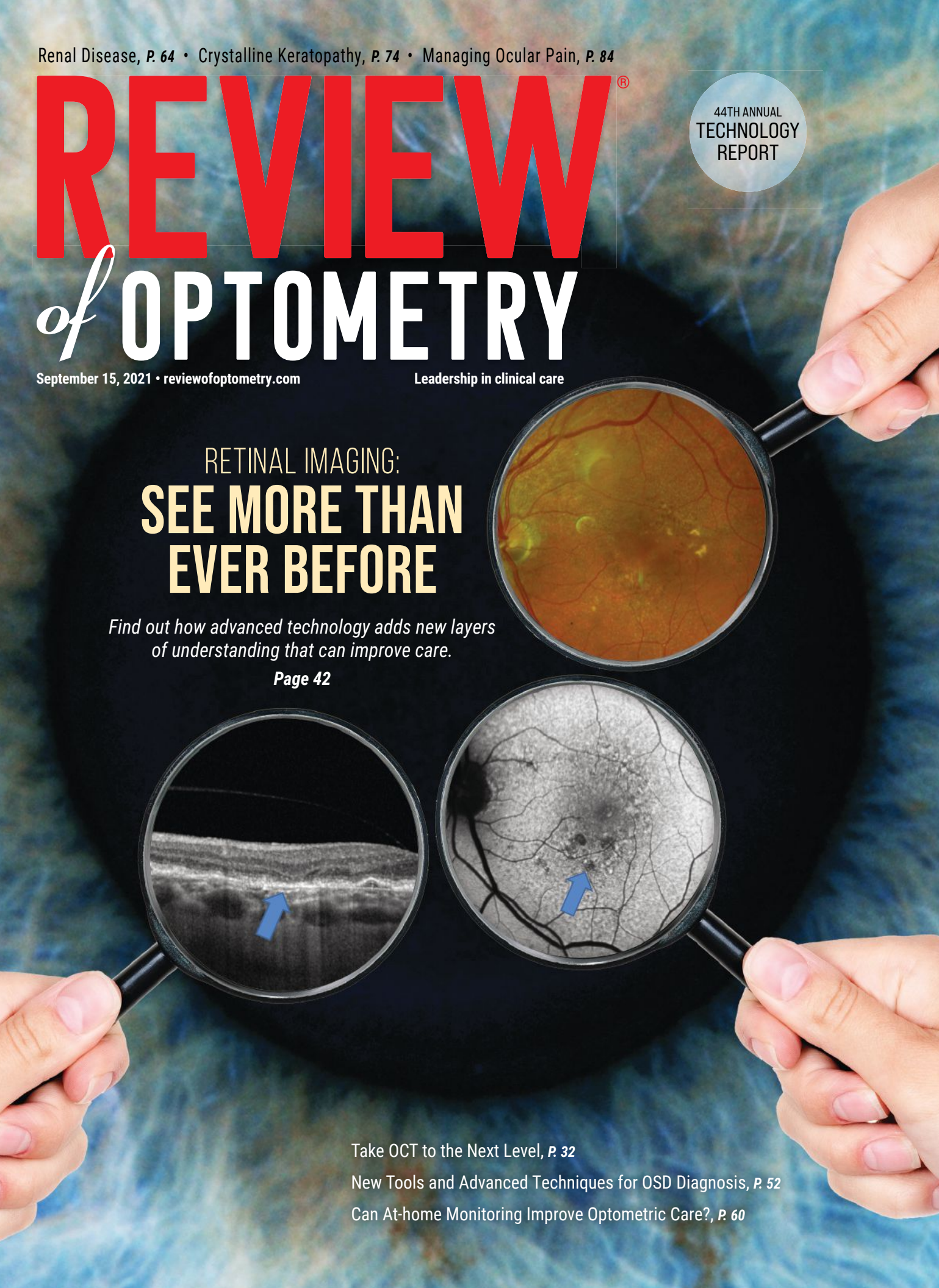
Find out how advanced technology adds new layers of understanding that can improve care.

Page 42



Take OCT to the Next Level, *P. 32*

New Tools and Advanced Techniques for OSD Diagnosis, *P. 52*

Can At-home Monitoring Improve Optometric Care?, *P. 60*

When patients rely on artificial tears alone, inflammation may persist.
Xiidra can disrupt the chronic inflammatory cycle in dry eye disease.*
It can provide lasting symptom relief in as little as 2 weeks.[1-5]†

*Xiidra blocks LFA-1 on T cells from binding with ICAM-1 that may be overexpressed on the ocular surface in dry eye disease and may prevent formation of an immunologic synapse which, based on in vitro studies, may inhibit T-cell activation, migration of activated T cells to the ocular surface, and reduce cytokine release. The exact mechanism of action of Xiidra in DED is not known.[1,2,5]

†The safety and efficacy of Xiidra were assessed in four 12-week, randomized, multicenter, double-masked, vehicle controlled studies (N=2133). Patients were dosed twice daily. The mean age was 59 years (range, 19-97 years). The majority of patients were female (76%). Use of artificial tears was not allowed during the studies. The study end points included assessment of signs (based on Inferior fluorescein Corneal Staining Score [ICSS] on a scale of 0 to 4) and symptoms (based on patient-reported EDS on a visual analogue scale of 0 to 100). Effects on symptoms of dry eye disease: a larger reduction in EDS favoring Xiidra was observed in all studies at day 42 and day 84. Xiidra reduced symptoms of eye dryness at 2 weeks (based on EDS) compared to vehicle in 2 out of 4 clinical trials. Effects on signs of dry eye disease: at day 84, a larger reduction in ICSS favoring Xiidra was observed in 3 out of the 4 studies.[1]

Indication

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

Important Safety Information

- Xiidra is contraindicated in patients with known hypersensitivity to lifitegrast or to any of the other ingredients.

NOVARTIS

Novartis Pharmaceuticals Corporation
East Hanover, New Jersey 07936-1080

Important Safety Information (cont)

- In clinical trials, the most common adverse reactions reported in 5-25% of patients were instillation site irritation, dysgeusia and reduced visual acuity. Other adverse reactions reported in 1% to 5% of the patients were blurred vision, conjunctival hyperemia, eye irritation, headache, increased lacrimation, eye discharge, eye discomfort, eye pruritus and sinusitis.
- To avoid the potential for eye injury or contamination of the solution, patients should not touch the tip of the single-use container to their eye or to any surface.
- Contact lenses should be removed prior to the administration of Xiidra and may be reinserted 15 minutes following administration.
- Safety and efficacy in pediatric patients below the age of 17 years have not been established.

For additional safety information about XIIDRA®, please refer to the brief summary of Prescribing Information on adjacent page.

References: 1. Xiidra [package insert]. East Hanover, NJ: Novartis Pharmaceuticals Corp; June 2020. **2.** Bron AJ, de Paiva CS, Chauhan SK, et al. TFOS DEWS II Pathophysiology Report. *Ocul Surf.* 2017;15(3):438-510. **3.** US Food and Drug Administration. Code of Federal Regulations, Title 21, Volume 5 (21CFR349). Accessed May 25, 2021. https:/www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfcfr/CFRSearch.cfm?CFRPart=349&showFR=1 **4.** Jones L, Downie LE, Korb D, et al. TFOS DEWS II Management and Therapy Report. *Ocul Surf.* 2017;15(3):575-628. **5.** Pflugfelder SC, Stern M, Zhang S, Shojaei A. LFA-1/ICAM-1 interaction as a therapeutic target in dry eye disease. *J Ocul Pharmacol Ther.* 2017;33(1):5-12.

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

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

1 INDICATIONS AND USAGE

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

4 CONTRAINDICATIONS

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

6 ADVERSE REACTIONS

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

- Hypersensitivity *[see Contraindications (4)]*

6.1 Clinical Trials Experience

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

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

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

6.2 Postmarketing Experience

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

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

8 USE IN SPECIFIC POPULATIONS

8.1 Pregnancy

Risk Summary

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

Data

Animal Data

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

8.2 Lactation

Risk Summary

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

8.4 Pediatric Use

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

8.5 Geriatric Use

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

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Long-term Statin Use Linked to Glaucoma

Patients who took rosuvastatin specifically were at increased risk, research suggests.

Several investigations have assessed the potential link between statin use and glaucoma onset or progression; however, the findings have been mixed. Looking into this association, a recent study that included middle-aged and elderly Australians found that long-term statin use was associated with a higher risk of glaucoma onset, specifically in users of rosuvastatin, a potent cholesterol-lowering medication.

The investigative team from China and Australia evaluated 10 years of medical claims from a large cohort of Australians who were over 45 years old. The onset of glaucoma was defined as having at least three claims of antiglaucoma medications. Among 252,545 eligible participants, 6,748 glaucoma patients were placed in the case group, along with an age-, gender- and cardiovascular disease–matched control group of 20,431 individuals who didn't take any glaucoma medications.

The case group had more statin users, representing 41% of individuals, compared with the control group (38%). The researchers found statin use wasn't tied to glaucoma onset but it posed an increased risk in participants who took statins for a longer period (over three years vs. less than one year).

Photo: Getty Images

Be extra vigilant with patients who have taken rosuvastatin for longer than three years.

The reason for the increased risk of glaucoma onset in participants with a longer duration of statin use wasn't clear, the authors noted. Still, they considered a few possibilities, including the mitochondrial toxicity of statins and an increased level of IOP. The significant link between long-term statin use and glaucoma onset could also be attributed to the confounding effect of hypercholesterolemia, which is the main indication for statins, they added.

"Finally, we also noticed that consultations with ophthalmologists and optometrists might be another potential confounder, with a higher frequency of eye-related visits in glaucoma patients in this study. Nevertheless, it cannot fully explain the association between statin use and glaucoma onset, especially in long-term statin users and rosuvastatin users," the authors wrote in their paper.

Considering the types of statins prescribed, participants who took rosuvastatin were more likely to have glaucoma. On the other hand, other statins, specifically simvastatin, pravastatin, fluvastatin and atorvastatin, weren't notably tied to the onset of glaucoma.

Perhaps not surprisingly, individuals who took higher statin doses appeared to be at greater risk for glaucoma onset compared with those taking lower doses.

Yixiong Y, Wang W, Shang X, et al. Association between statin use and the risks of glaucoma in Australia: a 10-year cohort study. Br J Ophthalmol. August 4, 2021. [Epub ahead of print].

IN BRIEF

Research has pointed to changes in the retinal pigment epithelium (RPE) as indicators of scleral growth and the possible presence of high myopia, but a recent study found that **changes in the RPE may even represent early progression of the disease**. Using SD-OCT images from 35 pediatric patients, researchers evaluated the relationship between the contour of the posterior eye and subsequent myopia progression.

They found that the baseline contour of the RPE and chorioscleral interface (CSI) did not predict disease progression; however, **axial length increased within the year if the baseline contour of the RPE was more prolate** than that of the CSI. Eyes with higher myopia usually had a more prolate posterior ocular contour.

"We investigated whether the one-year change in the posterior ocular contour correlated with the one-year progression of myopia and found that it was indeed significantly correlated with both the one-year change in axial length and spherical equivalent of refraction (SER)," the researchers wrote in their study. "Briefly, **a more prolate RPE contour was associated with greater elongation of axial length and a more negative SER**, which was similar to the relationship between the CSI contour and the axial length." **Observing these specific posterior ocular contour patterns could help you identify the patients at greater risk of myopia onset or progression.**

Xu S, Hu Y, Cui D, et al. Association between the posterior ocular contour pattern and progression of myopia in children: a prospective study based on oct imaging. Ophthalmic Physiol Opt. 2021;41:1087-96.

Caffeine Enhances Dynamic Visual Acuity

As you no doubt know from your morning coffee or tea, caffeine can give your brain a boost. In addition to improved cognition, acute caffeine ingestion has also been associated with improved visual function. Researchers analyzed this widely consumed psychostimulant's effects on dynamic visual acuity (DVA) and reported that it seems to be ergogenic.

DVA is defined as the ability to resolve fine details when there's relative motion between the target and the observer. It's important for real-world scenarios like ball sports, driving and piloting. This cognitive process is complex and highly sensitive to external factors such as diurnal variations, level of expertise, sleep deprivation and psychostimulants.

"Caffeine in particular is known to increase the velocity of rapid eye movements," the researchers explained in their paper. They noted that caffeine may also have an effect on contrast sensitivity, ocular aberrations and accommodation. "In addition, the ingestion of caffeine has been shown to enhance visual processing, facilitating the detection of visual stimuli and response preparation."

The placebo-controlled, double-blind crossover study was the first of its kind to measure acute caffeine effects on DVA. It included 21 low-caffeine consumers (average age: 22.5 years). On two different days and in a random order, each participant ingested either caffeine (4mg/kg) or placebo. Their DVA was measured after 60 minutes of ingesting the capsule.

The researchers found significantly greater accuracy for both the horizontal and random motion paths of DVA after caffeine ingestion. Caffeine intake was also associated with a faster reaction time for horizontally but not randomly moving targets. Study participants reported significantly higher levels of perceived activation after consuming caffeine in comparison with placebo.

"Caffeine is a central nervous stimulant that acts as an adenosine receptor antagonist," the researchers explained. "Our results showed that caffeine consumption increased subjective levels of activation, which agrees with many studies that provide that caffeine enhances alertness and feelings of wakefulness and energy."

Other studies have reported improved accuracy and response speeds after caffeine ingestion. "In the same line, we observed that caffeine reduced the time used to detect the moving objects in the DVA test. The faster reaction times observed under the effects of caffeine could be explained by the positive influence of caffeine on stimulus processing and decision-making. However, we just observed this effect (faster reaction time after caffeine intake) for horizontally moving targets, but not with the random motion path, which could be due to the shorter presentation time of the horizontal targets."

DVA depends on target velocity. The faster the target speeds, the worse the reaction times, for both caffeine and placebo. "Interestingly, our results showed that participants correctly identified smaller moving stimuli after caffeine ingestion compared with ingestion of the placebo," the investigators noted. "This finding was observed for both horizontally and randomly moving targets, which suggests that caffeine ingestion improved DVA." They added that eye movement and contrast sensitivity, both implicated in DVA performance, are also sensitive to caffeine.

Photo: Getty Images

In this study, those who ingested caffeine appeared to have a faster reaction time and improved performance of DVA.

From their findings, the researchers concluded that caffeine ingestion improves DVA performance. "The ingestion of caffeine could be recommended in tasks that have demanding attentional requirements and/or tasks that require good resolution of moving targets such as driving or dynamic sports."

Importantly, however, the behavioral response to caffeine depends on habitual intake. The researchers said it's likely that the effect of caffeine on DVA was more pronounced in this experimental sample.

Redondo E, Jiménez R, Molina R, et al. Effects of caffeine ingestion on dynamic visual acuity: a placebo-controlled, double-blind, balanced-crossover study in low caffeine consumers. Psychopharmacology. August 22, 2021. [Epub ahead of print].

IN BRIEF

Researchers recently investigated whether **primary open-angle glaucoma (POAG) is associated with autoimmune disease (AiD)** in ophthalmic surgery patients. They found a higher prevalence of AiD in POAG patients, confirming that an association between the two does exist.

The analysis revealed the overall prevalence of AiD to be **17.4% for POAG patients and 10.1% for controls**. A higher number of POAG patients had more than one AiD compared with controls (6.4% vs. 3.4%).

"The most prevalent AiD in the POAG group was rheumatoid arthritis (4.6%), followed by psoriasis (4.1%), non-infectious anterior uveitis (2.9%) and Graves' disease (1.7%)," the researchers wrote in their study. The researchers concluded, **"Having an AiD was associated with 2.62-fold increased odds of POAG** relative to controls."

Additional risk factors associated with POAG included older age, diabetes and non-white ethnicity. Systemic steroid use (either intravenous or oral) for longer than four weeks was also more significantly associated with POAG patients with AiD than without (16.7% vs. 2.8%).

These findings suggest that "autoimmunity should be explored further in the pathogenesis of POAG."

Lorenzo MM, Devlin J, Saini C, et al. The prevalence of autoimmune diseases in primary open angle glaucoma patients undergoing ophthalmic surgeries. Ophthalmology. August 3, 2021. [Epub ahead of print].

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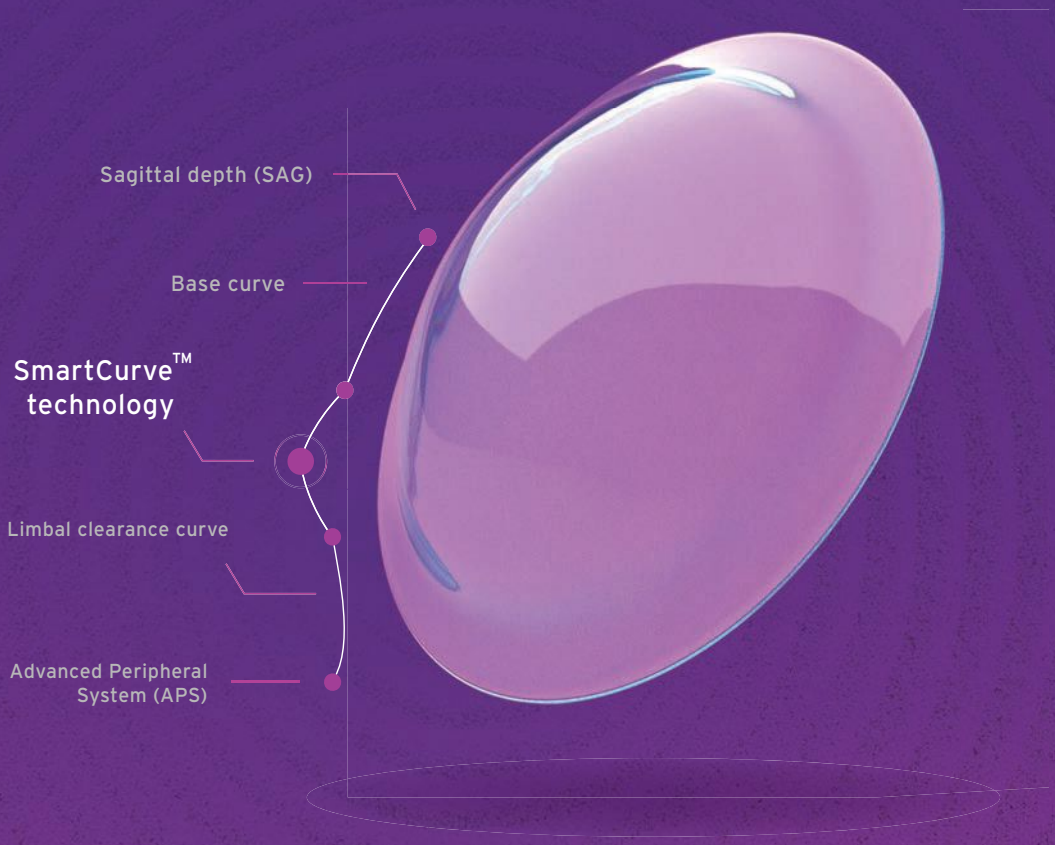

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Blue Light Filtering IOLs Don't Reduce AMD Risk

It's been suggested that pseudophakic eyes may experience greater retinal damage from intense near-ultraviolet light sources than crystalline lenses, resulting in an increased risk of age-related macular degeneration (AMD). Blue light filtering intraocular lenses (BF-IOLs) have been hypothesized to offer photoprotective benefits and are often used in an attempt to prevent retinal damage in cataract patients post-surgery. However, when researchers tested this theory recently, they found that BF-IOLs offer no tangible benefit over non-filtering IOLs in reducing patient risk for AMD development.

This Taiwanese study included 186,591 patients who had bilateral cataract surgery between 2008 and 2013 and were followed for up to 10 years. Of these patients, 11.3% had BF-IOL implants and 88.7% had conventional IOL implants. The researchers used propensity score matching (PSM) to balance the baseline characteristics between the two groups.

The incidence rate of AMD after cataract surgery was 11.59 per 1,000 person-years. After PSM, there was no difference in AMD incidence rates between patients with BF-IOLs and those with non-filtering IOLs. "The incidence rate of non-exudative AMD and exudative AMD (per 1,000 person-years) was 9.95 and 1.22 for the BF-IOL group, and 11.13 and 1.44 for the non-BF-IOL group, respectively," the researchers wrote.

However, before PSM, the AMD incidence in the BF-IOL group was lower than in the non-BF-IOL group. The researchers explained, "Patients in the BF-IOL group tended to be younger, with a higher income, more non-manual workers, more patients from urban and suburban areas and fewer chronic diseases. Patients with longer life expectancy and higher income were willing to pay extra money to implant premium IOLs, such as the BF-IOL, and because the patients were younger, the incidence of AMD was also lower."

So, why are BF-IOLs still being recommended to patients as though they offer an additional layer of protection? The study authors argue it's because there's not enough evidence to say whether they confer a benefit. A study on ophthalmologists' clinical practice patterns found that, "Although about 70% of respondents consider that there is little evidence in the protective effect of BF-IOLs on macular health, the most frequent reason for prescribing these lenses was a general safety measure against blue light."

If you and your cataract surgery patient want to take the extra precaution to preserve ocular health and vision, recommending BF-IOLs over conventional ones won't hurt, but this research doesn't confirm it'll help, either.

Lee JS, Li PR, Hou CH, et al. Effect of blue light-filtering intraocular lenses on age-related macular degeneration: a nationwide cohort study with 10-year follow-up. Am J Ophthalmol. August 16, 2021. [Epub ahead of print].

Ocular Surface Diseases in COVID-19 Patients

SARS-CoV-2 is mainly transmitted through respiratory droplets. Notably, some COVID-19 patients have ocular manifestations, including conjunctival hyperemia, chemosis, epiphora and increased secretions. However, the association between SARS-CoV-2 and ocular surface disease (OSD) is poorly described.

Looking to quantify this relationship, a recent study enrolled 20,157 participants from six districts of China. The team tested serum samples for immunoglobulin G and M antibodies against the SARS-CoV-2 spike protein and nucleoprotein using magnetic chemiluminescence enzyme immunoassays. SARS-CoV-2 prevalence (determined by IgG and IgM antibody results) for the entire study population was 0.9%. They also tested throat swabs for SARS-CoV-2 RNA.

Of the total subjects tested, 8.7% had some form of OSD, 62.3% had some form of eye problem excluding OSD and 29% had no ocular involvement of any kind. The ocular surface diseases found in the first group included dry eye, keratitis, conjunctivitis, pterygia, eyelid tumors, trichiasis, cysts, nevi and dacryocystitis. Non-OSD ocular conditions were not enumerated in the published study.

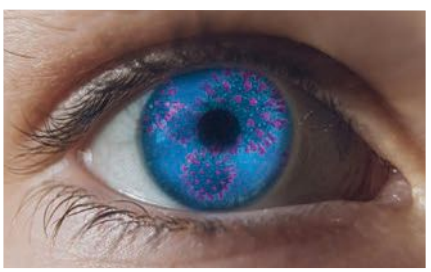

Photo: Getty Images

COVID-19 patients did have a higher prevalence of OSD, though researchers don't yet know if this was caused by the virus or the other way around.

The seroprevalence of SARS-CoV-2 was higher in the OSD population than the other two groups. The odds ratios of increased risk of susceptibility in the OSD group relative to the others were 1.47 and 2.17, respectively. Similar results were also observed with respect to sex, age, time and location.

It is still unknown whether the ocular surface conditions were manifestations of COVID-19 or risk factors for contracting the disease. Regardless, "Increasing awareness of eye protection during the pandemic is necessary, especially for individuals with OSDs," the researchers concluded."

Li S, Qiu Y, Tang L, et al. Association of ocular surface diseases with SARS-CoV-2 infection in six districts of China: an observational cohort study. Front Immunol. August 6, 2021. [Epub ahead of print].

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Dr. Michelle Hammond

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Dr. Reza Moradi

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Time of Year Influences IOP Level

Study shows pressure was highest around December and January and lowest in August and September.

Previous studies on IOP have found higher levels in winter compared with summer months. A recent study aimed to investigate the factors that influence changes in IOP, confirming seasonal fluctuations.

A total of 76,160 individuals were included, broken into three groups. Dataset A (3.9% of subjects) included patients examined with Goldmann applanation tonometry between 2011 and 2018. Dataset B (4.3% of subjects) was performed from 1999 to 2002 and the much larger dataset C (1.7% of subjects) consisted of all first visits during 2012 to 2017. Regression analyses assessed the relationship between the time of year and IOP.

IOP was highest in the colder months (December and January) and lowest in the warmer months (August and September). For dataset A, mean IOP was highest in December (15.7±3.7mm Hg) and January (15.7±3.8mm Hg) and lowest in September (14.5±3.1mm Hg). This suggests conventional quarterly analysis—January to March, for example—can "conceal time-of-year relationships due to inadequate statistical power and timing of IOP variation," the researchers noted in their *Journal of Glaucoma* paper on the study.

Multiple linear regression analysis, with a November to October reordering, detected an annual downward IOP trend, and an analysis of dataset B confirmed this. Fourier analysis on datasets A and B combined supported a 12-month IOP cycle for both eyes, and an analysis of dataset C showed a repeating pattern where IOP trended downward around April and then back upward around October.

"Although not discernible with the smaller datasets A and B, it was interesting that the larger dataset C suggested a 'plateau' of IOP that existed from about October to April, followed by an ensuing trough that developed from May to September," the authors noted in their study. "It is evident that we cannot make firm conclusions about this definitive pattern, but it would be interesting to see how even larger datasets from around the world may, or may not, mirror these observations."

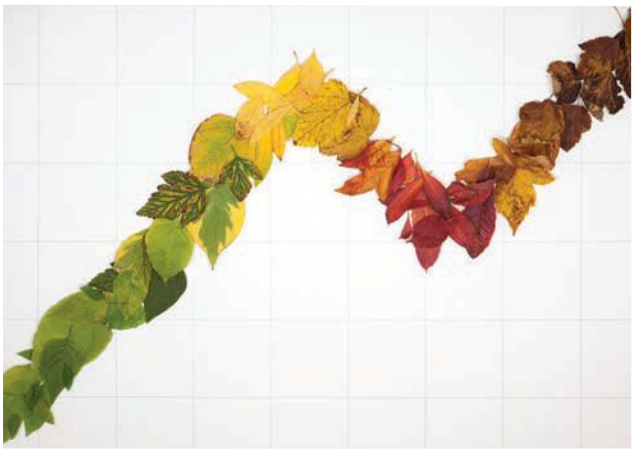

Photo: Getty Images

Winter seems especially hard on IOP, with elevated levels found in a recent study. This means that management might need to vary throughout the year.

Patients with IOP-lowering medication and a history of cataract surgery were excluded from datasets A and B, but findings from previous studies showed similar results in both healthy and glaucomatous eyes. The authors noted even small IOP differences "can be influential on glaucoma-related outcomes."

"Although time-related variation in IOP may be small, it is demonstrable with an adequately sized dataset and appropriate methods and may be worthy of further study relative to glaucoma control," the authors explained in their study. "It is noteworthy that the Early Manifest Glaucoma Trial showed that IOP lowering of just 1mm Hg yielded a 10% to 13% risk reduction in visual field deterioration. Further, the Ocular Hypertension Treatment Trial demonstrated that this amount of IOP lowering resulted in a 10% reduction in the risk of glaucoma conversion. To maintain a level reduction in risk of glaucoma progression, this suggests that, at least for certain patients, management might need to be altered during certain times of the year."

Exercise has been shown to have an effect on IOP, and with many forms of exercising more possible in the summer months for most people, that could have played a part in these results.

Additionally, seasonal IOP variation magnitude or trends may differ year-to-year depending on the different daily and monthly temperatures.

"For example, if the temperature in November is warmer or cooler in one year vs. another, does this have any effects on relative IOP measurements? We believe this may be an interesting question to explore but the effects may be extremely small and difficult to measure without careful adjustment for many other variables," the researchers wrote. "The increasing availability of very large datasets may make this a more feasible question to answer, but a potential problem may be that any effects could also be negated by spending more time indoors."

Morettin CE, Roberts DK, Newman TL, et al. Time-of-year variation in intraocular pressure. J Glaucoma. August 17, 2021. [Epub ahead of print].

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- Acquired ptosis may be associated with neurologic or orbital diseases such as stroke and/or cerebral aneurysm, Horner syndrome, myasthenia gravis, external ophthalmoplegia, orbital infection and orbital masses. Consideration should be given to these conditions in the presence of acquired ptosis with decreased levator muscle function and/or other neurologic signs.
- Alpha-adrenergic agonists as a class may impact blood pressure. Advise Upneeq patients with cardiovascular disease, orthostatic hypotension, and/or uncontrolled hypertension or hypotension to seek medical care if their condition worsens.
- Use Upneeq with caution in patients with cerebral or coronary insufficiency or Sjögren's syndrome. Advise patients to seek medical care if signs and symptoms of potentiation of vascular insufficiency develop.
- Upneeq may increase the risk of angle closure glaucoma in patients with untreated narrow-angle glaucoma. Advise patients to seek immediate medical care if signs and symptoms of acute narrow-angle glaucoma develop.
- Patients should not touch the tip of the single patient-use container to their eye or to any surface, in order to avoid eye injury or contamination of the solution.

ADVERSE REACTIONS

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

DRUG INTERACTIONS

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

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

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

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

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

1 INDICATIONS AND USAGE

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

2 DOSAGE AND ADMINISTRATION

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

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

4 CONTRAINDICATIONS

None.

5 WARNINGS AND PRECAUTIONS

5.1 Ptosis as Presenting Sign of Serious Neurologic Disease

Ptosis may be associated with neurologic or orbital diseases such as stroke and/or cerebral aneurysm, Horner syndrome, myasthenia gravis, external ophthalmoplegia, orbital infection and orbital masses. Consideration should be given to these conditions in the presence of ptosis with decreased levator muscle function and/or other neurologic signs.

5.2 Potential Impacts on Cardiovascular Disease

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

5.3 Potentiation of Vascular Insufficiency

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

5.4 Risk of Angle Closure Glaucoma

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

5.5 Risk of Contamination

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

6 ADVERSE REACTIONS

6.1 Clinical Trials Experience

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

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

7 DRUG INTERACTIONS

7.1 Anti-hypertensives/Cardiac Glycosides

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

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

7.2 Monoamine Oxidase Inhibitors

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

8 USE IN SPECIFIC POPULATIONS

8.1 Pregnancy

Risk Summary

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

Data

Animal Data

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

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

8.2 Lactation

Risk Summary

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

8.4 Pediatric Use

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

8.5 Geriatric Use

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

10 OVERDOSAGE

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

PATIENT COUNSELING INFORMATION

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

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Lab-grown Brains Show Eye Development

Stem cell-derived organoids treated with vitamin A grew little optic cups.

Scientists have managed to nurture small clumps of the human brain, giving them the ability to grow their own eyes, or at least two functionally integrated optic vesicles that respond to light. These tiny brains, called brain organoids, are self-assembled aggregates resembling the embryonic human brain in both cytoarchitecture and cell type.[1] They're grown from induced pluripotent stem cells (iPSCs), which can be differentiated into a variety of cell types *in vitro*.

Until now, brain organoids were thought to be "chaotic three-dimensional tissues" unable to follow the self-patterning rules of embryos. However, these brains spontaneously developed bilaterally symmetric optic vesicles in what would be the forebrain region.[2] While the eye structures are rudimentary, they have both a lens and a retina and can send signals to the brain tissue.

To encourage the brain organoids to grow eyes, Jay Gopalakrishnan, PhD, of the University Hospital Düsseldorf in Germany, and his team modified an iPSC protocol for differentiating into neural tissue by adding retinoic acid, a vitamin A derivative key for embryonic eye development, 20 days into the brain organoid's development.

The researchers reported that 72% of 314 brain organoids treated with 60nM of retinol acetate reproducibly attempted to assemble optic vesicles by around day 30, and visible eye structures developed within 60 days. The organoid's' development timeframe parallels that of human embryonic retinal development.

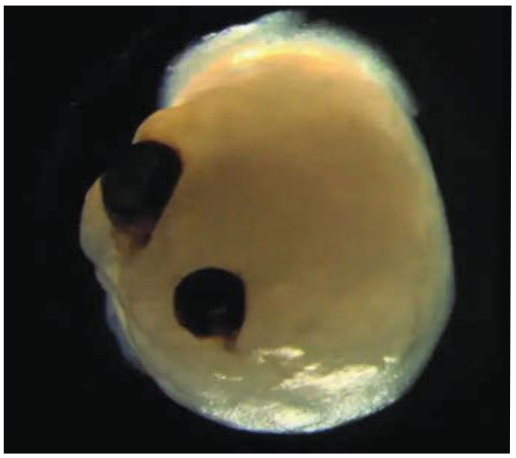

Photo: Dr. Elke Gabriel, PhD

This ambitious project may one day allow scientists to grow artificial retinas suitable for transplantation in blind and visually impaired patients.

Analysis revealed that the optic vesicles contained primitive corneal epithelial and lens-like cells, retinal pigment epithelial cells, retinal progenitor cells, axon-like projections and electrically active neuronal networks, showing retinal connectivity to the brain.

"In the mammalian brain, nerve fibers of retinal ganglion cells reach out to connect with their brain targets, an aspect that has never before been shown in an *in vitro* system," said Dr. Gopalakrishnan.

"Interestingly, various light intensities could trigger photosensitive activity of optic vesicle-containing brain organoids, and light sensitivities could be reset after transient photobleaching," he said. "Thus, brain organoids have the intrinsic ability to self-organize forebrain-associated primitive sensory structures in a topographically restricted manner and can allow interorgan interaction studies within a single organoid."

The researchers say the relatively short time to generate eye-like structures is crucial for the future of *in vivo* developmental biology because such a timeframe will allow for multiple experimental setups. Brain organoids containing optic vesicles will pave the way for "personalized organoids and retinal pigment epithelium sheets for transplantation," they said. They believe such organoids will help to model retinopathies emerging from early neurodevelopmental disorders.

1. Qian X, Song H, Ming G. Brain organoids: advances, applications and challenges. Development. 2019;146:8.

2. Gabriel E, Albanna W, Pasquini G, et al. Human brain organoids assemble functionally integrated bilateral optic vesicles. Cell Stem Cells. July 10, 2021. [Epub ahead of print].

IN BRIEF

Myopic choroidal neovascularization (mCNV) is a serious condition that leads to significant visual impairment and even legal blindness in some cases. Previous studies have reported **decreasing macular vessel density** in high myopia compared with normal eyes. A team in China analyzed the macular microvascular alterations in this patient population as well as the efficiency of anti-VEGF therapy for mCNV. They found that **mCNV eyes undergo several changes to the retina** and that **anti-VEGF was unable to completely eliminate CNV lesions in most cases.**

After analyzing OCT-A images from 123 patients divided into an mCNV group, a high myopia group and a normal group, researchers found that the logMAR **BCVA, superficial and deep vessel density and foveal avascular zone region density (FD) of the mCNV group were lower than the two other groups**. The superficial and deep vessel density and FD of the high myopia group were also lower than the normal group.

They also compared mCNV patients pre- and post-anti-VEGF therapy at one, two, three and six months and found improvements in logMAR BCVA and decreases in central macular thickness, area and flow area of CNV lesions.

The team reported a **50% mean reduction ratio of the lesions**. In two cases (5%), they observed lesion regression of 100%. They wrote that "the superficial and deep vessel density, area of foveal avascular zone, A-circularity index and FD didn't change in the mCNV group after anti-VEGF treatment."

Mao J, Shao Y, Yu J, et al. Macular density alterations in myopic choroidal neovascularization and the effect of anti-VEGF on it. Int J Ophthalmol. 2021;14(8):1205-12.

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1. CooperVision data on file 2021. Rx coverage database; 14 to 70 years. **2.** CVI data on file 2021. Based on number of prescription options available in the USA across all soft lenses reported by the 4 main manufacturers. **3.** Around the clock axis in 10° from Plano to -6.00DS in -0.75DC, 1.25DC and -1.75DC. **4.** CVI data on file 2021. Based on number of prescription options available in the USA across all soft 1-day toric lenses as reported by the 4 main manufacturers. **5.** CooperVision data on file 2020. Kubic masked online survey; n=404 US ODs who prescribe toric soft CLs.

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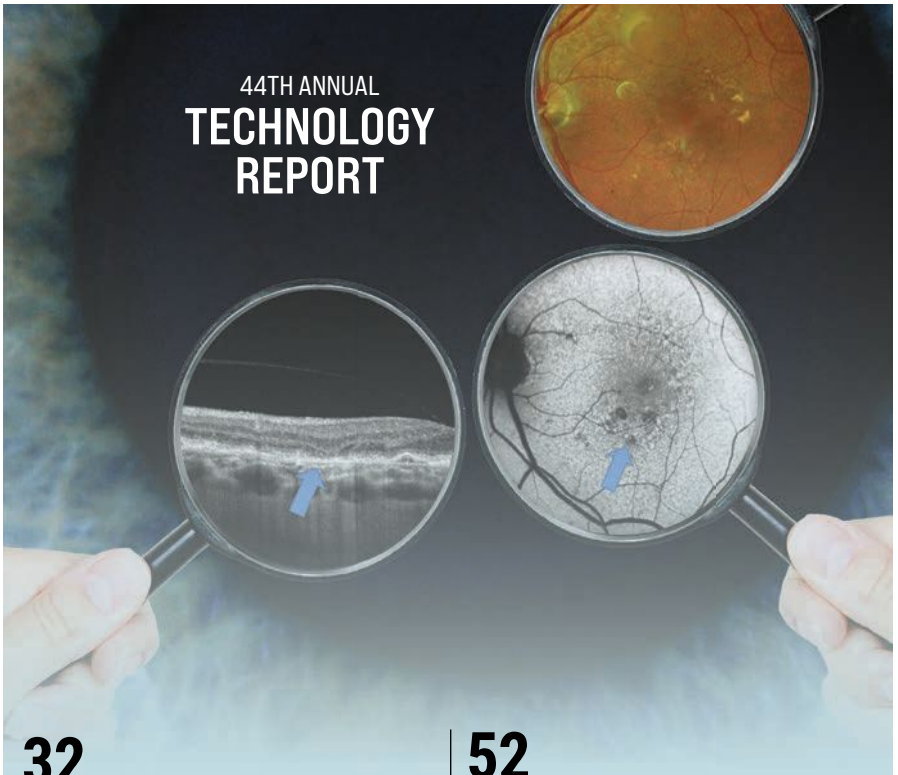
FEATURES

REVIEW OF OPTOMETRY • Vol. 158, No. 9 • SEPTEMBER 15, 2021



32
Take OCT To the Next Level
While you can get by using this technology for basic purposes, it offers so much more than meets the eye.
By Carolyn Majcher, OD, and Sylvester Cobbina, OD

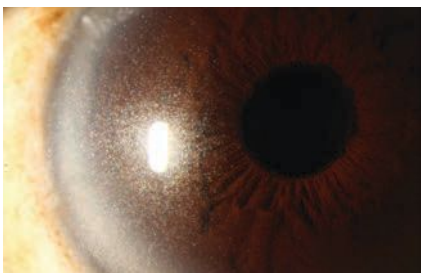
42
Retinal Imaging: See More Than Ever Before
Find out how advanced technology adds new layers of understanding that can improve care.
By Jessica Haynes, OD, James Williamson, OD, and Mohammad Rafieetary, OD

52
New Tools and Advanced Techniques for OSD Diagnosis
If you want to spot cases earlier and understand the patient's status more deeply, these options can augment your dry eye work-up.
By Cecelia Koetting, OD

60
Can at-home Monitoring Improve Optometric Care?
Doctors no longer make house calls, but devices can. These new tools can provide an abundance of data on how patients do outside the exam room.
By Catlin Nalley, Contributing Writer

64
The Eye/Kidney Connection
Be vigilant for the ocular presentations of oculoretinal syndromes and renal disease.
By Joseph J. Pizzimenti, OD, and Carlo Pelino, OD

74
Crystalline Keratopathy: Etiologies, Ocular Manifestations and Treatment Protocols
Learn to distinguish the various clinical signs and perform the proper testing to uncover the underlying issue and initiate treatment.
By Luanne Chubb, OD



84
When Your Patient Complains of Ocular Pain
Asking the right questions is key to successfully sizing up this common symptom.
By Alison Bozung, OD

—EARN 2 CE CREDITS

DEPARTMENTS

5

NEWS REVIEW

Clinical, legislative and practice development updates for ODs.

20

OUTLOOK

Color in Black and White

An AMA editorial aims to clarify how race and ethnicity are discussed in journal articles.

Jack Persico, Editor-in-Chief

22

LETTERS TO THE EDITOR

Feedback and ideas from the optometric community.

24

THROUGH MY EYES

Rethinking Profitability

With changes from COVID-19 come opportunity. Applying more technology can help position your practice toward success.

Paul M. Karpecki, OD

26

CHAIRSIDE

Chart It Like You Mean It

Writing it down doesn't automatically make it true, but, trust me, you're better off for it.

Montgomery Vickers, OD

28

CLINICAL QUANDARIES

Up in Flames

How to use appropriate but aggressive therapy.

Paul C. Ajamian, OD

30

CODING CONNECTION

Remote Testing's Impact

Monitoring patients at home can help you in the office.

John Rumpakis, OD, MBA

92

CORNEA AND CONTACT LENS Q+A

MRI CL Considerations

Impurities may interact with magnetic fields and cause problems for practitioners and patients alike.

Joseph P. Shovlin, OD

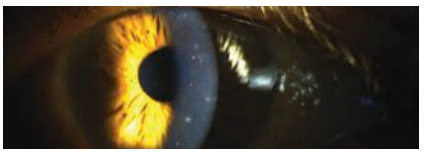
94

URGENT CARE

Tread Lightly

A traumatic tire explosion was to blame for hundreds of corneal foreign bodies.

Rachael Lloyd, OD



98

RETINA QUIZ

Persistent Problems

Poor ocular history contributed to this recent diagnosis.

Tania Patel, OD

102

THE ESSENTIALS

Tear Film Fighters

While often underappreciated, these important components serve to protect our eyes.

Bisant A. Labib, OD

105

PRODUCT REVIEW

New items on the market to improve clinical care and strengthen your practice.

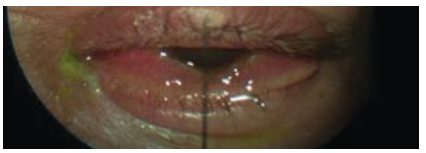
106

DIAGNOSTIC QUIZ

That's Swell of You

A complex medical history complicates assessment of a recent-onset red eye.

Saurin Patel, OD



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BY JACK PERSICO
EDITOR-IN-CHIEF
OUTLOOK

Color in Black and White

An AMA editorial aims to clarify how race and ethnicity are discussed in journal articles—and the medical profession broadly.

The institutions of American healthcare have begun to reckon with the potential for unintentional bias in the ways doctors are trained and how they communicate—with each other and with patients—about race and ethnicity.

Here in optometry, we've seen the creation of advocacy groups such as Black Eyecare Perspective and numerous task forces on diversity, equity and inclusion. Addressing the issue more broadly, the American Medical Association recently published guidance in *JAMA* on how authors of scientific articles could use terms with greater care to be more accurate about the populations studied while avoiding old habits that might come off as dismissive to underrepresented populations.

Some of the standard demographic categories used in medical research encompass literally billions of people with vastly heterogenous physical as well as cultural characteristics. With that in mind, just how useful is, for instance, "Asian" as a descriptor? There are obvious benefits to clinical care from recognizing racial and ethnic components of disease processes, and the AMA effort seeks to enhance the precision of that analysis.

The recommendations also try to walk the line between encouraging doctors to bring more nuance to their understanding of how race and ethnicity imbue a person with identity while taking care not to define individuals solely by those associations.

We have a lengthy news story about the AMA guidelines on our website and will cover the topic more fully in an upcoming print issue, but here are some key recommendations specific to the use of wording in publications:

- Using specific racial and ethnic categories is preferred over collective terms when possible. Categories included in groups labeled as "other" should be defined.
- When collective terms are used, merging of race and ethnicity as "race/ethnicity" is no longer recommended. Instead, "race and ethnicity" is preferred, with the understanding that there are numerous subcategories within these two areas.
- The general term "minorities" should not be used when describing groups or populations because it is overly vague and implies a hierarchy among groups. Instead, writers should include a modifier when using the word "minority" and should not use the term as a standalone noun for racial and ethnic minority groups and individuals.
- The nonspecific group label "other" for categorizing race and ethnicity is uninformative and may be considered pejorative.
- Authors are encouraged to provide greater detail about the distribution of multiple racial and ethnic categories.
- Racial and ethnic terms should not be used in noun form. Instead, the adjectival form is preferred.

We at *Review* will also look for ways to be more descriptive in the language used to discuss race and ethnicity as predisposing factors for potential health risks. Disparities in access to medical care among different populations should also be recognized and addressed by authors. These efforts would give us all a fuller picture of the state of healthcare today, and what to do about it. ■

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References: 1. Bron AJ, et al. *Ocul Surf*. 2017;15(3):438-510. doi:10.1016/j.tos.2017.05.011 **2.** Jones L, et al. *Ocul Surf*. 2017;15(3):575-628. doi:10.1016/j.jtos.2017.05.006

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LETTERS TO THE EDITOR

Feedback and ideas from the optometric community.

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Female ODs Can Thrive in Private Practice

As a female optometrist in private practice, I'd like to share my perspective on the topic of underrepresentation of women in this employment setting, as described in the June editorial, "Profit and Loss," and the July article, "Optometry at Work: The How, When and Where of Who Delivers Care."

On Instagram, I follow numerous ODs, many of whom are great, young influencers. Some female optometrists have posted about how women in our field make much less than our male counterparts, and I cannot help but wonder if it's because so few of us ladies are in private practice—or is it truly that males in commercial or ophthalmology practices simply earn more than females? Do women in private practice make less than men in private practice? This wasn't discussed in your article, and it's worth exploring. The overall trend in compensation for optometrists, and the wider workforce of the entire country, suggests this is likely.

Regarding the notion that the added burden of childcare makes self employment a harder proposition for us as female business owners, I think this is where we (as women) can be our own worst enemy if we aren't careful. I feel like I live somewhere between the woman who shouts, "I am woman, hear me roar—you *can* have it all!" and the woman who worries, "As a mother, your children should come first." I have embraced the realization that to be a mother and an optometry practice owner, both of those types of women can exist in me at the same time. Some days, one speaks louder in my head than the other.

Balancing family and professional obligations does not *have* to make self employment a harder proposition for us as women. In many ways, practice ownership puts me in charge of my schedule and thus allows me to be present at school events and volunteer opportunities, which only increases my patient load because I am known in my community thanks to parental participation. In another sense, this gig requires superior creativity—creating a school room in my practice, for example, for "virtual school" during Covid.

Do I have days where I have to take off due to my child being sick? Yes, if I cannot turn to a family member for their help. Do I sometimes have to bring my kids to work? Yes, which makes for interesting patient conversations when my two-year-old comes out of her make-shift office playroom into my crowded lobby and yells, "I just pooped in the potty!"

The hustle of it all is incredibly rewarding, and I am not sorry that I am doing it.

—Haley A. Perry, OD, Elite Eye Care
Asheville, NC

Optometry is Losing its Vision Care Touch

As a very experienced optometrist with decades of time in clinical practice (I just received acknowledgement from the AOA regarding 60 years of AOA membership) and a history of a multitude of optometric certifications, I am sad to say that it seems that the essence of optometry—our depth of knowledge of visual care—is being lost, with too many graduates entering into a profession which now looks like non-surgical ophthalmology, and only vaguely like optometry.

I recently retired from a practice that included behavioral optometry, developmental vision, adult and pediatric vision therapy, neuro-optometric rehabilitation, low vision care, contact lens care, ortho-K *and* full-scope medical optometry.

> **"We are doing well as a profession with regard to ocular pathology diagnosis and treatment, but we need to recover the important thing that separates us from ophthalmology."**

During my long career, I have had myriad young associates pass through my practice, virtually all of whom were vague about visual analysis, unsure about the binocular vision system and not interested in the relevance of the interaction of accommodation and convergence. They tended to ignore near-point retinoscopy, were clueless about PRA/NRA findings, were not really sure how to interpret blur/break/recovery binocular vision testing at distance or near and were also relatively clueless about when vision therapy vs. prism eyeglasses can help binocular vision issues, and they performed only perfunctory refractions.

I can picture the long-deceased Drs. Skeffington, the father of behavioral vision care, and Feinbloom, the pioneer of low vision optics, being upset at the loss of the visual essence of optometry.

An actively practicing colleague of mine recently joked that he hired a well-trained new graduate who knows lots about ocular pathology diagnosis and treatment, but who seemed to view a phoropter near-point rod "mostly as an effective back scratcher."

We are doing well as a profession with regard to ocular pathology diagnosis and treatment, but we need to recover the important thing that separates us from ophthalmology: our in-depth knowledge and treatment of the visual system.

—Errol Rummel, OD, FAAO, FIALVS,
Fellow Emeritus NORA and COVD
Jackson, NJ

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BY PAUL M. KARPECKI, OD
CHIEF CLINICAL EDITOR

Rethinking Profitability

With changes from COVID-19 come opportunity. Applying more technology can help position your practice toward success.

During the pandemic, practices that rely heavily on optical and contact lens sales did not seem to fare as well as those who had a significant medical eyecare component; among investors, the smart money was squarely on medical optometry. At the same time, internet sales of spectacles and contact lenses were well beyond record highs. The strong message here: maintain a quality optical and contact lens practice, yes, but continue to build your medical eyecare component. Judicious investments in technology can help steer you in that direction.

Retinal Imaging

These systems have advanced significantly over the years. It's a crowded field, with many new technologies competing for your attention—and investment dollars. New advances include higher resolution systems with confocal imaging (Eidon, iCare), ultrasound (DGH) and ultra-widefield imaging (Optos). OCT-A, though pricey, has allowed us to detect diabetic retinopathy early by revealing changes in the foveal avascular zone or peripheral non-perfusion.

But don't lose sight of the value of autofluorescence or *en face* OCT imaging. These will become especially important should we have an approved therapeutic agent for geographic atrophy/dry AMD. OCT has become a necessity for optimal management of glaucoma and macular disease, but it offers so much more, as you'll see in this month's article detailing the sophisticated uses of OCT including interpretation of well-controlled vs. progressing glaucoma and anterior segment applications. AMD can also be diagnosed early and managed for progression with dark adaptometry (Maculogix) testing.

In-office Procedures For DED

Practices with meibography also tend to offer in-office therapies. Over a decade ago, Drs. Donald Korb and Caroline Blackie discussed the dental model as an optimal future for optometry. The "brushing and flossing" element would be lid scrubs, of which there are many options; newer choices include the MYBO Clean daily eyelid cleaning brush, SleepTite light seals and Bruder mask hydrating compresses.

Like at a dental office, it all begins with imaging and advanced diagnostics, which might include not only meibography but also osmolarity and tried-and-true ocular surface staining. Our in-office procedures include blepharoexfoliation (Blephex), thermal pulsation (*Table 1*); intense pulsed light (Marco, Lumenis) and low-level light therapy (Marco).

Table 1. Thermal Systems for Meibomian Gland Expression

Device	Manufacturer
LipiView II	Johnson & Johnson
TearCare	Sight Sciences
Systane iLux	Alcon
Thermal 1-Touch	OcuSoft

Glaucoma Diagnostic Advances

Corneal hysteresis, which can be measured by the ORA device (Reichert) or adjusted with a CATS tonometer (Reichert), has become essential in my approach to glaucoma care. Another interesting metric is pupil testing; until the RAPDx device (Konan Medical), we didn't have the accuracy to measure and detect the subtle APDs present in almost all patients with early glaucoma.

I often debate starting a drop in many of my glaucoma suspects/borderline cases, but a high hysteresis and no APD gives me peace of mind to simply follow that patient. Years later, they remain non-progressing/suspects.

Turning the Tables

Since optometry conducts 85% of all comprehensive eye exams, we likely see the majority of patients with cataracts. Some ambitious optometric practices even go so far as adding an in-office operating room and hiring a cataract surgeon. There are companies that specialize in this, such as iOR Partners. For the right practices, it's generally very profitable.

Another important avenue for future revenue will be the sale of private-pay meds. It's anticipated that therapies for ptosis (RVL) and presbyopia (Allergan to Orasis) will be offered through eyecare offices. Now's the time to ensure your state allows you to participate.

Advances in technology lead to better diagnoses, which in turn lead to appropriate treatments—and it all sets your practice apart and insulates you from the cut-throat world of optical dispensing. Consider the lessons of COVID-19 as a clear direction for positioning your future practice. ■

1. Besada E, Frauens BJ, Makhlouf R, et al. More sensitive correlation of afferent pupillary defect with ganglion cell complex. J Optom. 2018;11(2):75-85.

About Dr. Karpecki

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

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BY MONTGOMERY VICKERS, OD

CHAIRSIDE

Chart It Like You Mean It

Writing it down doesn't automatically make it true, but—trust me—you're better off for it.

Put it in writing. If you don't put it in writing, it doesn't count. Simple as that. It doesn't exist. It was never even done, after all.

No, I didn't learn that in optometry school. My office manager, who also happens to be my wife, taught me that with a lot of patience on her part because, trust me, I am not someone who learns easily. It took me 10 years before I remembered to write down intraocular pressures (IOPs) on patient charts. I may or may not have considered IOPs as being pass/fail up until that point. If a pressure was high, I wrote it on the chart. If the pressure was acceptable, why waste the ink? That was my thinking at least.

My wife very often reprimanded me that if a patient's IOP was not written on the chart, it meant I did not run the test at all—even if I did, which I always did. I tried unsuccessfully to convince her time and time again that saving ink would also save the endangered Bengal tigers. She just would not listen.

So, ever the contrarian, one time I wrote on a patient's chart that their electromagnetic aura was within normal limits. I figured if it is true that not writing down the result of a test meant I did not do the test even if I did, then it would stand to reason that if I wrote down the results of a test, it meant I did the test even if I didn't.

This floated like a cinderblock. My wife was not impressed by my so-called wit. Suffice it to say, I never crossed her again. Though, I do often wonder if I ever whited out that entry on the poor patient's chart.

So, yes, write your findings down. And make sure you do the test in the first place, too.

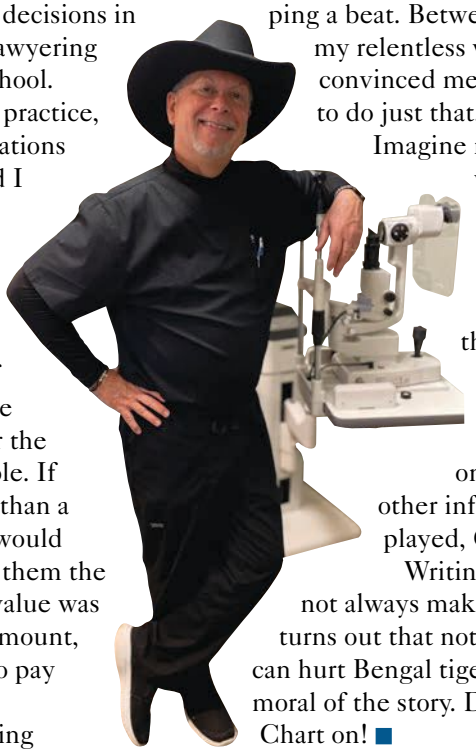
Now, the tricky part is this: putting it in writing does not always make it so. This is why they invented lawyers. I had always thought they invented lawyers so everyone would see that we all have something in common… *i.e.*, making fun of lawyers. My dad was a lawyer. I chose not to make fun of lawyers when he was around. This was one of my top 10 good decisions in life. After all, his lawyering put me through school.

When I sold my practice, against my protestations the buyers insisted I take inventory of every spectacle frame in the building, using the current listed wholesale value of each frame to come up with a value for the inventory as a whole. If the value was less than a certain amount, I would have to reimburse them the difference. If the value was greater than said amount, they would have to pay me the difference.

This was in writing and, therefore, pure and true, right? Wrong. The value was around $10,000 higher than expected (as I had warned my practice's buyers it would be when I wanted to just leave the frames out of the flat-fee deal), and to this day, I still have never seen a dime of the money they agreed to pay in writing. So much for being bound by your words. Unfortunately, it wasn't worth the $10,000 in legal fees it would cost to force them to pay. Them: 1. Me: 0.

But charts are different. My wife was right. Information has to be clearly stated on the chart.

I have previously mentioned that Dr. Bodie, the OD who hired me out of school and sold me his practice when he retired, had also insisted my charting be so complete that another doctor could pick up a chart and then start taking care of the patient in question without skipping a beat. Between Dr. B and my relentless wife, they've convinced me to work harder to do just that.



Imagine my surprise when, years later, I was looking through Dr. Bodie's old charts and found thousands of 7x9 notecards that only had the patient's name on them… no other information. Well played, George.

Writing it down does not always make it so. But it turns out that not writing it down can hurt Bengal tigers. That's the moral of the story. Don't forget it. Chart on! ■

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

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Up in Flames

Appropriate but aggressive therapy can make the difference.

Q A 34-year-old Hispanic male presented with a two-week history of a painful, red left eye. Exam reveals 2+ cells and flare, mild posterior synechia and an early cyclitic membrane. He's been using a topical antibiotic without improvement in symptoms. What's the diagnosis, initial treatment and potential work-up indicated if the condition recurs?

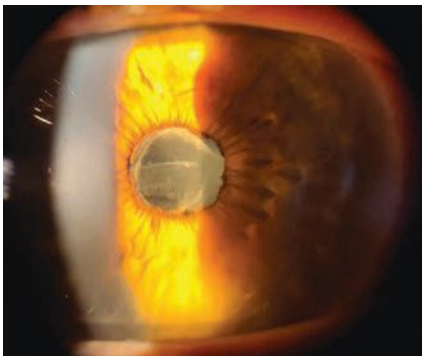
A "The presence of anterior chamber cells and flare, combined with the posterior synechiae, indicates iris involvement (iritis), while the cyclitic membrane suggests ciliary body involvement as well (cyclitis)," says Chris Wroten, OD, of Bond-Wroten Eye Clinic in Louisiana. "Thus, we would classify this as an iridocyclitis and treat it more aggressively than we would with a run-of-the-mill iritis."

"Don't forget to rule out infectious and traumatic etiologies, including chlamydia and thyroid eye disease, as potential causes of recalcitrant, monocular conjunctivitis," Dr. Wroten notes. This patient reported good systemic health while denying contact lens wear, previous eye surgery, known trauma or potential foreign bodies.

Treatment

Dr. Wroten recommends prescribing Durezol (difluprednate 0.05%, Novartis) every one to two hours while awake, with atropine 1% twice daily and 10% phenylephrine in-office to break the synechia. Because pharmacies don't stock these meds, have some on hand and send them home with them, to be used QID if the patient has no contraindications.

"Steroids need a couple of days to control inflammation, so don't be tempted to believe treatment isn't working," he advises. Control any nasolacrimal drainage issues and prolong ocular contact at the same time by inserting collagen plugs in the affected eye(s). Add other medications such as topical or oral non-steroidal anti-inflammatories if the inflammation does not resolve quickly enough.



Only when the cells and flare from uveitis resolve dramatically should ODs begin a slow taper of the steroid to prevent rebound.

For complex noninfectious cases, consider either oral prednisone or sustained-release steroid implants, such as Dextenza (0.4mg dexamethasone ophthalmic insert, Ocular Therapeutics) and Ozurdex (dexamethasone intravitreal implant; Allergan).

One can consider subconjunctival, sub-Tenon's or intravitreal steroid injections, but recognize that most chronic steroid therapies may cause elevated intraocular pressure, posterior subcapsular cataracts, opportunistic infections and delayed healing post-op.[1,2]

Lab Work and Follow-up

If the uveitis is bilateral, chronic, recurrent or recalcitrant, or additional clinical signs emerge, Dr. Wroten suggests starting with a complete blood count with differential to rule out infectious etiologies, in addition to tests for general systemic inflammation as well as other targeted labs based on the patient's age and clinical history.

Uveitis occasionally precedes positive lab tests by months to even years. "If suspicion of systemic etiology persists, repeat labs at appropriate intervals even if a previous work-up was negative," Dr. Wroten says.

If signs and symptoms are improving satisfactorily and the synechia are broken at the one-week follow-up, keep the patient on the aggressive steroid plan and gradually taper the dilating agents. Only when the cells and flare resolve dramatically should you begin a slow taper of the steroid over a number of weeks. Tapering too quickly will result in rebound, and the drops will need to be increased again for a longer period of time.

In cases where steroids are contraindicated or ineffective, consider systemic immunosuppressant medications such as methotrexate, mycophenolate mofetil and azathioprine.[3]

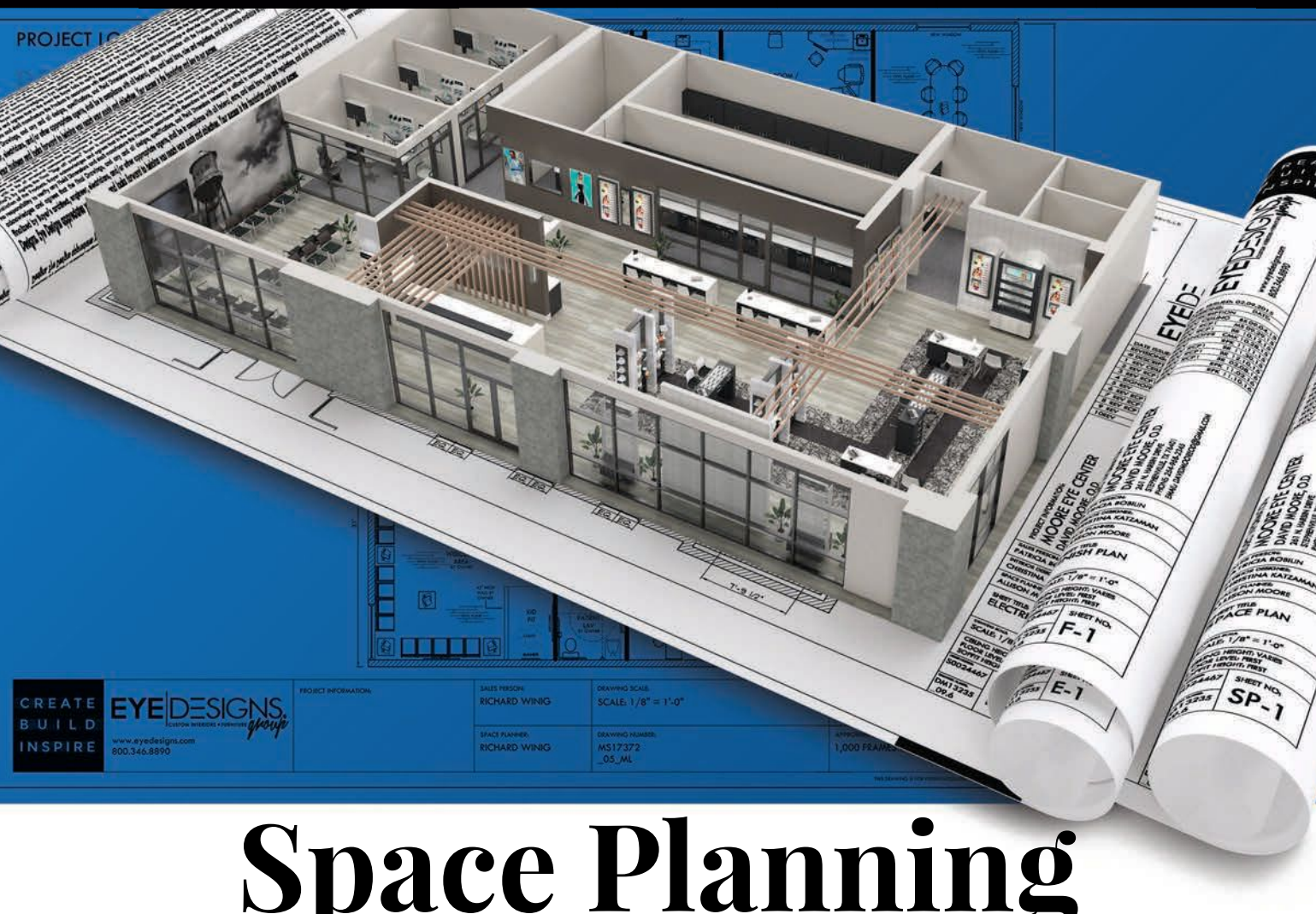
"Our goal is to restore a white, quiet eye with good vision and without long-term inflammatory sequela, so appropriate but aggressive therapy early on is my preference," Dr. Wroten says. "If the condition is unresponsive to common therapies, consider a consult to a uveitis or retinal specialist for consideration of other therapies." ■

1. Food and Drug Administration. Dextenza (dexamethasone ophthalmic insert) 0.4 mg,for intracanalicular use. www.accessdata.fda.gov/drugsatfda_docs/label/2019/208742s001lbl.pdf. Accessed August 17, 2021.

2. Food and Drug Administration. Ozurdex (dexamethasone intravitreal implant). www.accessdata.fda.gov/drugsatfda_docs/label/2014/022315s009lbl.pdf. Accessed August 17, 2021.

3. Bethke, W. The latest treatment approaches for uveitis: don't hesitate to use immune-suppressing medications in severe patients, surgeons say. Rev Ophthalmol. 2017;24(8):35-9.

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



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BY JOHN RUMPAKIS, OD, MBA
CLINICAL CODING EDITOR

Remote Testing's Impact

Monitoring patients at home can help you in the office.

One aspect of at-home testing and monitoring not often spoken about is its effect on evaluation and management office codes when the patient either presents to the office or if you are using a 992XX code for a telemedicine-based encounter. However, there are some caveats and rules that we must consider when applying this paradigm—especially when the testing is done by an Independent Diagnostic Testing Facility (IDTF).

Medical Decision-making

Determining your evaluation and management (E/M) code level, whether with a new or established patient, uses the current definition of total time or medical decision-making (MDM). Total time consists of nine elements:

- Preparing to see the patient.
- Taking additional or reviewing previously taken history.
- Performing your medically appropriate exam.
- Counseling/educating patient, family, caregiver.
- Ordering medication, tests or procedures.
- Referring and communicating with other health care providers.
- Documenting the medical record.
- Independently interpreting test results and communicating them.
- Coordination of care.

When using MDM, keep in mind the three categories to score its level:

- Number and complexity of problems addressed.
- Amount and/or complexity of data to be reviewed and analyzed.
- Risk of complications and/or morbidity.

Diagnostic Testing

Let's use a common remote test, the ForeseeHome by Notal Vision, as an example of an at-home monitoring technology used for age-related macular degeneration. Notal has established a Category III code for its test and has established itself as an IDTF.

> **Understand that the physician/IDTF roles are pivotal in being able to use the information in coding for your E/M visits.**

An IDTF only accepts patients referred by an attending physician who is providing a consultation or treating a beneficiary for a specific medical problem and who uses the results in the management of the patient's specific medical problem. The IDTF bills for and gets paid for its testing directly from the carrier—an important distinction.

The Category III code that Notal uses as an IDTF is:

- 0379T: Visual field assessment, with concurrent real time data analysis and accessible data storage with patient-initiated data transmitted to a remote surveillance center for up to 30 days; technical support and patient instructions, surveillance, analysis and transmission of daily and emergent data reports as prescribed by a physician or other qualified health care professional.

Understand that the physician/IDTF roles are pivotal in being able to use the information in coding for your E/M visits. The physician orders the tests but is not responsible for administering it, nor getting paid to interpret it. That is the role of the IDTF. Whether we are using time or MDM to score our encounter, it is vital that we are not getting reimbursed in any way for the remote monitoring activity.

Review the Data

When using time to score the E/M visit, the physician can measure the component of "independently interpreting test results and communicating them" to the patient or responsible caregiver. This information can also be entered into the medical record and that can add additional time. If using MDM, this would impact our second category of "amount and/or complexity of data to be reviewed and analyzed" under the bullet point of "review of the results of each unique test."

Reviewing data from a test that we have not been paid for by other means is an important differentiator here. For example, if a visual field had been ordered and performed by the practitioner, the interpretation of that test would already be included in the reimbursement of that specific test and could not be used to add to the E/M visit.

Technology will continue to advance, and patient convenience is going to be a critical driver along with the clinical merits. Understanding your role in independently interpreting test results that are done by an IDTF or other practitioners not only plays a significant part in patient management but also contributes to the amount of time spent or the amount and complexity of data required, thus impacting the coding of your E/M office visit. ■

Send your coding questions to rocodingconnection@gmail.com.

About Dr. Rumpakis

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

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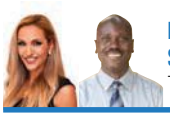
TAKE OCT TO THE NEXT LEVEL

While you can get by using this technology for basic purposes, it offers so much more than meets the eye.



BY CAROLYN MAJCHER, OD, AND SYLVESTER COBBINA, OD
TAHLEQUAH, OK

More and more optometrists are investing in optical coherence tomography (OCT) for their practices. With the increase in adoption of this technology comes the question of how to expand its uses from conventional and common to sophisticated but still practical. This article looks to explore a few ways ODs can ramp up their OCT usage.

OCT-A in Diabetic Retinopathy

OCT angiography (OCT-A) is a noninvasive, dyeless imaging technology that provides volumetric maps of the retinal and choroidal vascular systems, as well as information on blood flow. Because it employs OCT imaging technology, vascular information can be viewed alongside or superimposed on structural data. This allows for precise localization of vascular lesions and for structural correlations to be drawn.

As diabetic retinopathy predominantly affects inner retinal circulation, OCT-A preset *en face* displays of interest include the superficial and deep capillary plexuses as well as the vitreous or vitreoretinal interface. Retinal nonperfusion, apparent on the superficial and deep capillary enface displays, is often most prominent in the midperiphery and is best visualized with montage angiography imaging (*Figure 1B*). The greater the degree of nonperfusion, the greater the vascular endothelial growth factor (VEGF) release and the higher the risk of current or future proliferation.

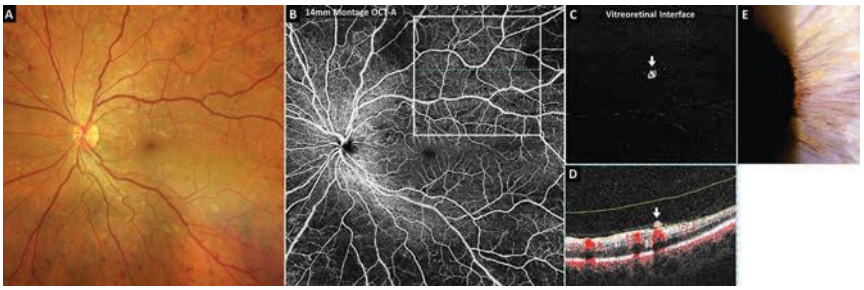


Fig. 1. OCT-A of subtle proliferative diabetic retinopathy.

Montage OCT-A is incredibly useful in detecting small areas of preretinal neovascularization in eyes with diabetic retinopathy and, therefore, aids in the earliest diagnosis of proliferative-stage disease possible. The volumetric dataset provided with OCT-A imaging also applies to montage images so that neovascularization can be easily isolated and identified using the preset vitreoretinal interface or vitreous enface display. The vitreoretinal interface includes data from the vitreous just anterior to the retina. In a healthy eye, it should be void of signal, or black. Areas of preretinal neovascularization will appear hyperreflective (*Figure 1C*).

Figure 1 includes OCT-A imaging in a treatment-naïve eye with 20/20 acuity. Color photography and funduscopy revealed what was initially thought to be very severe nonproliferative diabetic retinopathy (*Figure*

About the authors

Dr. Majcher is an associate professor and the director of residency programs at the Oklahoma College of Optometry Northeastern State University in Tahlequah, OK. She is a paid speaker and consultant for Regeneron Pharmaceuticals and Carl Zeiss Meditec.

Dr. Cobbina is a family practice/ocular disease resident at the Oklahoma College of Optometry Northeastern State University and an Army optometrist. His contribution to this article does not represent the views of the US Army, the Department of Defense or its components.

1A). A 14mm x 14mm montage OCT-A superficial capillary plexus scan revealed significant midperipheral retinal nonperfusion (*Figure 1B*). The superior temporal 8mm OCT-A vitreoretinal interface image revealed a small preretinal neovascular membrane (*Figure 1C*). The corresponding OCT cross-sectional B-scan with blood flow overlay in red highlighted the preretinal neovascularization (white arrow) which rests on the retinal surface and appears to be clinging to a shallowly detached posterior hyaloid membrane (*Figure 1D*). This eye also exhibited subtle iris rubeosis (*Figure 1E*).

Recent research suggests subtle neovascularization is often missed in eyes clinically graded as severe nonproliferative but can be detected with montage OCT-A. Researchers performed montage OCT-A on 27 eyes clinically graded as having nonproliferative diabetic retinopathy.[1] Of the seven eyes originally graded as severe nonproliferative, montage OCT-A detected neovascularization in four (57%). Of note, the neovascularization would have been missed in two of these eyes if only a single 6mm x 6mm angiography macular scan was done.

There is a shifting paradigm toward earlier treatment of diabetic retinopathy, even in the nonproliferative stages, with anti-VEGF agents to prevent vision-threatening complications. Therefore, accurate staging and early detection of proliferative disease is of the upmost importance so that patients can be referred appropriately.

OCT-A in Retinal Venous Occlusion

Vision decline in retinal venous occlusive disease without neovascularization may be due to macular edema, macular ischemia and/or macular hemorrhage. Most clinicians are familiar with the utility of OCT in the evaluation of macular edema; however, it is important to also be aware that macular ischemia apparent via OCT-A may also be present and contributing to vision loss.

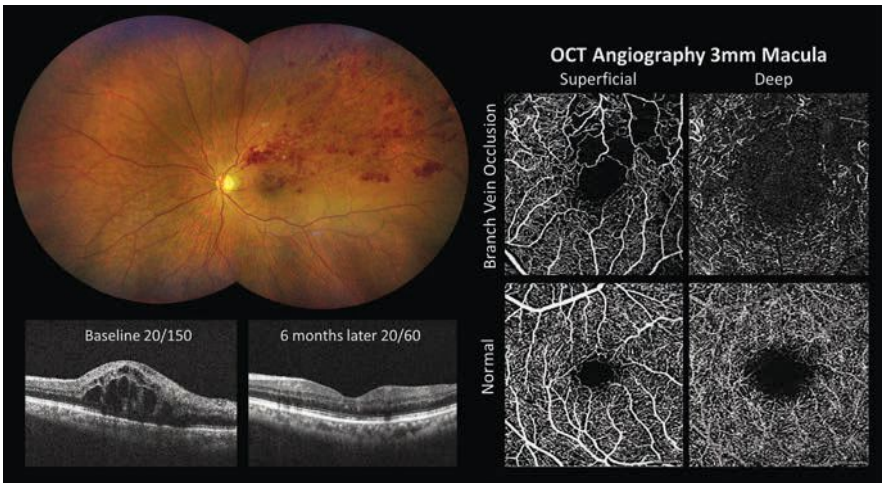


Fig. 2. OCT-A detection of macular ischemia in branch retinal vein occlusion.

The prognosis for visual improvement is guarded in eyes with both macular edema and macular ischemia, the latter of which is thought to be mostly non-reversible despite appropriate anti-VEGF therapy. Both macular ischemia and retinal nonperfusion are typically invisible with fundoscopy alone unless acute or extensive. OCT-A, however, readily demonstrates retinal and macular nonperfusion. Macular ischemia is evident on OCT-A through enlargement of the foveal avascular zone.

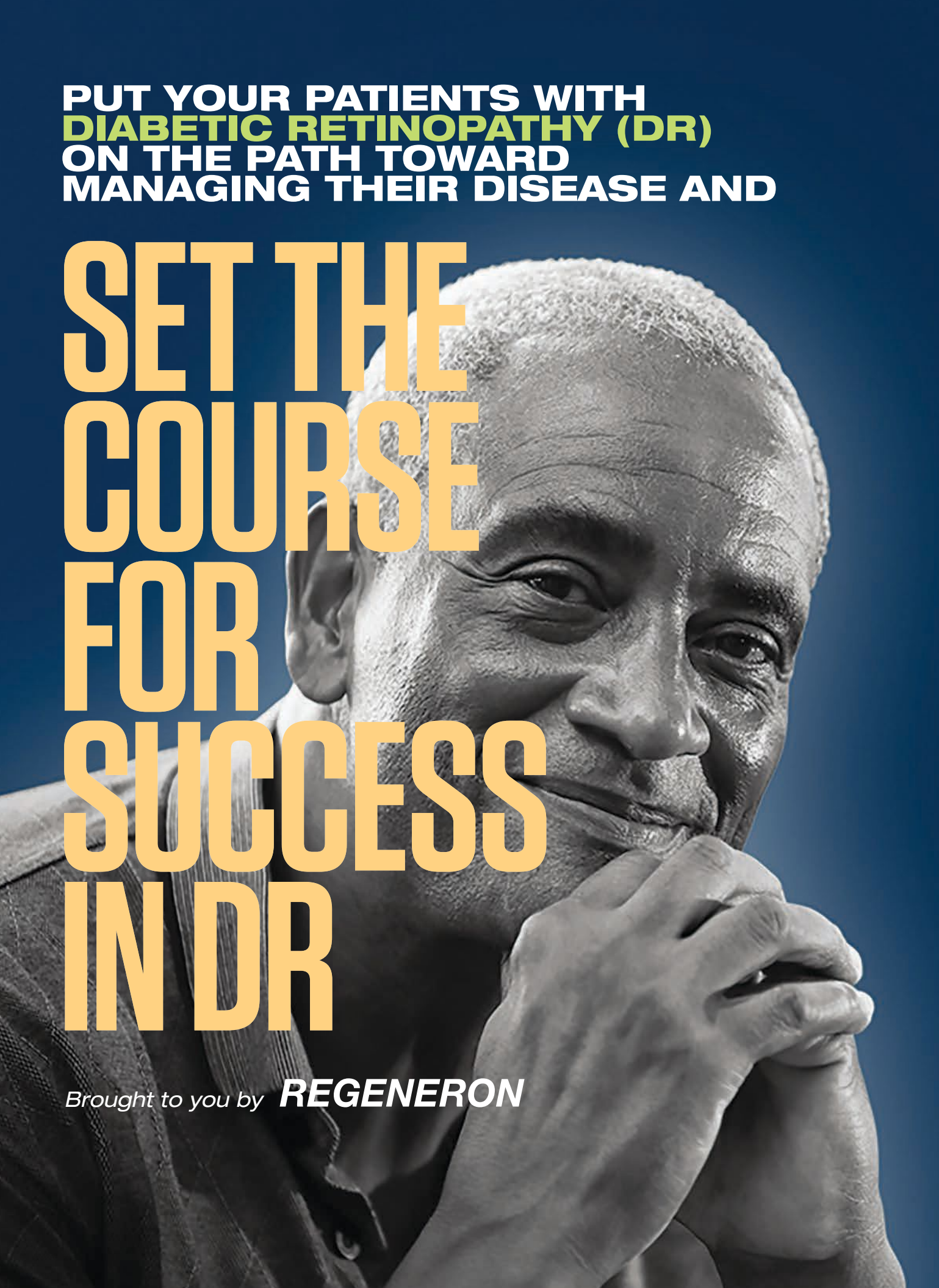
Figure 2 shows OCT-A imaging in an eye with macular edema and macular ischemia secondary to superior temporal branch retinal vein occlusion. Significant macular edema was present at baseline and vision improved from 20/150 to 20/60 following six months of anti-VEGF treatment. A 3mm macular OCT-A scan suggested that the reduced vision was due to macular ischemia. The superficial capillary plexus enface image showed an enlarged foveal avascular zone with irregular contour and retinal nonperfusion within the superior macula.

Another clinical application of OCT-A in retinal venous occlusion is the detection and quantification of retinal capillary nonperfusion using montage imaging.[2] Estimating the degree of retinal nonperfusion is useful for differentiating ischemic from nonischemic occlusions and predicting which eyes are at greatest risk for developing neovascularization. Ischemic branch retinal vein occlusions, or those with greater than five disc areas of nonperfusion, are at high risk of developing posterior segment neovascularization such as neovascularization of the disc or retina elsewhere.[3] Similarly, eyes with ischemic central retinal vein occlusions, defined as having 10 or more disc areas of nonperfusion, are at high risk for developing iris and angle neovascularization as well as subsequent neovascular glaucoma.[4]

OCT in Drusen Subclassification

In the evaluation of age-related macular degeneration (AMD), OCT interpretation should not merely be limited to the identification of drusen. Determining the type of drusen, through the assessment of its location and morphological characteristics, is crucial in clinical decision-making. For instance, hard drusen in the macula, which we are familiar with as a risk factor for AMD, can be associated with other types of drusen that carry more aggressive pathological risks for conversion of nonexudative AMD to its exudative form.

In *Figure 3*, fundus photography gives a general presentation of drusen with pigmentary changes in the macula. Careful review of OCT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According to the AOA, you should refer patients with[3]:

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

Monitor your patients with DR[3,4]

The AOA recommends frequent monitoring of patients[3]

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

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

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AOA = American Optometric Association.

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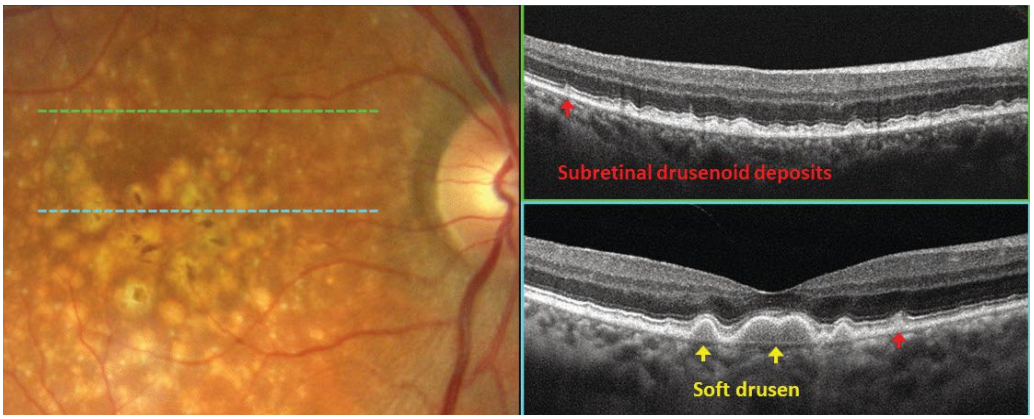


Fig. 3. OCT subclassification of drusen in AMD.

scans revealed distinct subtypes of drusen. Subretinal drusenoid deposits (SDD, red arrows), also referred to as reticular pseudodrusen, can be identified below the sensory retina and are associated with an intact retinal pigment epithelium (RPE). Another subtype of drusen, soft drusen (yellow arrows), is associated with drusenoid RPE detachments and is characterized by hyporeflective areas beneath the mounds of RPE. When these two types of drusen are present, reflecting compromise of the photoreceptors and RPE, the risk of conversion to exudative AMD increases.

In a study of patients with neovascular AMD in one eye, the five-year incidence of neovascularization in the contralateral eye with SDDs was 25.7%, and with soft drusen, it was 46.2%.[5] The presence of both SDDs and soft drusen resulted in an incidence of 76.4%.[5] It is therefore key to distinguish these types of drusen on OCT scans for patient education and to set appropriate monitoring intervals.

OCT-A in Macular Degeneration

This technology allows for noninvasive assessment of the retinal and choroidal vasculature and is therefore useful in detecting, classifying and morphologically characterizing neovascular membranes that may complicate diseases of the outer retina such as AMD. OCT-A aids in earlier detection of subretinal and choroidal neovascular membranes (CNV) in AMD, allowing for prompt treatment and vision preservation.

Conventional structural OCT provides valuable information regarding the presence of CNV membranes by allowing the clinician to observe secondary manifestations of CNV, such as fluid accumulation and retinal and subretinal thickening. The addition of OCT-A provides another tool for CNV detection since it allows the membrane itself to be seen.[6,7] There are various enface preset displays that are specifically designed to aid in the detection of subretinal and CNV membranes, including the outer retina and the outer retina choriocapillaris whose segmentation boundaries include a combination of photoreceptors, RPE and the choriocapillaris.

Figure 4 shows OCT and OCT-A imaging in an eye with new-onset exudative neovascular AMD and acuity of 20/25^{-1}. Fundus examination revealed multiple macular drusen, non-central geographic atrophy and grayish thickening of the temporal fovea with adjacent subretinal hemorrhage (*Figure 4A*). Structural OCT of the superior macula (*Figure 4B*) and through the center of the fovea (*Figure 4C*) revealed outer retinal thickening and a hyperreflective subretinal mass (yellow arrow) with adjacent subretinal fluid. A 3mm OCT-A scan of the macula outer retina choriocapillaris enface preset (*Figure 4D*) demonstrates a well-formed neovascular complex; the corresponding segmentation boundaries are shown in yellow (*Figure 4E*).

In some cases, OCT-A may reveal subclinical neovascular membranes before the onset of exudation, a condition referred to as nonexudative or quiescent CNV. A nonexudative CNV membrane is a well-defined neovascular complex visualized with OCT-A without exudative features via ophthalmoscopy such as exudate or hemorrhage. Additionally, no fluid is present via structural OCT imaging and the CNV membrane does not leak with intravenous fluorescein angiography.[8] This lesion carries with it a substantial risk for conversion to the exudative form of the disease; therefore, these are eyes that need to be monitored very closely. Research suggests that eyes with nonexudative CNV have a nearly 15-fold increased risk of subsequently developing exudation.[9-11]

OCT-A in Macular Telangiectasia Type 2

Macular telangiectasia type 2 (MacTel2) is characterized by bilateral perifoveal telangiectatic capillaries as well as mild and slowly progressive vision loss in the fifth to sixth decades of life.[12] Prevalence is estimated to be 0.1% in people 40 years and older, and both environmental and genetic factors are thought to play a role in the pathogenesis.[12] Fundus features include a prominent reddish foveal appearance with parafoveal (especially temporal) retinal opacity, crystalline deposits, blunted right-angle venules and hyperpigmented plaques.[12] These findings can be incredibly subtle in early stages of the disease; therefore, MacTel2 is commonly misdiagnosed as a macular hole, macular degeneration or central serous chorioretinopathy.

Structural OCT and OCT-A are invaluable tools that aid in the challenging diagnosis of MacTel2. Structural OCT often reveals early loss of the foveal photoreceptors eventually resulting in severe outer retinal thinning and atrophy with possible internal limiting membrane draping.[12] Loss of tissue may result in pseudocystic formation or dark subfoveal atrophic cleft development. OCT-A further assists in diagnosis by revealing bilateral dilated telangiectatic perifoveal capillaries, which are typically most prominent within the deep capillary plexus of the temporal region, enlargement of the foveal avascular zone and blunted right-angle venules.[13,14]

Figure 5 shows imaging of a patient in his mid-fifties with MacTel2 and 20/20 vision. Structural OCT of the right and left eyes shows loss of the photoreceptor ellipsoid zone and outer nuclear layers within the temporal fovea (*Figures 5B and 5E*). A 3mm OCT-A scan of both eyes demonstrates enlargement of the foveal avascular zone within the superficial capillary plexus as well as dilated telangiectatic capillaries within the deep capillary plexus, especially temporally (*Figures 5C and 5F*). The outer retina choriocapillaris enface display of the right eye demonstrates a hyperreflective transmission artifact within the temporal fovea due to loss of the outer retina with no evidence of subretinal neovascularization.

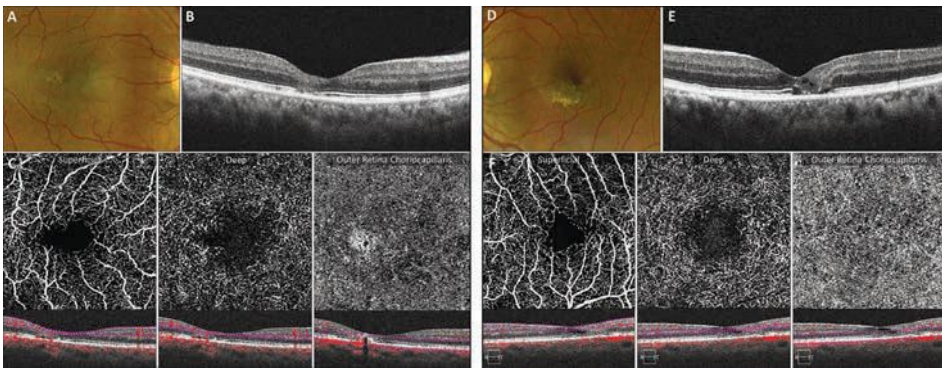


Fig. 5. OCT-A abnormalities in early MacTel2.

OCT-A also aids in the early detection of subretinal neovascularization, which is a more severe and potentially treatable complication of MacTel2.[15] Eyes with subretinal neovascularization tend to have a poorer visual prognosis; early detection and treatment is imperative.[12,15]

EDI-OCT in Choroidal Tumors

Advances in OCT technology such as swept-source OCT and enhanced-depth imaging OCT (EDI-OCT) allow for deeper imaging down into the choroid and are invaluable in the assessment of high-risk choroidal tumors. EDI-OCT penetrates an additional 500µm to 800µm deeper compared with traditional OCT imaging and can be easily performed with just the click of a button or check of a box on most commercially available OCT models with newer software upgrades.[16]

It is important to use the OCT caliper tool to measure choroidal tumor thickness from the posterior aspect of the RPE down to the posterior border of the tumor (often immediately adjacent to the hyperreflective inner sclera) through the region where the tumor appears to be thickest.[17] Combining EDI-OCT with image averaging (condensing all raster lines down into one line) will provide the highest-resolution imaging of the choroid and the best chance of identifying the posterior border of a tumor. Prior studies suggest that ultrasonography significantly overestimates tumor thickness compared with EDI-OCT, with one study finding that small choroidal melanoma tumor thickness was overestimated by 55% on ultrasonography compared with EDI-OCT.[17]

Differentiating choroidal nevus from small choroidal melanoma and predicting which choroidal nevi are at greatest risk for malignant transformation has long been a diagnostic challenge. The mnemonic "To Find Small Ocular Melanoma" helps us remember the high-risk features predictive of malignant transformation.[18] This well-known mnemonic was recently updated to "To Find Small Ocular Melanoma Doing IMaging" to incorporate risk factors visualized with multimodal imaging modalities including OCT.[19] The "T" stands for

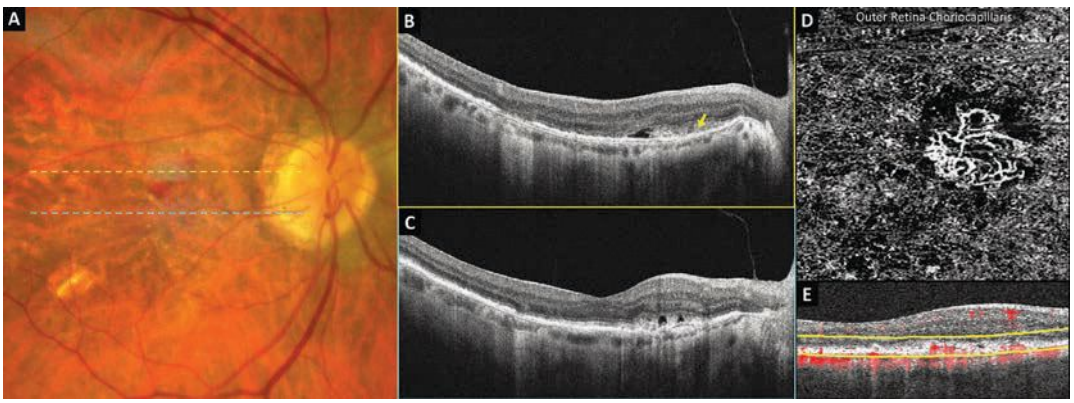


Fig. 4. OCT-A detection of neovascular AMD.

tumor thickness greater than 2mm with ultrasonography.[19] As previously mentioned, however, it is important to be aware that a 2mm ultrasound does not equal a 2mm OCT, since ultrasound overestimates tumor thickness.[19] A more reasonable tumor thickness cutoff for suspicion if using EDI-OCT is approximately 890µm.

The "F" stands for subretinal fluid via OCT imaging, and the "S" stands for symptomatic vision loss of 20/50 or worse.[19] The "O" is for orange pigment or lipofuscin deposition that is best visualized with fundus autofluorescence (FAF), and "M" stands for melanoma acoustic hollowness, or a dark internal appearance, via ultrasound.[19]

Lastly, the "Doing IMaging" stands for a diameter greater than 5mm as measured with widefield or ultra-widefield color fundus photography.[19]

Figure 6 shows FAF and EDI-OCT imaging in an eye with a suspicious pigmented choroidal tumor being monitored closely by ocular oncology. Upon FAF imaging, most of the tumor is heterogeneously hypoautofluorescent (*Figure 6B*). A strong hyperautofluorescent signal is present on the most peripheral aspect of the tumor and likely signifies a "gutter" of RPE irritation from chronic waxing and waning subretinal fluid that has sunk inferiorly with gravity. Tumor thickness (red line) was measured from the posterior aspect of the RPE/Bruch's membrane down to the posterior border of the tumor (dashed blue line) using EDI-OCT and was found to be 1,450µm (*Figure 6C*).

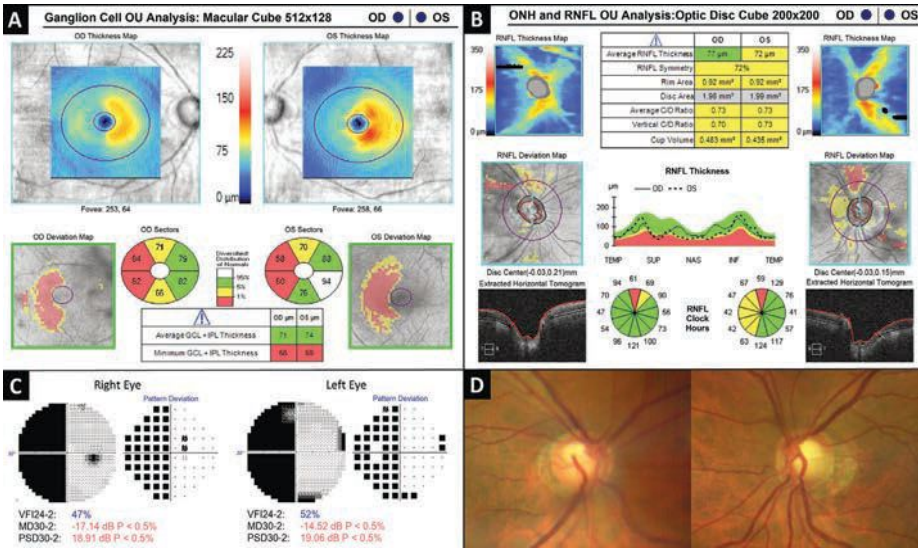


Fig. 7. Hemi-macular GCIPL thinning as a result of retrochiasmal visual pathway damage.

OCT in Retrochiasmal and Chiasmal Damage

Macular ganglion cell analysis algorithms can measure the combined thickness of the ganglion cell and inner plexiform layers (GCIPL), providing quantitative measurements that can detect subtle damage to the inner retina.[20]

Retrochiasmal lesions. Recall that the retrochiasmal visual pathway includes the optic tract, lateral geniculate nucleus (LGN), optic radiations and occipital cortex. A lesion involving any of these structures may result in corresponding homonymous hemi-macular GCIPL thinning.

For example, a right-sided retrochiasmal visual pathway lesion will result in temporal GCIPL thinning of the right eye and nasal GCIPL thinning of the left eye in addition to a left homonymous hemianopia. In the instance of an optic tract lesion, GCIPL thinning occurs due to retrograde degeneration, in which atrophy travels back toward the presynaptic cell body (ganglion cell).[21] In the case of a lesion of the optic radiations, transsynaptic retrograde degeneration occurs across the synapse at the LGN.[21] Anterior lesions of the retrochiasmal visual pathway, such as those involving the optic tract or LGN, result in faster and more severe GCIPL loss than posterior lesions involving the occipital lobe.[22]

Researchers found that the majority of hemi-macular GCIPL thinning occurs within six months (as early as two months in anterior lesions) and stabilizes at two years.[22] Therefore, if GCIPL thinning is found to be progressing after two years, multiple lesions (such as repetitive strokes) should be suspected.[22]

Figure 7 depicts a patient in his sixties who suffered a right-sided intraventricular hemorrhage

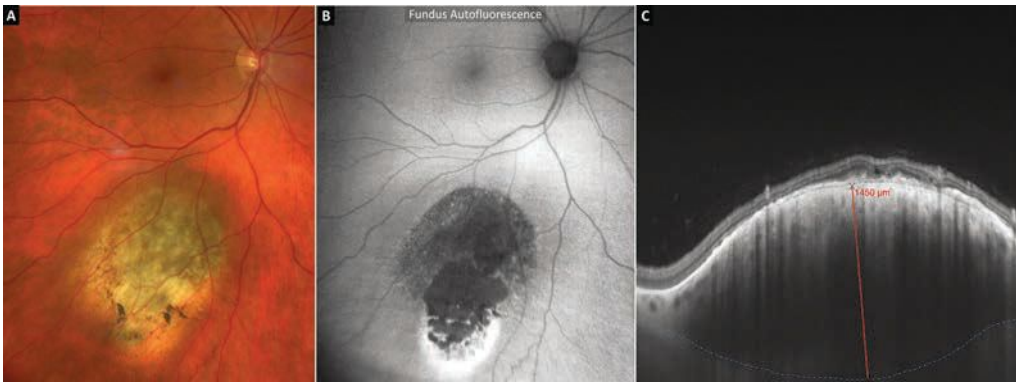
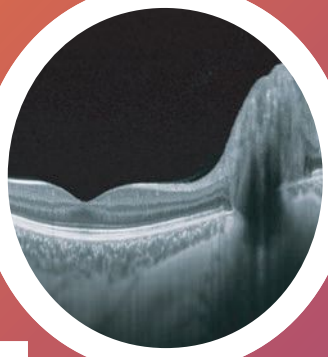
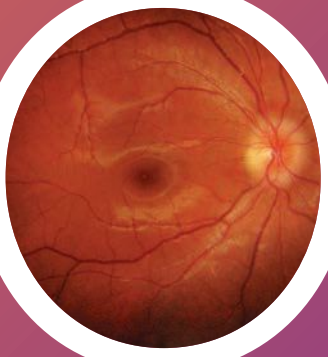


Fig. 6. EDI-OCT and thickness measurement of a suspicious pigmented choroidal tumor.

Take a closer LOOK





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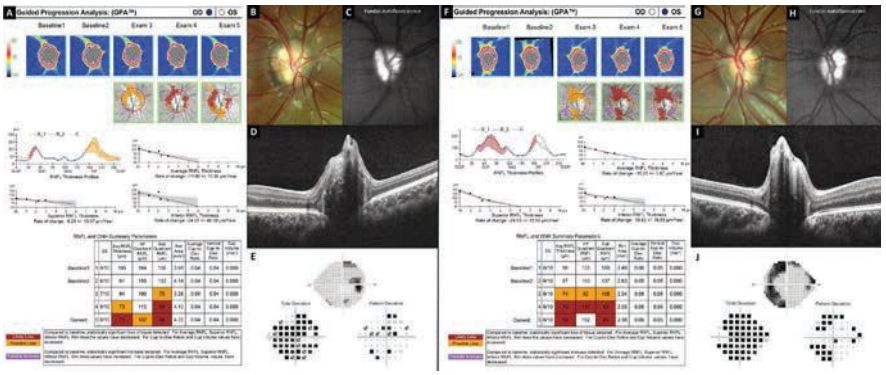

Fig. 8. Optic nerve and RNFL progression analysis demonstrating loss of the nerve fiber layer in optic nerve head drusen.

three years prior. Automated visual field testing (*Figure 7C*) revealed a near complete left homonymous hemianopia which corresponds to bilateral hemi-macular GCIPL thinning evident on OCT ganglion cell analysis (*Figure 7A*). The optic nerves demonstrated bilateral cupping along with temporal and nasal "band" pallor of the neuroretinal rim tissue of the left nerve (*Figure 7D*).

Retrochiasmal disorders include ischemic stroke (most common), neoplasm, hemorrhage, trauma and aneurysm.[23] A retrochiasmal lesion causing GCIPL thinning may be easily confused with and misdiagnosed as glaucoma. Red flags for a non-glaucomatous etiology include a homonymous pattern with vertical respect which may be a feature of both ganglion cell thinning and visual field loss. When these kinds of features are present, further investigation including neuroimaging should be considered.

Chiasmal lesions. A chiasmal visual pathway lesion, such as a compressive pituitary adenoma, may also result in hemi-macular GCIPL thinning. Chiasmal lesions are more likely to result in bi-nasal or heteronymous GCIPL thinning vs. the homonymous loss that commonly occurs with retrochiasmal lesions. Research suggests OCT ganglion cell analysis may be more sensitive than standard automated visual field testing in the detection of early chiasmal compression.[24] Therefore, OCT ganglion cell analysis should be included in the workup for patients suspected of chiasmal compression.

OCT in RNFL Progression

Most newer OCT systems include glaucoma progression analysis software that can be very valuable for assessing changes in RNFL thickness and optic nerve head morphology over time. OCT guided progression analysis will do most of the work for you to determine whether optic nerve pathology is worsening or improving. The clinical utility of OCT progression analysis software goes far beyond glaucoma and can be used to monitor many other conditions, including idiopathic intracranial hypertension, disc drusen, ischemic optic neuropathy and optic neuritis.

Figure 8 shows evidence of progressive RNFL thinning in a teenager with severe optic nerve head drusen confirmed with B-scan ultrasonography, FAF (*Figures 8C and 8H*) and EDI-OCT (*Figures 8D and 8I*). Analysis of the right eye (*Figure 8A*) shows decreasing RNFL thickness especially inferior, while the left eye (*Figure 8F*) shows decreasing RNFL thickness especially superior. This analysis represents a time span of three years.

Takeaways

As OCT continues to become more popular, it's worth revisiting how you currently use this technology and how you can expand its uses to better serve your patients and treat a wider range of ocular conditions. ■

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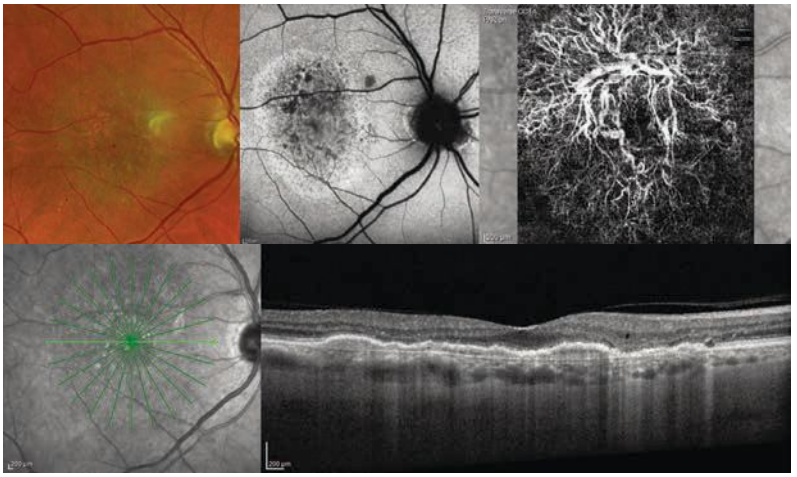

Fig. 1. AMD patient with a disc-shaped region of pigmentary abnormalities on clinical exam, also demonstrated on the FAF (top middle image). OCT shows an elongated pigment epithelial detachment (PED) with minimal evidence of overlying exudation such as subretinal or intraretinal fluid. OCT-A (top right), however, shows an extensive choroidal neovascular network underlying the PED.

When diagnosing and managing posterior segment pathology, a wide variety of technologies aid us, including posterior segment imaging, neuroimaging and tests of visual function. Some conditions may be diagnosed and managed based on the clinical examination alone, but even in common conditions such as age-related macular degeneration (AMD) and diabetic retinopathy (DR), diagnostic imaging may show unique aspects of the disease that cannot be seen clinically. Additionally, more complicated pathologies often require a battery of tests to eliminate differentials and arrive at the correct diagnosis. Inspect the cases and examples below to see how diagnostic testing can be used to further understand a pathology or to arrive at a correct diagnosis or treatment plan.

Examine how newer technologies such as optical coherence tomography (OCT), fundus autofluorescence (FAF) and OCT angiography (OCT-A) have changed how we understand and visualize posterior segment pathology. Notice how advances in these technologies such as OCT with enhanced-depth imaging (EDI) and the ability to obtain widefield imaging in technology such as FAF is changing how we view the retina. Nevertheless, older diagnostic tools such as visual field testing, B-scan ultrasound, fluorescein angiography (FA) and indocyanine green angiography (ICGA) can still provide critical information.

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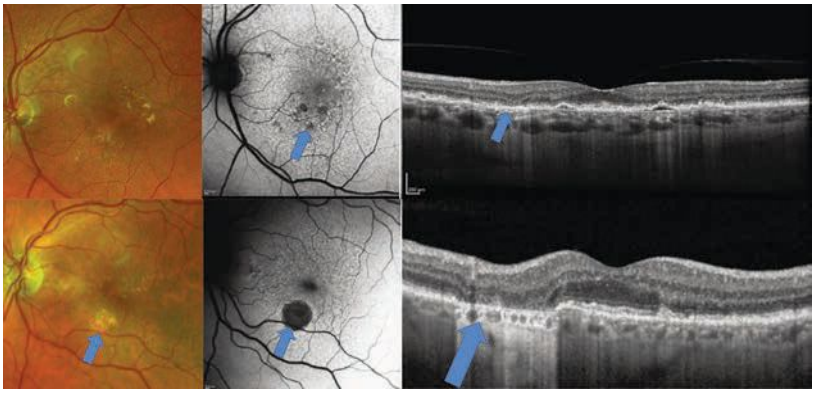

Fig. 2. Top images show a patient who would be categorized clinically as intermediate-stage AMD due to pigmentary changes. The FAF shows diffuse alterations to the RPE with areas of hypo-autofluorescence suggestive of early GA. In this region on the OCT, there is atrophy of the outer nuclear layer also suggestive of early GA. The bottom images show a patient with a frank area of GA that is visible clinically but seen even more easily with FAF.

Small but Mighty

Standard staging of AMD is based on clinical examination—primarily focusing on the size of macular drusen and the presence or absence of macular pigmentary changes.[1] The AREDS1 trial determined that eyes with large-sized drusen (greater than 125µm) and/or pigmentary changes had increased risk of developing advanced-stage disease and benefited from vitamin supplementation.[2] At the time, clinical examination and FA were the tools used to determine presence of advanced AMD with presence of choroidal neovascularization (CNV) or geographic atrophy (GA).

Now, the use of multimodal imaging—including OCT, OCT-A and FAF—provides us unique insight into who is at risk for progression to advanced-stage AMD and who may already show early signs of advanced disease. OCT and OCT-A allow for earlier detection of CNV, even occult or non-exudative CNV (*Figure 1*).[3] OCT cross-section scans may show presence of sub- or intra-retinal fluid suggestive of exudative AMD that is not visible clinically. OCT-A may show the presence of vasculature in the outer retina or avascular zone suggestive of CNV that is not visible clinically, and may not even show signs of "fluid" on OCT.

OCT and FAF are of great use in identifying early regions of GA, which may be crucial if treatment for GA is approved in the future (*Figure 2*).[4] OCT cross-section scans can be evaluated for atrophy of the outer retina including the outer nuclear layer and the retinal pigment epithelium (RPE). Additionally, increased reflectivity called "hyper-reflective columns" extending into the choroid may be signs of RPE atrophy on OCT cross-section scans. Areas of GA will show on an FAF as dark regions of hyporeflectivity.

OCT and FAF were particularly helpful in this case of a 70-year-old white male who presented for an AMD evaluation with 20/20 vision OU (*Figure 3*). On clinical examination, the patient had neither large drusen nor pigmentary changes. A 12-line radial OCT with EDI and FAF of the macula demonstrated the presence of reticular pseudodrusen (RPD). This is a particular phenotype of drusen that may be missed on clinical exam but is well visualized with OCT or FAF.

On OCT, RPD present as hyper-reflective projections on top of the RPE, and on FAF, they present as diffuse reticular alterations to the FAF signal.[4] Identification of this phenotype is important as patients with RPD have even higher incidence of developing advanced-stage AMD (both CNV and GA) than patients with large drusen.[5,6] In addition, they tend to have worse visual function, which could be evident on functional tests such as dark adaptation.[7,8] Educate these patients heavily on the risk of conversion to advanced disease and monitor them more carefully.

Stages of DR

DR, like AMD, is classically staged based on clinical examination.[9,10] In addition, clinically significant macular edema, an essentially outdated term, judged presence of macular edema based on clinically visible thickening of the retina. Since these categorizations, OCT and OCT-A have been developed and give us a wealth of information regarding diabetic retinopathy that cannot be visualized with fundus exam alone.

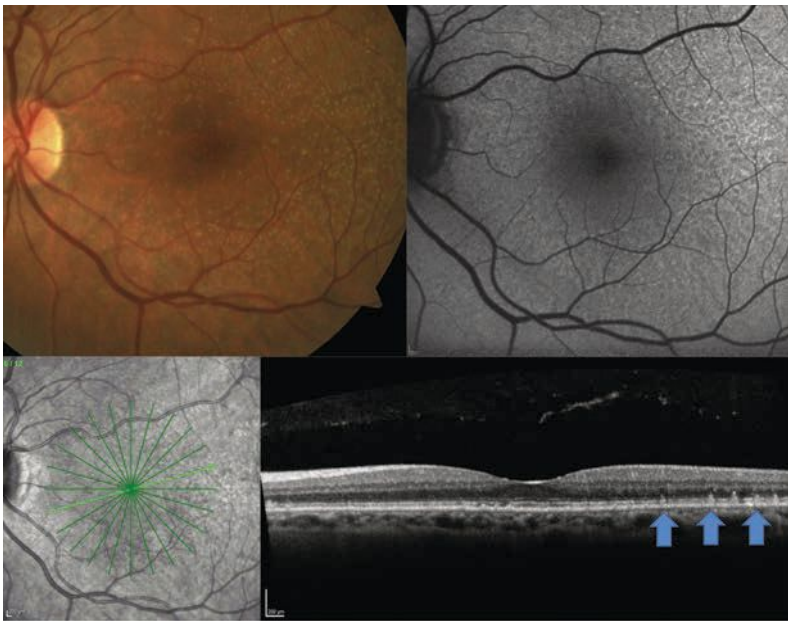

Fig. 3. A patient with clinically visible small- to medium-sized drusen shows presence of RPD on FAF and OCT.

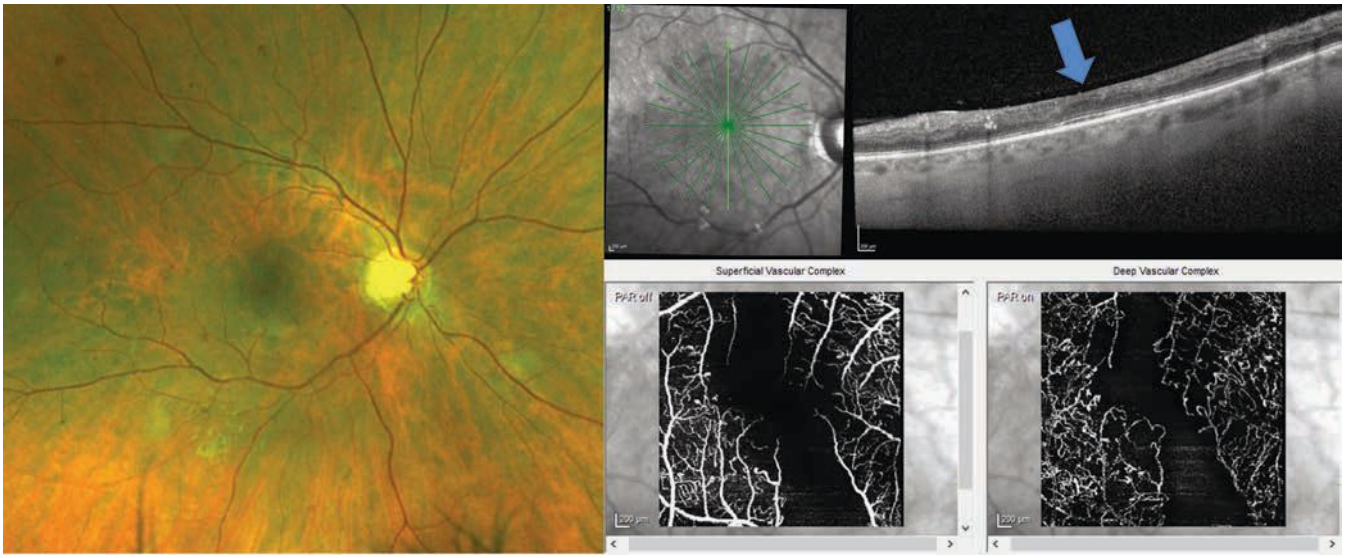

Fig. 4. Patient with clinically visible moderate nonproliferative DR with intraretinal hemorrhages, microaneurysms and cotton wool spots has diffuse inner retina thinning on the OCT cross-section scan (blue arrow shows position of the fovea). OCT-A confirms significant macular ischemia both in the superficial and deep capillary plexi that feed the inner retina.

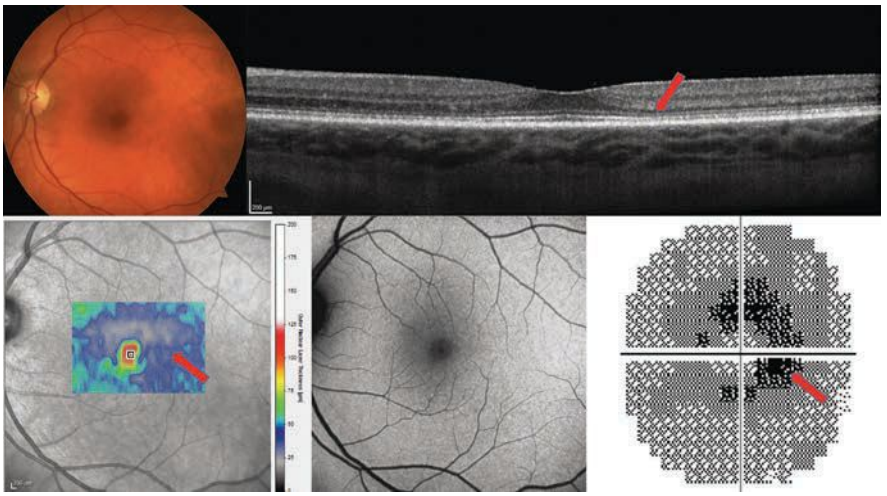

Fig. 5. The cross-section OCT shows a very thin outer nuclear layer, especially temporally (red arrow) but no frank loss of the photoreceptor integrity line. The thickness map of the ONL only also appears very thin (bottom left). The FAF is essentially normal. The 10-2 visual field shows a ring scotoma most dense nasally and is highly suggestive of plaquenil maculopathy. Findings were bilateral.

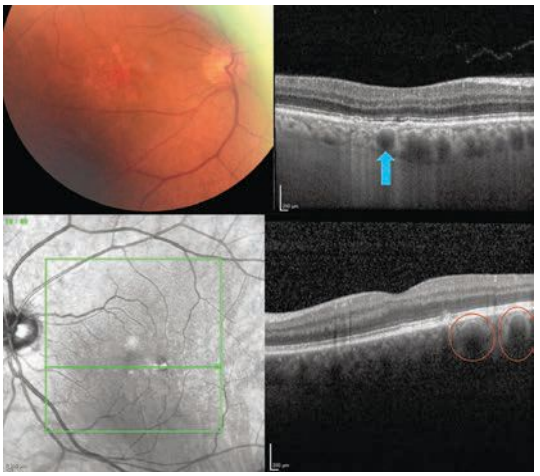

Fig. 6. A well-delineated pachyvessel (top; blue arrow) and several adjacent ones in a patient with RPE disruption mimicking AMD (bottom), a 72-year-old Caucasian on AREDS2 for AMD. Note the thickened choroid (which should be thinner in AMD) and two pachyvessels (red circles), one of which is just below the RPE disruption.

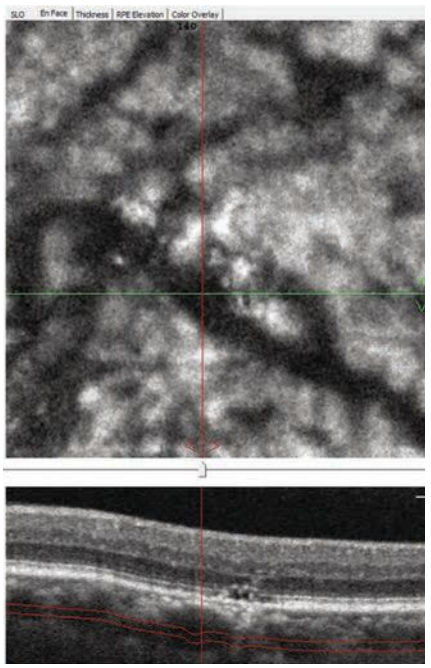

Fig. 7. *En face* OCT-A allows viewing at different depths and can highlight pathologically dilated choroidal vessels (pachyvessels), which typically show an abrupt termination vs. gradually tapering.

For example, patients may have presence of diabetic macular edema on OCT that is not visible clinically. OCT cross-section scans may also show regions of inner retinal thinning suggestive of capillary non-perfusion.[11] OCT-A may show microvascular alterations that are not detected clinically, including macular ischemia, and

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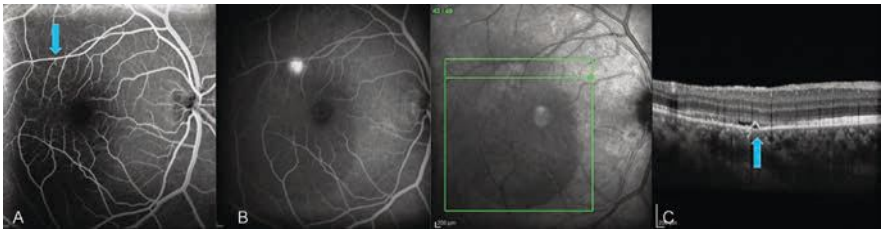

Fig. 8. Multimodal imaging of a 34-year-old male with CSC. (A) FA in the early phase highlights the triangular-shaped PED found on spectral-domain OCT (blue arrows). (B) Late-phase FA shows leakage from this area, confirming an RPE microrip. Note the centrally diminished choroidal fluorescence in late phase due to the large serous RD. (C) EDI SD-OCT (Spectralis, Heidelberg) showed choroidal thickness of about 500µm with a triangular-shaped PED and neurosensory RD, the latter of which increased as it got closer to the fovea. Multimodal testing altered the initial treatment plan of observation to one of referral for laser treatment. FA allows for visualization of the area of leakage that can be targeted with laser treatment. This cannot be seen with OCT or OCT-A.

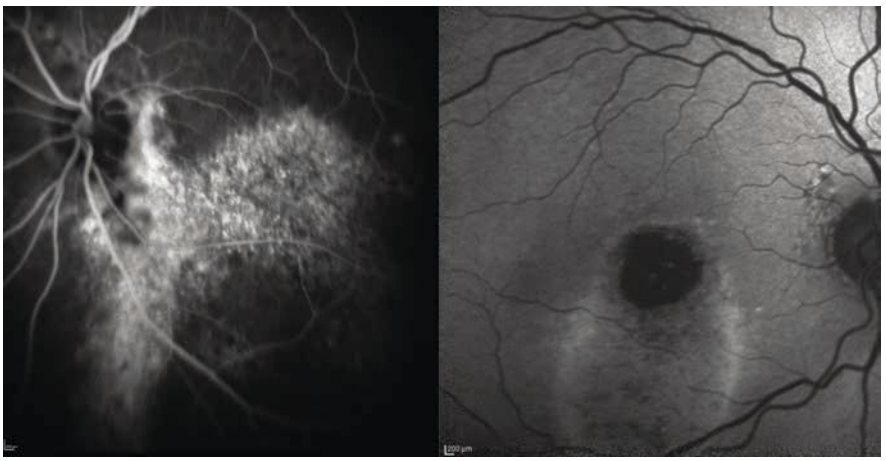

Fig. 9. Chronic serous detachment from CSC leading to RPE disruption shown by FA (left) and FAF (right) in two different patients. FAF is very useful with CSC patients and can shed some light on the condition's chronicity with the discovery of gravitational tracts due to shifting subretinal fluid.

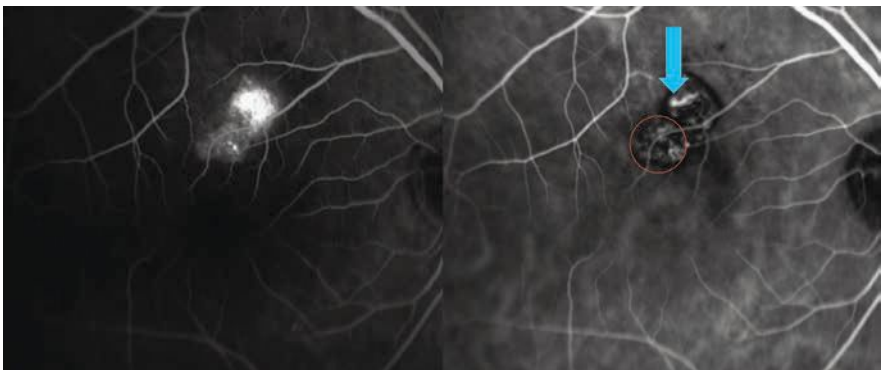

Fig. 10. FA doesn't give a detailed view of choroidal pathology (left) due to the molecule's small size and ability to seep through choriocapillaris fenestrations, as well as a shorter excitation wavelength that gets blocked by the RPE. ICGA (right) employs a larger molecule that is protein-bound and excites in the infrared range, therefore reaching the deeper structure. In this patient with polypoidal choroidal vasculopathy, or aneurysmal type 1 neovascularization, the BNVs can be seen with a dendritic-like pattern (red circle). The "hot-spot" (blue arrow), which is pathognomonic for PCV, denotes the condition's polyp. These findings are not well-visualized with FA (left).

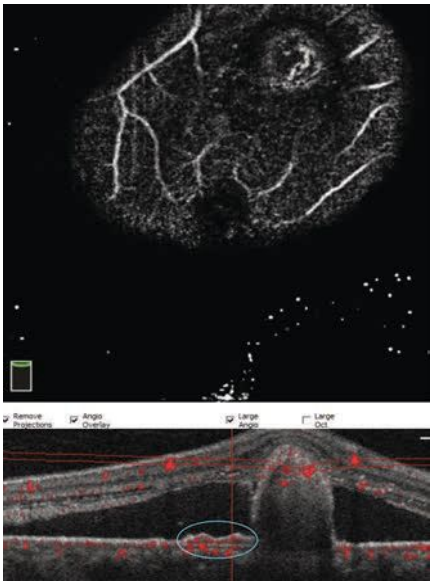

Fig. 11. With significant pathology, OCT-A sometimes has trouble segmenting. Here, manual manipulation of the scan identifies the polyp. These represent further growth of the BVN under the shallow RPE detachment (double layer sign, blue circle) and continuing to proliferate on the undersurface of the RPE detachment.

can be helpful to distinguish intraretinal microvascular abnormalities from retinal neovascularization.[12]

OCT and OCT-A were of great use in diabetic retinal vascular damage in a 37-year-old male type 2 diabetic for 10 years who funduscopically presented with moderate nonproliferative DR with presence of intraretinal hemorrhages, cotton wool spots and microaneurysms (*Figure 4*). The patient had poor blood sugar control with a most recent A1c of 10%. The patient complained of blurry vision OD even with his new glasses and was correctable to only 20/30 OD.

A 12-line radial OCT without EDI was performed showing significant inner retinal thinning, especially in the vertical meridian (*Figure 4*). Inner retinal atrophy on OCT in a diabetic patient should raise alarm for potential macular ischemia.[11] A 10° x 10° OCT-A was taken to evaluate the central macula vasculature confirming significant atrophy of both the deep and superficial vascular plexus.[12] These findings could not be visual-

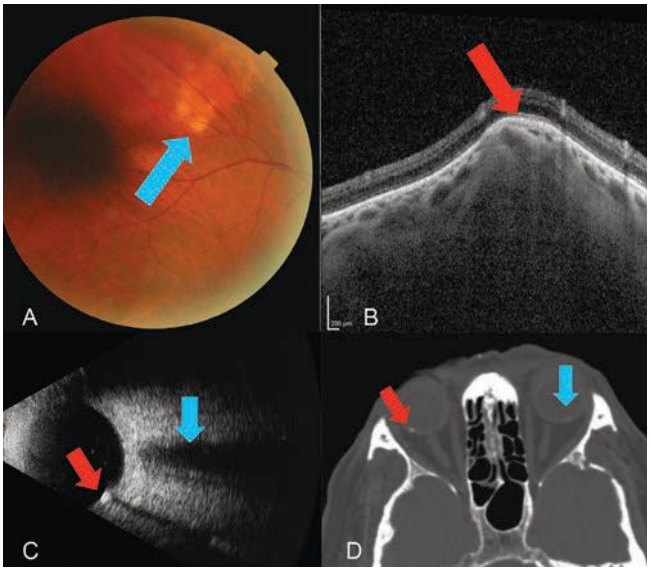

Fig. 12. (A) Fundus photo of the yellow, subretinal lesion (blue arrow) located in the superior temporal quadrant of the right eye. (B) EDI-OCT (Spectralis, Heidelberg) through the SCC reveals a type 2 "rolling" lesion. Note the choroidal thinning above the lesion (red arrow). (C) B-scan ultrasound using the 10MHz probe highlights the echo-dense SCC (red arrow) with acoustic orbital shadowing which appears much smaller but similar to the optic nerve which is shown above this area (blue arrow). A choroidal melanoma would demonstrate low to medium internal echoes. (D) A previously ordered CT scan on a patient with SCC of the right eye (red arrow) and a subtle one in the left (blue arrow), which was not visible on clinical exam. Though not necessary in this case, sometimes radiological testing can be a form of multimodal imaging.

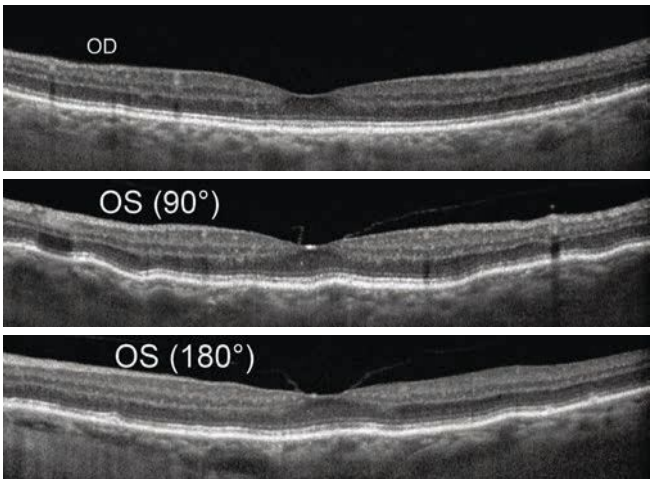

Fig. 13. Baseline OCT shows OD (top) mild disruption of the RPE however normal macular contour and choroidal thickness. OS (middle and bottom) shows mild choroidal folds that are typically more visible in vertical scan line as seen here. Mild flattening of foveal pit by vitreomachular adhesion and traction and similar choroidal thickness as compared with OD.

ized clinically and suggest more advanced and significant disease. The level of ischemia is likely the cause of the patient's reduced visual acuity and symptoms. The patient

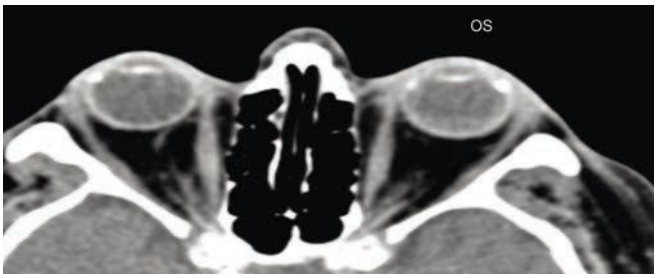

Fig. 14. Previously performed CT scan shows normal orbit and globe OU.

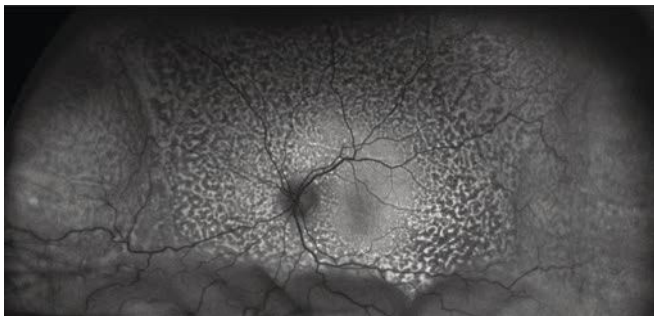

Fig. 16. Widefield FAF shows extensive pigmentary alteration of the left eye.

was counselled regarding the finding and need for strict blood sugar control. In addition, the patient should be followed more closely for potential complications, such as retinal or iris neovascularization. It was recommended for the patient to have a widefield FA to evaluate peripheral retinal nonperfusion and rule out presence of clinically undetectable retinal neovascularization.

Keeping it Old School

With new technologies such as OCT, OCT-A and FAF that can give us a plethora of information in a few short minutes or even seconds, we sometimes fail to acknowledge when older technologies such as visual field testing can still be useful.

Patients who take hydroxychloroquine (HCQ) must be monitored for macular toxicity, and current guidelines from the American Academy of Ophthalmology still recommend that patients have annual testing with both SD-OCT and visual field annually after five years of medication use.[13] Patients with increased risk for toxicity should be seen annually following initiation of HCQ. Adjunct tests such

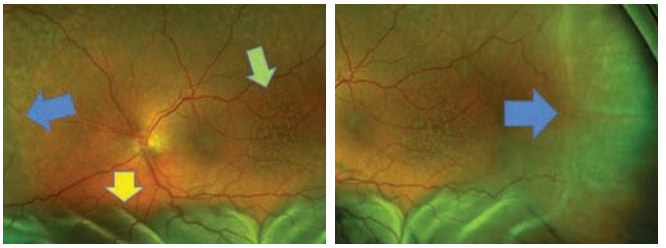

Fig. 15. Choroidal detachment in the left eye (blue arrow). Serous RD (yellow arrow) and pigmentary changes (green arrow) are also shown.

as FAF and multifocal electrogram may also be considered. Classically 10-2 visual field testing has been recommended either with white or red stimuli, but recently it is recommended that those of Asian descent have 24-2 visual field testing as findings may present more peripherally and not in the parafoveal region.[13]

Figure 5 shows the importance of continued visual field testing for a 172lb female patient who had been taking HCQ 200mg BID for 20 years for rheumatoid arthritis. She had no history of kidney or liver disease. OCT showed no frank loss of the photoreceptor integrity line but was suspicious for parafoveal outer nuclear layer thinning. Is this toxicity or a variation of normal? FAF was essentially normal with no red flags raised. The most convincing evidence for toxicity was the 10-2 visual field that showed repeatable bilateral ring scotomas. This along with the OCT finding and the high risk for toxicity at 20 years of medication use made it a must to advise the rheumatologist to discontinue the medication.

The Plot Thickens

For years, the B-scan ophthalmic ultrasound offered the only multimodal glimpse of the choroid, albeit a low resolution one. Because of this, separating it from the surrounding retina and sclera was impossible. This, of course, changed with the availability of OCT, and more specifically, the 2008 description of EDI.[14] By focusing the instrument forward, the researchers captured inverted but detailed high resolution images of the choroid.

Using this technique provided a fresh look at this structure and led to one publication on pachychoroid pigment epitheliopathy (PPE) and an introduction of the "pachychoroid spectrum."[15] The article's title represented

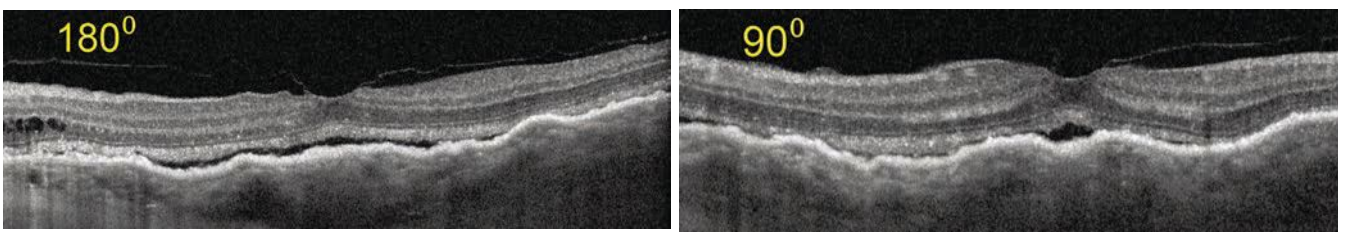

Fig. 17. OCT shows Increased choroidal thickness and folds as compared with the baseline OCT. Also, subretinal fluid as the result of serous RD is notable.

the first entity, followed by central serous choroidopathy (CSC), pachychoroid neovasculopathy (PNV) and finally polypoidal choroidal vasculopathy (PCV), also known as aneurysmal type 1 neovascularization. Later, focal choroidal excavation and peripapillary pachychoroid syndrome (PPS) were added to the spectrum. The pachychoroid phenotype is not completely understood. It may result from choroidal vascular hyperpermeability, which leads to choroidal thickening, compression of the choriocapillaris and subsequent ischemic damage to the RPE and outer retina.

But even after several years, some practitioners have yet to embrace and properly vet the spectrum, leaving them vulnerable to misdiagnosing conditions such as AMD and placing patients on costly, unneeded supplements.

This occurred with two patients who were previously diagnosed with AMD and prescribed AREDS2 (*Figure 6*). In both cases, an EDI-OCT (Spectralis, Heidelberg Engineering) revealed a thickened choroid with pachyvessels, which are pathologically dilated structures that can also be seen with OCT-A (*Figure 7*). RPE and outer retinal damage typically occur just above the pachyvessel. These presentations are consistent with PPE.

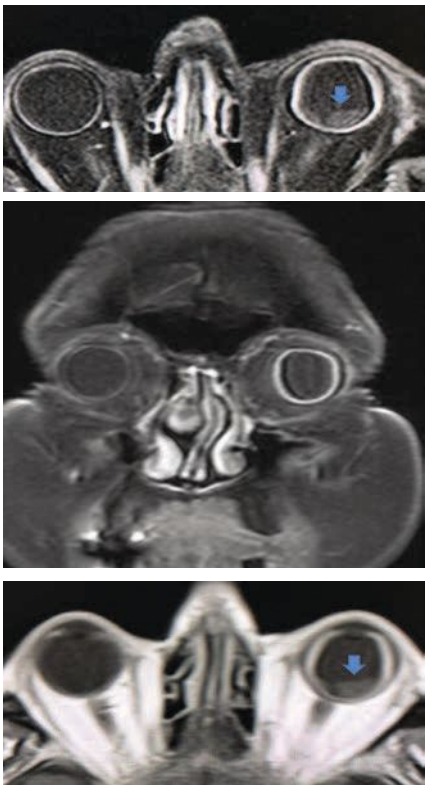

Fig. 18. Coronal (middle) and sagittal (top and bottom) with and without enhancement shows a difference in scleral thickness of the left vs the right eye. In the sagittal views, the presence of detached areas can be seen (blue arrow).

Persistent damage can disrupt the RPE barrier and allow fluid to seep into the subretinal space which causes a serous retinal damage and signifies CSC (*Figure 8*). Chronic serous detachments can lead to advanced RPE disruption (*Figure 9*). PNV consists of a type 1 neovascularization and a shallow RPE detachment known as the "double-layer" sign (*Figure 10*). PNV lacks extensive hemorrhage and exudate, unlike PCV, which has branching vascular networks (BVNs) and polyp formation on the undersurface of a much larger RPE detachment (*Figures 10 and 11*).

The B-side of Multimodal Imaging

For those who practiced in the pre-OCT era, the B-scan ophthalmic ultrasound represented one of the only—and possibly earliest—forms of multimodal imaging. A choroidal melanoma, for instance, possesses distinct ultrasonographic properties, including acoustic hollowness with low to medium internal reflectivity.[16] Knowing this allows the practitioner to differentiate between a suspicious finding and something more benign.

In this case, a 69-year-old Caucasian male presented with an elevated, yellow, subretinal lesion outside the superior temporal arcade OD (*Figure 12A*). An EDI-OCT through the area demonstrated choroidal thinning above the lesion (*Figure 12B*). B-scan ultrasonography highlighted an echo-dense placoid mass with acoustic orbital shadowing resembling the presence of two optic nerves (*Figure 12C*).

Sclerochoroidal calcification (SCC) is a deposition of calcium within the sclera. They present as single or multiple, round focal lesions along the superior temporal arcades most commonly seen in the elderly as an incidental finding. SCCs appear pale yellow, white or orange and can be flat or up to 6mm in height.[17] One study analyzed 179 eyes of 118 patients with SCC and found the vast majority to occur in Caucasians (98%) with a mean age of 69 (32 to 95 range) and a 60:40 female-to-male ratio.[18] The worst statistic, however, was the abysmal rate of practitioners who diagnosed the condition correctly (5%).[18] Interestingly, these can be picked up on computed tomography (CT) imaging (*Figure 12D*).

A Diagnosis of Exclusion

When discussing imaging technologies, many should also consider the utility of non-ophthalmic procedures that might help determine a patient's diagnosis or etiologic factors of their conditions. Examples include carotid ultrasound or magnetic resonance imaging (MRI)/CT imaging, cardiac imaging and neuroimaging by CT or MRI.

The final diagnosis of this case was made by an MRI of the orbit. A 79-year-old white female returned with recent vision loss of her left eye. Her ocular history was remarkable for asymptomatic unilateral choroidal folds OS, which were detected four years ago and had been stable until her recent presentation (*Figure 13*). No underlying etiology was discovered for the choroidal folds, and due to the stability and asymptomatic nature including a "normal" CT, no further investigation was pursued (*Figure 14*).

On this visit, her visual acuity OS had dropped from 20/20 to 20/70. Her intraocular pressures (IOPs) were 16mm Hg OU which was in the range of prior visits. All entrance testing and anterior segment findings were normal, but the left eye fundus exam had drastically changed. There was evidence of choroidal and serous retinal detachment (RD), (*Figure 15*) and an unusual pigmentary presentation seen more drastically by widefield FAF (*Figure 16*).

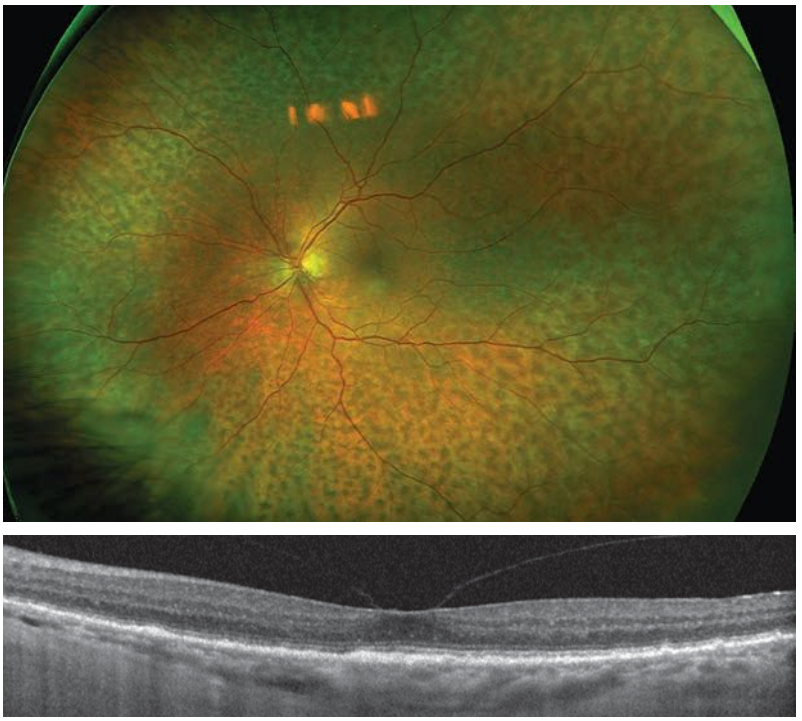

Fig. 19. One year post-op, fundus photography (top) shows persistent pigmentary change but complete resolution of choroidal and retinal detachments. OCT (bottom) shows resolution of subretinal fluid, normalized choroidal thickness and absence of choroidal folds.

OCT also revealed drastic changes as compared with the baseline examination (*Figure 17*).

Concerned with a neoplastic association or causation, the care team ordered a thin-slice MRI of the orbit, which then showed absence of neoplasm. However, an extremely thick scleral wall of the left eye as compared with the normal right eye (*Figure 18*). This finding lead to the diagnosis of uveal effusion syndrome (UES).

UES is a rare syndrome with no specific risk factors, usually found in otherwise healthy adults and is essentially a diagnosis of exclusion. There are three forms of the disease. Type 1 seen in nanopthalmic eyes (short axial length and usually hyperopic), type 2 seen with normal axial length and clinically abnormal sclera and type 3 with normal axial length and clinically normal sclera. Types 2 and 3 are thought to be the result of congenital scleral anatomy or abnormality.

Associated signs and symptoms are blurred vision, normal to elevated IOP, detachment of the choroid and ciliary body, serous RD with no or minimal inflammatory findings and localized areas of hypertrophy or hyperplasia of the RPE known as "leopard spots."[19,20] Ultrasound biomicroscopy and MRI are useful in diagnosis by measuring the scleral thickness.[21] The patient underwent typical surgical management known as sclerectomy or scleral window.[22] This resulted in resolution of the choroidal folds and detachment as well as the serous RD and vision improvement to 20/25 (*Figure 19*).

Conclusions

Each technology available provides a unique insight into retinal structure and function. Multimodal imaging and use of ancillary tests of visual function may paint a clearer picture of the pathological process. More difficult cases may require extensive testing to rule out potential differentials and arrive at the correct diagnosis. It is crucial for the provider to be aware of the benefits of each technology to best diagnose and manage posterior segment disease. ■

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NEW TOOLS AND ADVANCED TECHNIQUES FOR OSD DIAGNOSIS

If you want to spot cases earlier and understand the patient's status more deeply, these options can augment your dry eye work-up.

BY CECELIA KOETTING, OD
NORFOLK, VA

Ocular surface disease affects the majority of our patients in varying ways and to different degrees. By and large, it is not difficult to identify. We can see it at the slit lamp in the form of reduced tear lake, punctate staining and debris along the eyelid margin. We also see it in the faces of our patients when we ask them to describe how their eyes feel. Often, we can assess and manage dry eye with just a few standard tools and sufficient time and attention to the patient.

However, as diagnostic technology has improved our ability to differentiate the underlying causes of the many conditions that fall under the umbrella term of 'ocular surface disease,' we can now customize treatment more precisely, improving outcomes. We can also use more high-tech tools to document baseline status to quantify change over time and to convey to the patient the extent of their condition in hopes of getting their buy-in on efforts to control it. These are demonstrable benefits to both the patient and the practitioner. The question becomes: where does new technology fit into our practice and how can we use it to truly impact our patients' treatment plan for the better? In this article, we'll look at several newer concepts in meibomian gland assessment.

Meibography

Secretions of the meibomian glands in both the upper and lower eyelids help to form the lipid layer of the tear film. Meibomian gland dysfunction (MGD) makes expression of these glands either difficult or near to impossible.[1] Decreased lipid layer leads to tear film instability, resulting in evaporative dry eye.[1] Patients experiencing poor meibomian gland function will have complaints that may include fluctuating vision, eyelid irritation and itching, and eyelids being sticky in the morning.[2] MGD induces structural changes to glands and the eyelid margins that can lead to destruction of the meibomian gland orifices or atrophy.[3]

We typically evaluate the meibomian glands at the slit lamp, looking for evidence of irregular lid margins; hyperemia, telangiectasias, pitting or meibum orifice plugging. Inferior lids are much easier to assess and appreciate changes, but an attempt should be made to the upper glands as well. Digital pressure on the glands using a cotton tip applicator or Meibomian Gland Evaluator (Johnson & Johnson) can help evaluate the expressibility and quality of the meibum released. Meibum quality can be described as clear, cloudy, granular or inspissated, grading as follows:[3,5]

Grade 1: olive oil, clear
Grade 2: turbid, cloudy

Meibography of a patient with severe dry eye disease. Note they have moderate atrophy of the glands with moderate truncation and loss of gland structure.

About the author

Dr. Koetting practices at the secondary/tertiary surgical center Virginia Eye Consultants in Norfolk, VA. Her primary focus is ocular disease, specializing in anterior segment and corneal disease, neuro-optometry and perioperative care. She also participates in clinical research and the maintenance of the referral network alongside the practices other optometrists. Dr. Koetting serves as the externship director and is adjunct faculty for several schools and colleges of optometry. She is a consultant for Glaukos, Eyevance, Ivantis, Alcon, Orasis and IVL, as well as a speaker for Dompé, Glaukos, RVL and Eyevance.

Grade 3: cloudy with debris
Grade 4: toothpaste-like or inspissated

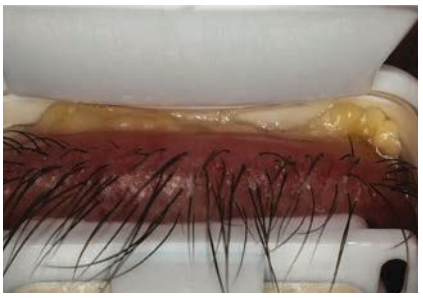
Historically, we have associated these slit lamp findings of expressibility and meibum quality with how we grade MGD. The more difficult it is to express the glands is thought to be a correlation to either gland atrophy or poor meibum quality. However, over the last few years with the advent and wider implementation of meibography, we are able have a direct view of the meibomian glands.

Meibography is specialized imaging that allows us to directly visualize the morphology and health of the meibomian glands with greater detail than slit lamp assessment alone. Using an infrared non-contact method, we can observe the meibomian glands in both the upper and lower eyelids, where we can see the true extent of the patient's MGD. Normal glands appear straight and lined up like a white picket fence. Abnormal glands will appear tortuous, dilated, congested or atrophied.[2,4]

A retrospective study of meibomian gland structure changes using LipiScan (Johnson & Johnson) infrared meibography on the inferior central lid margin of 400 preoperative patients of cataract surgery age compared the level of atrophy (meiboscore) with physical findings on a slit lamp exam, including expressibility with meibum grade.[5] The meiboscore grading system is as follows:

Grade 0: no loss of meibomian glands
Grade 1: 1% to 33% gland atrophy
Grade 2: 34% to 66% gland atrophy
Grade 3: greater than 66% atrophy

In this study, meibography revealed that 95.1% of patients had some level of gland atrophy.[5] When comparing the slit lamp findings of expressibility (moderate and difficult) or a meibum grade 2 or worse in the same patients, there approximately 20% more identified as having atrophy via meibography.[5] Further breakdown found a correlation between decreased expressibility of mebomian glands to increased mebomian gland atrophy but not between meibum quality and mebomian gland atrophy.[1] Knowledge



This is the same patient undergoing iLux treatment. Note the significant amount of yellowy-white, poor quality meibum being expressed.

of both functional and architectural severity of MGD is important for clinical decision making, including appropriate ocular surface treatment and surgical decisions. Routine use of meibography would allow clinicians to identify patients with MGD earlier so we can treat and halt progression before extensive damage is done.[4,6]

Current available meibographers include Keratograph 5M (Oculus), Meibox and MX2 (Box Medical Solutions), IDRA, OSA and OSA Plus (SBM Sistemi), Ocu-Cam UltraHD (OICO), LacryDiag (Quantel Medical), LipiScan (Johnson & Johnson), and the HD Analyzer (Keeler). Soon, a new version of the iLux (Alcon) will be available that includes a built-in meibographer with the ability to take videos and photos along with providing thermal treatment for MGD.

Information from meibography, slit lamp exam findings and physical expression helps us to grade dysfunction of the meibomian glands and identify the type of dry eye and its underlying causes, allowing us to implement appropriate treatments. Patients with any grade of MGD would benefit from gland expression coupled with in-office lid exfoliation. In patients with MGD, blepharitis, *Demodex* and ocular rosacea, exfoliation of the eyelid at the lash line helps to remove the inflammatory biofilm that causes chronic lid disease and discomfort.[4]

With greater diligence to good lid hygiene, we can help to halt or slow the progression of MGD and improve patients' symptoms.[7] Some options on the market include eyelid exfoliation using BlephEx (Alcon) or LidPro (Mibo Medical) and heated expression treatment such as iLux (Alcon), LipiFlow (Johnson & Johnson) or TearCare (Sight Sciences), or even Mibo ThermoFlo (Mibo Medical) coupled with expression in the slit lamp.

Interferometry

If meibography identifies MGD with greater sensitivity, its counterpart is interferometry, a tool that reveals how that dysfunction plays out on the ocular surface—again, in considerably more detail than our conventional tools.

Our earlier conception of a tri-layered tear film (lipid, aqueous, mucin) has since been updated to a model of an outer lipid layer overlying a mucoaqueous structure.[8,9] If the tear film components become unbalanced, it can cause the entire system to fail, leading to dry eye. Impaired tear film stability is considered an essential criterion for diagnosing abnormality of tear film per the TFOS DEWS II report.[6] As our concepts get more sophisticated and nuanced, so too should our techniques.

Tear film quality has most commonly been monitored using tear break-up time with application of sodium fluorescein, which in itself is known reduce the stability of the tear film.[6] Non-invasive tear break up time (NIBUT) can be obtained from placido disc images reflected by the cornea from the tear film, and can be done with many of the current corneal topography systems.[6] Other testing that can be performed to assess tear film quality includes thermography, Schirmer test, tear osmolarity, MMP-9 marker identification with InflammaDry (Quidel) and, though less readily available, tear film ferning.[6]

Another technique—a noninvasive one at that—to monitor tear film stability is interferometry, which analyzes both the tear film stability and thickness of the lipid layer in a semiquantitative method.[9] Interferometry can distinguish clinical subtypes of dry eye by looking at the balance between the lipid and aqueous layers of the film.[9] This test has been used in numerous

Moving From Theory Into Practice

By Ian Flitcroft, MA, D.Phil, FRCOphth

Several years ago, my hospital conducted a survey of optometrists in academia and private practice looking at the barriers for clinicians to start practicing myopia control. The obstacles reported related to limited training, an increased financial burden, changes to the standard clinical practice, incorporating new technologies and treatments, and lack of implementation support.

As a research scientist involved in myopia control, I investigated this further with my colleagues and we concluded that starting myopia management has little to do with science, but a lot to do with human psychology. In fact, one of the biggest hurdles eye care providers face in building a comprehensive myopia management program is first deciding where to start.

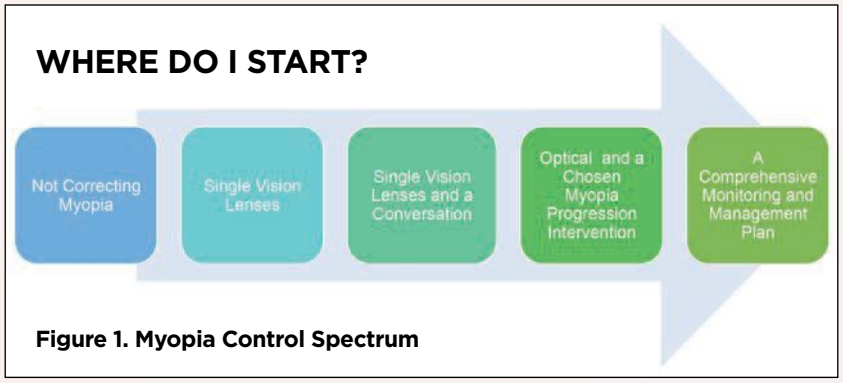


Figure 1. Myopia Control Spectrum

Image: Ian Flitcroft, MA, D.Phil, FRCOphth

Every clinician providing comprehensive eye care for a young demographic falls somewhere on the myopia control spectrum represented in Figure 1, based on my team's research. We found that optometrist involvement can range from those who strongly believe optometrists shouldn't be managing pediatric myopia, to those who offer a comprehensive diagnosis, monitoring, and management plan during each phase of the patient's myopia control journey. Our research also revealed that the vast majority of eye care providers treating myopia rely solely on single-vision lenses without addressing the need for monitoring and management of progressive myopia.

The reality is, if a clinician isn't providing a comprehensive diagnostic, monitoring, and management program, they are not effectively practicing myopia control. In fact, single-vision lenses alone are one of the least effective treatments for myopia.[1,2]

Start the Conversation

All eye care practitioners should start a conversation about myopia and myopia control with the parents of myopic children. Unfortunately, that's where many providers stop because they are unsure about increased chair time and the messaging to the parents. To facilitate this process, eye care providers can prepare simple messages that help explain that their goal is to minimize the advancement of patient's refractive error. Below are key points to consider when starting a dialogue with parents:

- **Communicate with the parents. If the child is already myopic, start with, "We need to slow the progression of the refractive error." If the child is at risk of becoming myopic, start with, "We need to delay or stop potential myopia."** If applicable, relate to the parents' myopia, as many myopic children are likely to have myopic parents.[3] Don't overpromise, consider the following plan, "Let's aim for the myopia to be half of what it would have been without treatment."
- **Don't feel the need to convince parents to get on board with myopia treatment, just provide the educational resources to make them aware that something can be done.** When facing resistance from parents, simply say, "We understand your concern, many parents have felt similarly when first learning about their child's myopia. When you bring your son/daughter back for their glasses check we can always discuss management and treatment options again."
- **Highlight successes in myopia control.** There's no better way of encouraging parents to get on board than to show them that myopia control can work!

Ian Flitcroft, MA, D.Phil, FRCOphth, is a pediatric ophthalmologist on staff at Temple Street Children's Hospital in Dublin, Ireland.

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The opinions expressed in this article are the author's own and do not constitute advice from Topcon on how to diagnose.

Strategies for Myopia Success

By Gary Gerber, OD

Despite reported barriers to implementing myopia control in an eye care practice today, some optometrists are succeeding in myopia care. What's different about these eye care providers? On the clinical side, nothing. These optometrists have the same clinical acumen, the same skills as their colleagues; they may have put in more time studying myopia control, but the training is out there, and anyone can certainly learn it. In addition, the technology to measure axial length is readily available.

I would argue the most important quality of eye care providers succeeding with myopia control is they think differently about myopia. They're leaders in their practices and let the staff know that "myopia matters, it's important, and we're going to do something about it."

How you think about myopia will determine what you do about it. So, the first thing to consider is: Is myopia a disease or is it a refractive error?

For example, the optometrist who approaches myopia as a disease will view a child who comes in for an optical chief complaint, who also presents with myopia, in the same vein as any other clinical finding (e.g., a presbyope who is also a glaucoma suspect).

Presenting Myopia Management to Parents

Once myopia is uncovered, it's important to consider how to approach the parent. Should you say the following? "*Can I make a suggestion?* As an option, *what do you think* about coming back next week for some more tests?" If you wouldn't make vague suggestions to a glaucoma suspect, don't make them to the parent of a myopic child.

The conversation should sound more like this: "We need to get your daughter new glasses to make her vision clear. But we can't stop there, because her vision will keep getting worse. I want her to come back next week for some more tests because I'm concerned your daughter's eye is bigger than it should be. And if it continues to grow, she could have some trouble later on in life."

Discussing the Costs of Myopia Control

One way to think about the out-of-pocket costs of myopia care is to compare them to orthodontia, which is not usually covered by insurance. What are parents paying for at the orthodontist? They're not paying for rubber bands and wire; they're paying for the ability to give their kid a better smile, leading to an overall better quality of life. When a parent comes in to manage their child's myopia, they're not paying for contact lenses or eye drops; they're paying to give their child a better quality of life, and the ability to see the world for as long as their child is going to be alive.

I suggest setting a fee structure that is very easy for parents to understand. I would tell the parent something like, "It's going to cost you 'X' dollars to treat your child's myopia for the next 'Y' years." A global fee is definitely the "Staples Easy Button" and how I recommend you approach this.

Preparing the Practice for Myopia Control

Eye care providers must invest some time and money to start managing myopia, and it's important that your fees cover the additional expenses for technology, training, etc. With the right fee structure, treating myopia can be a highly profitable venture. Consider the following investments you'll need to make:

1. Technology to measure axial length An optical prescription is not a proxy for measuring axial length; you must have both pieces of information. Within the market of devices to measure axial length, Topcon Healthcare's devices are first-class and affordable, and I recommend all providers take a look at them.

2. Staff training There are hard costs to initially train the staff and provide ongoing training. Your costs will depend on whether you conduct training after hours, during staff meetings or close the office for a dedicated training session.

3. Opportunity costs Speaking to parents and doing myopia consultations takes time away from other tasks. However, myopia management can bring lifetime patients to your practice. It's important to consider the pros and cons of dedicating time to myopia management.

The Time is Now

We keep hearing that myopia management is the next big thing in optometry. It's not the next big thing in optometry; it's a big thing right now. With the United States alone having more than 15 million myopic kids and growing, and with half the planet expected to be myopic by 2050,[1] the time to jump on to this is now.

Gary Gerber, OD, is the co-founder of Treehouse Eyes, America's first centers dedicated exclusively to providing myopia management services, with 48 centers in the US and 60 planned by the end of 2021.

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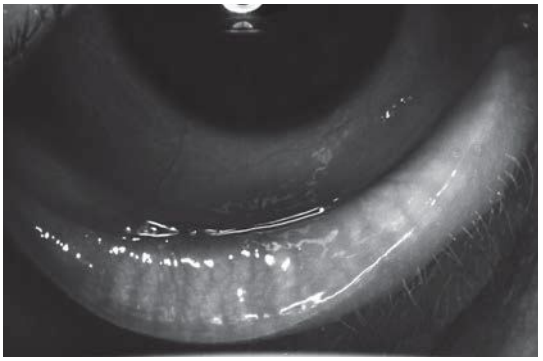

Meibography of a patient with severe dry eye disease. Note they have moderate atrophy of the glands with moderate truncation and loss of gland structure, OS greater than OD.

studies of patients with different underlying cause of dry eye and has provided more information and understanding of the tear film itself.

A previous study found that patients with both non-Sjögren's aqueous-deficient dry eye (ADDE) and MGD showed a very specific interferometric pattern that was different than those who were non-Sjögren's ADDE or MGD only.[8] Patients with only non-Sjögren's ADDE had a shortened NIBUT, an increased lipid layer thickness and a reduced tear secretion. In comparison, patients with only MGD had thin lipid layer but sufficient tear secretion and a shortened NIBUT.[8] This and other studies suggest that measuring these data points can potentially be used to diagnose and monitor dry eye disease.[10-12]

The LipiView II device (Johnson & Johnson) has both meibography and interferometry capabilities. Studies on the instrument's ability in the MGD are well documented; however, the diagnostic contribution to DED is not yet established.[6]

Corneal Sensitivity

Altered corneal sensitivity is not a new concept, but testing for it is widely underused. As a result, affected patients often go undiagnosed.

In dry eye, corneal sensitivity increases with disease severity.[6] In other conditions—most notably neurotrophic keratitis, surgery and trauma—sensitivity decreases. Testing corneal sensitivity can also help differentiate suspected herpes simplex keratitis from herpes zoster ophthalmicus.[13] Although quantitative measurements are not required, with newer treatments for neurotrophic keratitis like Oxervate (cenegermin-bkbj ophthalmic solution 0.002%, Dompé), some insurers could begin to require this in order to cover these medications.

While you can use a cotton wisp or floss to test corneal sensitivity, dedicated instruments exist to more quantitatively measure this, such as the Cochet-Bonnet (Western Ophthalmics) or non-contact air-jet esthesiometers. The handheld Cochet-Bonnet has a thin, retractable nylon monofilament, and the length can be adjusted anywhere from 5mm to 60mm, allowing the user to apply varying pressure. As the length is decreased the pressure increases and the length is recorded.[13]

The non-contact esthesiometer is better in measuring lower stimulus thresholds than the Cochet-Bonnett.[13] It assesses the corneal sensation threshold in an accurate and repeatable manner by measuring threshold sensitivity to a composite stimulus consisting of air pressure along with tear film evaporation and disruption.[13]

Tear Menicus Assessment

Located within the junction of the bulbar conjunctiva and eyelid margins, the tear meniscus is the fluid reservoir from which the precorneal tear film arises.[6] Patients with dry eye may experience a decrease in the menisci.[14] The easiest and most accessible way to measure it is via slit lamp exam using the beam; more high-tech methods include video-meniscometry and OCT meniscometry.[6] In anterior segment OCT applications, the high-resolution imaging produces detailed cross-section images of the anterior segment.[14,15] It can also gather information on tear meniscus height, depth and cross-sectional area measurements.[16]

Visual Fluctuation

Patients with ocular surface disease, particularly dry eye, often experience poor or fluctuating vision due to instability of the tear film—the eye's first (though admittedly least consequential) refractive medium. We typically elicit subjective reports of this phenomenon from the patient.

Those interested in gathering objective data can do so with the HD Analyzer (Visionmetrics/Keeler), which uses a laser diode to emit a light beam through the ocular media onto the retina; the light reflection is recorded by a camera. When there are irregularities in the air/tear film interface on the ocular surface, fluctuations in vision occur as light scatter.[17] This scatter is measured by the system as degraded retinal image quality.

The device quantifies the effect using a metric called objective scatter index (OSI).[18] The HD Analyzer measures changes in the OSI over a period of 20 seconds (taking one image every 0.5 second), with higher intraocular scatter correlating to higher OSI.[17] Normal OSI score is less than 1.[17] This is used to provide an analysis for the tear film quality and a concept called vision break-up time, which is the time it takes for a patient to lose one line of vision on the Snellen chart.[17] This approach is most revealing in cataract patients, given the greater significance of the crystalline lens in the ocular media, but can reveal distinctive patterns of tear film refractive quality as well.

Argentinean ophthalmologist Roger Zaldivar identified three tear film quality patterns that correlate with vision break-up time: (1) ladder (a continuous increase of OSI), (2) seesaw (instability of OSI without improvement after blinking) and (3) plateau (steady high OSI).[17] Plateau is normal, while the other two indicate abnormal tear film dynamics; the ladder pattern will have the highest OSI values.[17]

The HD Analyzer OSI scoring is also used to help determine whether a corneal or lens-based surgery is required.[18] Additionally, it is able to

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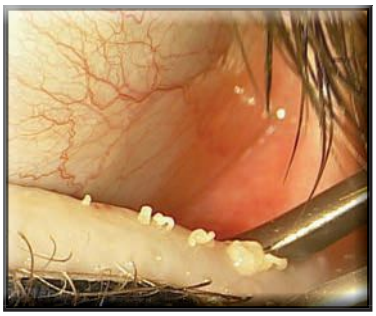

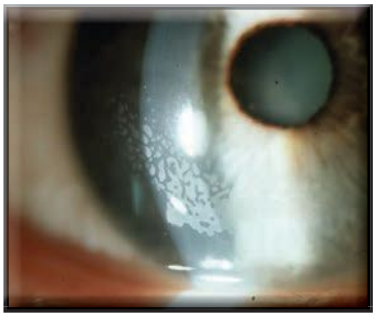

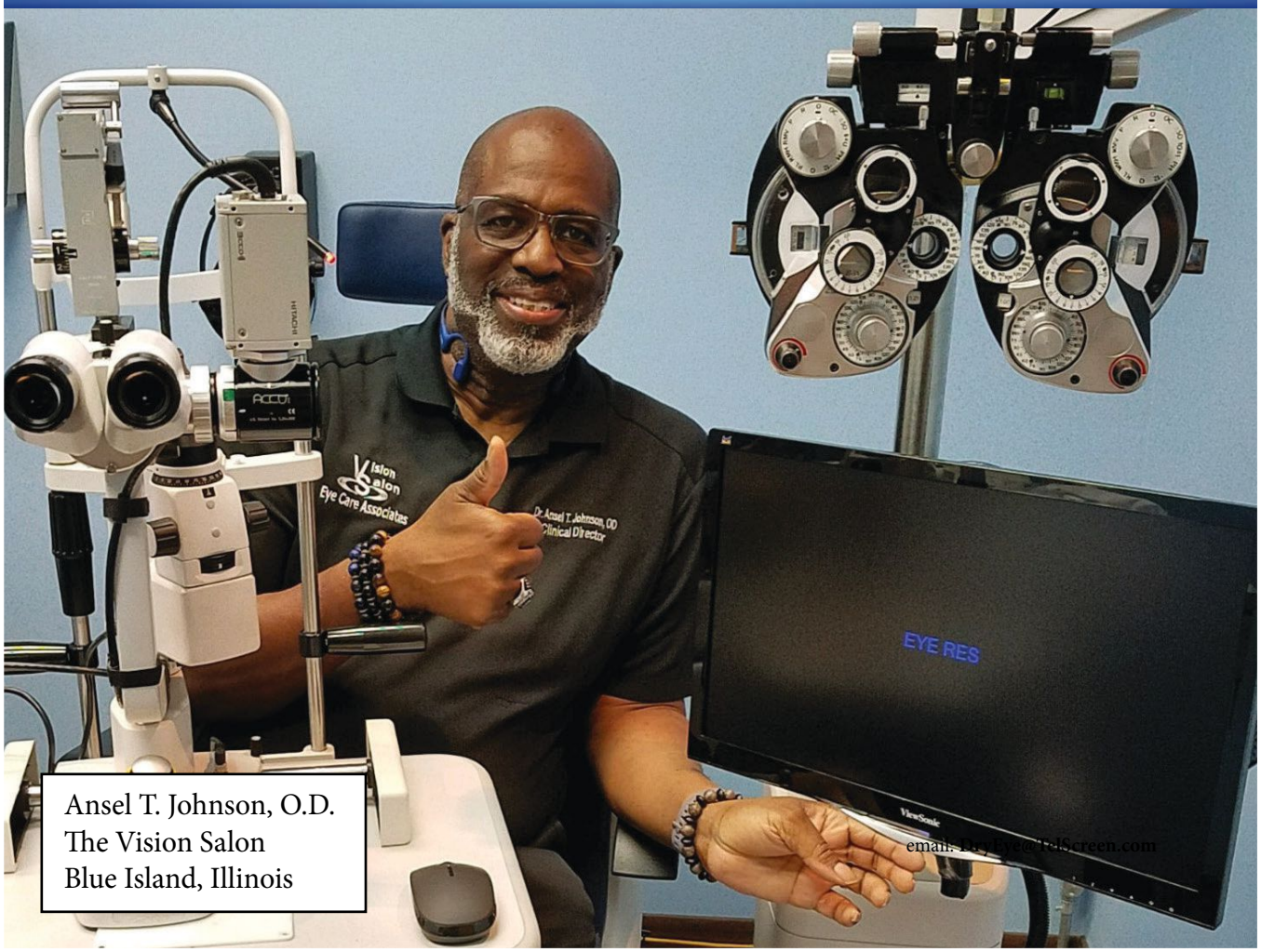

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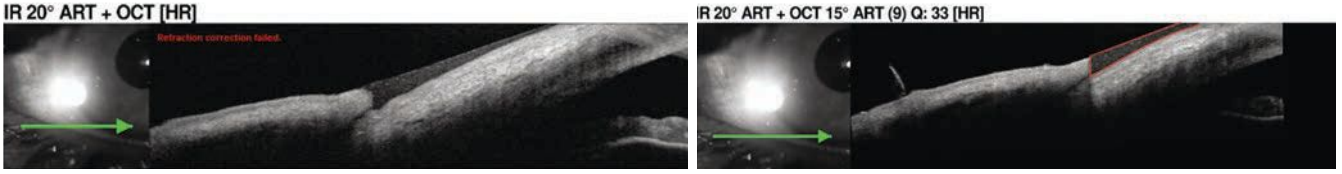

Tear meniscus measurement using the anterior segment lens on OCT. The tear meniscus is outlined in red in the right image, which can be further evaluated for quality.

perform high-definition images of the meibomian glands.[18]

How to Cover the Cost

When purchasing new technology, it's important to make sure it is a good investment for the clinic. If the equipment sits idle or fails to provide pertinent usable information, it could be a drain on a practice's resources. Currently, most advanced testing—similar to dry eye in-office treatments—is oftentimes reimbursed out-of-pocket. Many patients understand that having the right equipment and being able to provide a higher level of care can require additional expenses. Other times, we have to educate them on the importance of said testing so they see the added value it brings to their care.

Another thing to consider is the amount of reimbursement, even if you do decide to use the patient's insurance. In either case, it's good practice to have them sign an Advance Beneficiary Notice, which is a waiver of liability and should be given to the patient prior to providing the service. This ensures the patient is aware that the testing being given may or may not be covered and that they are financially responsible if it is not covered.

The CPT code 92285 is used for "external ocular photography with interpretation and report documentation of medical progress" and should be used for close-up photography, slit lamp photography, gonio photography, etc.[19] It is a bilateral code and is reimbursable by Medicare for the photo and interpretation.[19] These amounts are adjusted in each area by local wage indices.

Currently, the code 0330T is used for "tear film imaging, unilateral or bilateral with interpretation and report" and exists for tear film interferometry but is a Level III HCPCS code.[20] This means that it is used to track utilization and is reported to the carrier, but there is no current reimbursement associated with it. Since this code is more specific, it is to be used instead of CPT code 92285 for this testing.[20]

Similarly, the code 0507T is to be used for "near infrared dual imaging (*i.e.*, simultaneous reflective and transilluminated light) of meibomian glands, unilateral or bilateral, with interpretation and report" exists for meibography.[20] This is also a Level III HCPCS code and, for data collection, it's not reimbursable. Again, CPT code 92285 is not usable for this testing.

Regardless, if you decide to submit for reimbursement with the patient's insurance, don't forget to include your assessment in the patients plan or within their chart.

Is It Worth It?

Several of the advanced testing tools discussed here have more than one application—including some unrelated to dry eye. Those may be more obtainable and make more financial sense to add to your office's armamentarium. More applications means more use, possibly with better reimbursement. Higher volume practices or those specializing in dry eye or corneal disease would likely have more use of the more singular use tests. Facilities that partake in research may also benefit from having multiple of these as well.

While we can't have all the toys, there is no question as to the importance of the information that they can provide, even if some are more clinically relevant than others. It is important to look at your clinic, what technology is needed to help your patients now and what will help grow your practice into what you want it to be in the future. ■

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CAN AT-HOME MONITORING IMPROVE OPTOMETRIC CARE?

Doctors no longer make house calls, but devices can. These new tools can provide an abundance of data on how your patients do outside the exam room.

BY CATLIN NALLEY
CONTRIBUTING WRITER

While at-home monitoring is not a new concept in medicine—or optometry—recent years have seen an increasing interest in this technology and its role in patient care. This growing trend gained even more momentum during the COVID-19 pandemic with a change in how providers and patients both think about medical care.

"This shift toward more remote care is the future," notes Mohammad Rafieetary, OD, of the Charles Retina Institute in Memphis. "We will continue to see the expansion of telemedicine, and it is imperative that optometrists embrace new ways to provide care, including at-home diagnostic tools." And although incorporating these technologies into practice is not always simple, optometrists are in the position to take the lead when it comes to using these tools to enhance their approach to patient care.

So, how do ODs go about integrating these devices? It begins with a comprehensive understanding of what is available and how these tools can complement the care that is already available.

Photo: M&S Technologies

Melbourne Rapid Fields allow ODs to monitor patients between office visits.

Current Diagnostic Tools

There are a number of at-home diagnostic tools currently on the market and several others still in development. These devices can offer optometrists a way to improve the care they provide to patients with chronic conditions—such as moderately advanced glaucoma, intermediate age-related macular degeneration (AMD) and diabetic retinopathy—who could benefit from closer monitoring.

Glaucoma. The ability to more frequently evaluate patients with this chronic condition ensures ODs can adjust treatment plans quickly and avoid unnecessary complications. For instance, it often requires several visual field tests to see subtle changes in a patient, according to Southern College of Optometry's Paul Harris, OD. And so, depending on the frequency of office visits, it can take months to determine if the treatment plan needs adjustment.

With Melbourne Rapid Fields (M&S Technologies), a web-based visual field testing system, ODs can now monitor their patients between office visits. Dr. Harris, who has been involved in the testing of this device, notes that this system is an effective at-home option. "I'm highly impressed with the ability of the Melbourne Rapid Fields to do 24-2, 30-2 and 10-2," he says. "At-home monitoring is not only convenient for the patient, but also allows for more frequent progression analyses."

When starting a patient on this device, Dr. Harris and colleagues conduct the first test in the office to help patients understand correct usage. Then, they will have patients use the system once a week for the first month. This provides enough data for effective progression analyses moving forward. Following the first month, patients are advised to test once a month for two months and then once every three months after that.

The OD is notified whenever a patient uses the device, allowing them

to review the data and determine if further action is necessary. This may require a patient to up their frequency of use or schedule an office visit for further evaluation, according to Dr. Harris.

"This tool does not supplant office visits or the role of the optometrist," he emphasizes. "What this system actually does is increase the diagnostic skill of the eye care professional as well as the timeliness of intervention."

Another notable visual field analyzer intended for home use is the VisuAll (OllEyes), a virtual reality headset with models available for office or home use. The device is capable of administering all common testing protocols (*e.g.*, 24-2, 10-2, 30-2), according to the company. Testing time is about three minutes for threshold fields.

A different sort of device for glaucoma patients to use at home is Triggerfish (Sensimed)—a smart contact lens that continuously measures and records ocular changes correlated with intraocular pressure (IOP). The FDA granted market clearance in March 2016 for this device, which includes an antenna that wirelessly transfers data from the lens to a portable recording device. The resulting data provides the time of day the eye's volume is highest and lowest. It is important to note that the device does not directly measure IOP.

The ongoing measurement of IOP—a significant modifiable risk factor for glaucoma progression—is a critical aspect of disease management. However, measuring IOP in-office cannot always capture daily fluctuations and patterns. At-home IOP measurement tools, such as the iCare Home tonometer (CenterVue), could provide valuable data for providers. Patients self-administer the test and data is sent to a cloud server accessible by the doctor for evaluation.

Photo: CenterVue

The iCare Home uses rebound tonometry to obtain IOP measurements.

AMD. At-home monitoring has been a part of AMD management for decades in the form of the humble Amsler grid, which may help detect progression of dry AMD to the wet form of the disease when it is still in a treatable stage. However, it has some shortcomings, including patient adherence. With recent technological developments, there are now more advanced options that allow optometrists to monitor these patients for even the smallest changes.

The Foresee Home (NotalVision) device is a simple test that can identify the signs of progression. "I've been prescribing Foresee Home since it became FDA-approved," notes Joseph Pizzimenti, OD, of the Rosenberg School of Optometry. "It has been nothing short of a game-changer for identifying functional change due to new choroidal neovascularization in AMD. The result has been earlier detection, earlier treatment, better visual function and enhanced quality of life. In my opinion, there are no drawbacks to using this device."

For best results, Dr. Rafieetary recommends daily use. However, he acknowledges that may be challenging for some patients. "In those cases, I will advise that they use the device every other day or at least a few times per week."

Typically, candidates for this device have intermediate AMD, explains Dr. Rafieetary. "Changes should not be rapid, but rather occur slowly over time," he says. "The device alerts us to any changes in the map. At that point, we will decide next steps, such as further testing to determine if their disease has progressed."

Patients who have progressed to advanced AMD no longer need to use Foresee. However, if progression has only occurred in one eye, ongoing use of the device for the eye that remains in the intermediate stage can still be useful, notes Dr. Rafieetary.

Photo: Notal Vision

The Foresee Home helps identify AMD progression.

Given new technologies such as Foresee Home, is the Amsler grid still valuable? According to Dr. Rafieetary, there is still a role for this tool in AMD management as both a complement to newer technologies as well as an option for patients who may not be able to adhere to or afford more advanced devices. For improved detection and outcomes, consider objective means such as the Foresee Home and subjective means such as the Amsler grid, suggests Dr. Rafieetary.

Another area of AMD management where at-home devices are being explored is in optical coherence tomography (OCT). One such device currently under development is the Notal Home OCT (NotalVision), an artificial intelligence-enabled tool that monitors neovascular AMD. After each use by the patient, the device uses a machine-learning algorithm to scan for retinal fluid; if it's present, a report is generated and sent to the doctor.

This tool is another way to enhance our overall disease management strategy, says Dr. Rafieetary. "At this point, at-home OCT is only intended and being developed for better management of wet AMD patients undergoing

Photo: OllEyes

The VisuAll headset for field testing comes in models for office and home use.

treatment," he explains. "However, application of these devices can expand to other conditions such as diabetic retinopathy."

There are potential drawbacks to consider, he acknowledges, including cost and quality of image resolution. Nevertheless, he says the technology shows promise. These challenges will be addressed as technology continues to advance and it is important that ODs remain up to date on ongoing research and developmental efforts, according to Dr. Rafieetary.

Patient Education & Adherence

An important—yet often challenging—aspect of at-home diagnostics is patient adherence. The effectiveness of any device depends on consistent and correct use. To ensure patients are adequately prepared, ODs must provide comprehensive education and support.

This begins with a thorough explanation of the at-home device, including its purpose, expectations and proper use. "The best way to get started is in the exam room with clear and succinct patient education," says Dr. Pizzimenti. "Let the patient know that you are prescribing this method of at-home testing and explain why."

To encourage adherence, he emphasizes the important role these devices can play and the benefits for ocular health and overall well-being. "I stress to the patient that closer monitoring can save vision and enhance the quality of their life," notes Dr. Pizzimenti.

When Dr. Rafieetary introduces the Foresee Home to his AMD patients, he starts by explaining the intent behind the device. "You want your patient to have the right expectations and understand why this device is a beneficial addition to their management plan," he notes. "Take the time to walk your patient through the process and rely on the support the industry provides for further patient education."

As with any aspect of care, it is important to recognize the unique needs of your patients, suggests Dr. Harris. "Patient adherence is an issue across the entire medical field," he notes. "Success depends on spending time with your patients, listening to their needs and concerns and getting to know them as a human being."

ODs must also be prepared—when necessary—to change their approach. If, despite ongoing education and support, a patient cannot consistently adhere to using an at-home diagnostic tool, the optometrist may need to opt for more frequent office visits instead. As with any protocol, one size does not fit all and not every patient is the right candidate for at-home diagnostics.

Key Considerations

There are several considerations for the optometrist who wants to integrate these devices into their practice, including insurance coverage as well as legal and privacy concerns.

"Providers who are working with these devices and associated software need to ensure they are fully HIPAA-compliant," says Dr. Harris. "All data must be encrypted end-to-end."

Insurance coverage and reimbursement depends on the device. For instance, the Foresee home is covered by Medicare, notes Dr. Pizzimenti, who only bills for in-office visits.

As technology continues to advance, there are concerns that home-based devices could undermine the value of in-office care; however, that doesn't have to be the case. In fact, as the primary providers of eye care, ODs are in the position to integrate these tools into the foundation of their practice, ensuring that their patients receive timely and effective interventions.

"At-home diagnostics should complement what we already do for our patients, not compete with it," emphasizes Dr. Rafieetary. "There's no question that the growth of these devices is going to continue. If we become early adopters, then we can not only inform our patients, but also become better providers of care." ■

WILL AT-HOME REFRACTION EVER MAKE THE GRADE?

Another element of at-home diagnostics is the concept of online vision screenings. Over the last several years we have seen an influx of apps that offer patients a way to renew their prescription without a trip to the eye doctor.

Most recently, Warby Parker expanded its virtual eye exam offerings to include an updated app called Virtual Vision Test that allows users to use their iPhone to renew their glasses and contact lens prescriptions remotely.

"COVID-19 has served as a tailwind, and many optometrists who once denounced telemedicine technologies are now embracing them," notes Brian Chou, OD, of San Diego. "This shift has opened the door for online companies to introduce online sight testing to an industry that is now warmer to the idea."

Dr. Chou anticipates ongoing development in this space, making it easier for consumers to renew their prescriptions. And the logical assumption, he notes, is that more consumers will delay or opt out of in-person exams as a result. The danger of optimizing for at-home convenience is if it comes at the expense of quality and what is best for patients.

This is a real concern with significant implications that must be addressed, but ODs can position themselves for success and continue to provide care that is critical to a patient's overall ocular health and well-being.

"As a profession, we must accept that online prescription renewal is here to stay," Dr. Chou says. "So, it is up to us to continue to advocate for our patients as well as the field of optometry. ODs can and will continue to play an important role in eye care."

AMD Standard of Care is Not Enough

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20/83 VA

Average at wet AMD diagnosis according to IRIS Registry real-world data[1]

HOME STUDY

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References: 1. Rao P et al. *Ophthalmology*. 2018;125(4):522-528. **2.** Domalpally A, Clemons TE, Bressler SB, et al. *Ophthalmol Retina*. 2019;3(4):326-335.

See website for FDA Indication for Use.

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THE EYE/KIDNEY CONNECTION

Be vigilant for the ocular presentations of oculoretinal syndromes and renal disease.

JOSEPH J. PIZZIMENTI, OD[1] **AND CARLO PELINO, OD**[2]
[1] SAN ANTONIO
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Every optometrist has stories of patients whose systemic disease(s) was only diagnosed after the onset of ocular symptoms and signs. Systemic processes can impact virtually every ocular tissue, as well as the orbit and visual pathway. Early detection, diagnosis and treatment of both the ocular manifestations and the underlying systemic condition can lead to improved visual outcomes and a reduction in severe health complications and morbidity.

While optometrists would be unlikely to identify kidney disease through an eye exam alone, when we do encounter a patient with a known history of kidney disease or one of the oculorenal syndromes, we need to be vigilant for the ocular manifestations.

The eye and the kidney share developmental, physiological and pathogenic pathways. Both the glomerulus and choroid have vascular networks that are similar in structure. The renin–angiotensin–aldosterone system (RAAS) is found in both the kidney and in various ocular tissues. This system is an important regulator of blood volume and systemic vascular resistance. Renin, angiotensin II and aldosterone act to elevate arterial pressure in response to decreased renal blood pressure, decreased salt delivery to the distal convoluted tubule and/or beta-agonism. Through these mechanisms, the body can elevate the blood pressure in a prolonged manner.[6]

Photo: Anatomy & Physiology

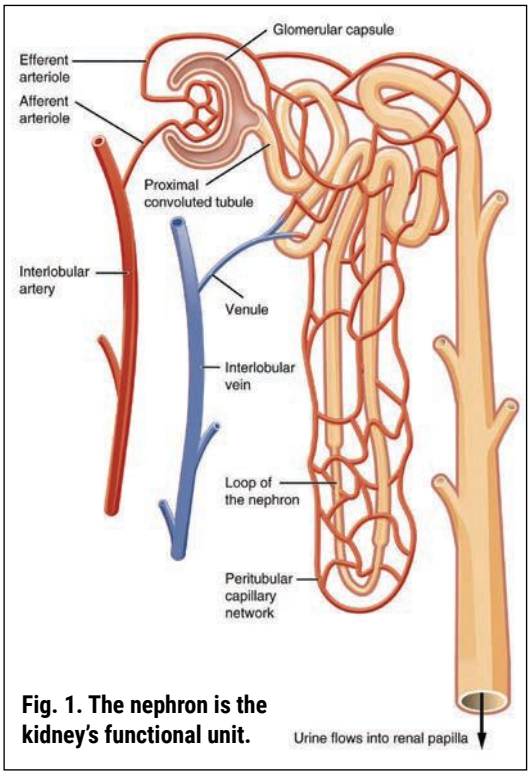

Fig. 1. The nephron is the kidney's functional unit.

In addition to the systemic RAAS, tissue-specific regulatory systems have been described in various organs, including the eye. These local regulatory systems, such as the one present in the retinal vascular endothelium, are responsible for physiologic changes. A local RAAS and its components have been detected in many structures of the human eye, from a possible role in aqueous humor dynamics and intraocular pressure to retinal vascular implications in hypertension and diabetes.[7]

Here, we will give you a clinical look at the eye in renal disease.

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THE KIDNEYS: FUNCTIONAL ANATOMY AND PATHOPHYSIOLOGY

Each of our two kidneys is about the size of a fist. Contained within each kidney are roughly a million nephrons. Comprised of a coiled tubule and an extensive network of capillaries, the nephron is the functional unit of the kidney (*Figure 1*).[1] Each nephron houses a special filter, or glomerulus, which filters our blood, removing toxins and excess water. Its thin walls allow smaller molecules, wastes and fluids to pass into the tubule, which ultimately is excreted as urine. Larger molecules, such as proteins and blood cells, do not get filtered and remain in the blood vessel. The renal tubule returns needed substances back to the blood stream. The two kidneys together filter about 200 liters of fluid every 24 hours.[2]

Renal health is central to maintaining correct balances and effective functioning of all the cells of the body. As cells break down, they produce acids. The extracellular matrix requires a stable composition of salts (such as sodium and potassium) and acidity. The foods we eat can either increase or lower the amount of acid in the body. Our kidneys balance the body's pH by either removing or adjusting the amounts of acid and buffering agents.[1,2]

The kidney is capable of detecting and responding to low levels of oxygen (hypoxia) through increased production of erythropoietin.[3] One adrenal gland sits on top of each kidney. These endocrine glands produce hormones that regulate metabolism, immune function, blood pressure and response to stress.[1,2]

Vitamin D has many roles in the body, including modulation of cell growth, neuromuscular function and glucose metabolism. Replacement of vitamin D in deficient populations by supplementation could reduce premature morbidity and mortality rates.[4] The kidneys are essential in helping the body use vitamin D. Evidence of a new, non-classical role for vitamin D has emerged to show regulation of the renin-angiotensin system and the nuclear factor (NF) κB pathway.[4] Chronically damaged kidneys have a weaker ability to convert vitamin D into its active form. Such patients may benefit from greater effort toward improving vitamin D intake (*Table 1*).

Most renal diseases attack the nephrons, leaving the kidney unable to remove waste. Causes of renal disease include genetic mutations, injuries and certain medicines. People are at a higher risk of kidney disease if they have hypertension, diabetes, heart disease or a first-degree relative with renal issues.[5] In chronic kidney disease (CKD), the nephrons are slowly and progressively damaged over many years. Some common diseases of the kidney include cysts, stones, infections and cancers.

The Oculorenal Syndromes

Interestingly, the period of organogenesis for both the eyes and the kidneys spans the fourth to sixth weeks of gestation.[8] Therefore, any deficits in embryogenesis during this time frame can cause anatomic and functional abnormalities in the two organs. A variety of ***human congenital oculorenal syndromes*** affecting both the eye and the kidney have been described.[9]

WAGR syndrome affects several body systems and is named for its main features: **W**ilms tumor, **a**niridia, **g**enitourinary anomalies and intellectual disability (formerly referred to as mental **r**etardation). Most people with WAGR syndrome have aniridia, typically the first noticeable sign.[10]

People with WAGR syndrome have a 45% to 60% chance of developing Wilms tumor, a rare form of kidney cancer that is most often diagnosed in children.[10] Patients with Wilms tumor are at an increased risk for developing ocular disorders, including aniridia and, less frequently, optic nerve hypoplasia resulting from inactivation of the aniridia gene Pax6.[10,11] Other ocular signs may also develop, such as cataracts, glaucoma and nystagmus.

The association of ocular coloboma with urinary anomalies may evoke a renal-coloboma or papillorenal syndrome, with coloboma involving the optic disc and adjacent retina. These patients have renal hypoplasia, with or without renal failure.[12]

Von Hippel-Lindau (VHL) syndrome is transmitted as an autosomal-dominant trait with variable penetrance. Its clinical manifestations include cerebellar and retinal hemangioblastomas, pancreatic cysts, renal cell carcinoma and pheochromocytoma.[13] The VHL gene is on chromosome 3 (3p25-26) and functions normally as a tumor suppressor by inhibiting transcription elongation.[14] Hemangioblastoma is a round, red tumor of the retina with a pair of feeding vessels showing an increase in diameter and tortuosity. It is benign.

Sturge-Weber syndrome (SWS) is a dermato-oculo-neural syndrome involving cutaneous facial nevus flammeus in the area of the first and/or second division of the trigeminal nerve, ipsilateral glaucoma, ipsilateral diffuse cavernous hemangioma of the choroid and ipsilateral leptomeningeal hemangioma.[15] The ocular component of SWS may also manifest vascular malformations of the conjunctiva, episclera, choroid and retina.

Tuberous sclerosis complex (TSC) is an autosomal-dominant disease with variable penetrance. It is a multisystem disorder characterized by the formation of angiomyolipomas or tubers affecting the brain, skin (called adenoma sebaceum), kidneys, eyes and heart.[8] The most suggestive ocular finding in TSC is retinal astrocytic hamartoma, found in 50% to 85% of patients (*Figure 2*).[8,16] Two different genetic loci have been identified in TSC: one on chromosome 9 (TSC1) and another on chromosome 16p (TSC2) that is immediately adjacent to the gene for the most common form of autosomal dominant polycystic kidney disease.[8,16]

TABLE 1. SOURCES OF VITAMIN D

Non-fat fortified milk	1 cup per day
Fish: salmon, tuna, sardines, mackerel, herring	At least three servings per week
"Sensible sunlight"	Five to 15 minutes, two to five times per week
Vitamin D3 supplements	1,000 IU per day

Bardet-Biedl syndrome (BBS) is a genetically heterogeneous disorder with the primary features of obesity, pigmentary retinopathy, polydactyly, renal malformations, intellectual disability and hypogenitalism. BBS is considered an autosomal recessive disorder that increases patient risk for developing diabetes, hypertension and congenital heart disease. Ocular abnormalities include rod-cone dystrophy, strabismus and cataract.[8,17]

Alport syndrome (AS). The classic phenotype as described by Alport is nephritis, often progressing to renal failure, and sensorineural hearing loss affecting both sexes in successive generations.[21] The ocular manifestations (present in 15% to 30%) of AS mainly involve the lens. Bilateral anterior lenticonus is the most cited specific abnormality.[8,19]

The Eye in Chronic Renal Disease

Chronic kidney disease (CKD) is characterized by a gradual loss of kidney function over time. CKD may be caused by uncontrolled diabetes. Hypertension causes CKD and CKD causes hypertension. Persistent proteinuria (protein in the urine) means CKD is present. High-risk groups for CKD include those with diabetes, hypertension and family history of kidney failure. African-Americans, Hispanics, Pacific Islanders, American Indians and seniors are at increased risk.

Glomerulonephritis is a family of diseases that cause inflammation and damage to the nephron. These disorders are the third most common type of kidney disease, after hypertension and diabetes. Inherited conditions, such as polycystic kidney disease, cause large cysts to form in the kidneys and damage of the surrounding tissue.

Lupus and other diseases that affect the immune system are associated with renal issues. Obstructions may be caused by problems like kidney stones, tumors or an enlarged prostate gland. Repeated urinary infections may also result in CKD.[20,21]

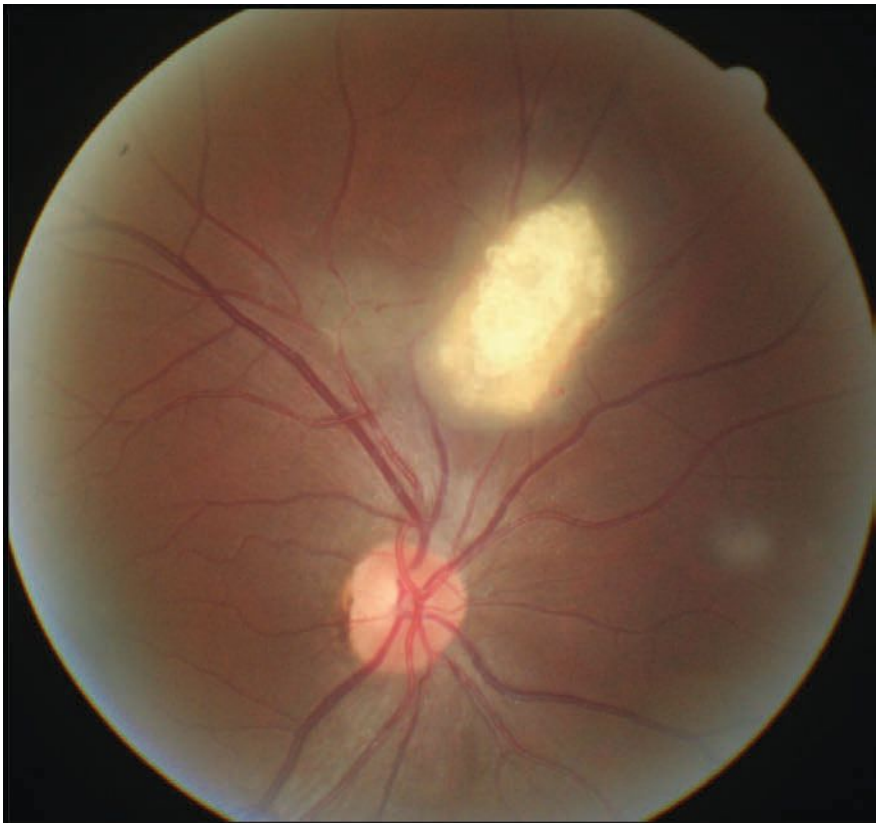

Fig. 2. The most suggestive ocular finding in TSC is retinal astrocytic hamartoma.

Chronic kidney disease has long been tied to eye disorders, including retinopathy (diabetic and hypertensive), glaucoma and cataract. Researchers recently found a high prevalence of visual impairment and major eye diseases in patients with chronic kidney disease (CKD)—and a strong association between the two. The investigators discovered that the prevalence of visual impairment and major eye diseases was two- to sevenfold higher in participants with CKD. They noted that CKD was associated with visual impairment, ocular disease and retinopathy, including diabetic retinopathy.[22]

In the Chronic Renal Insufficiency Cohort (CRIC) study group, investigators examined fundus photographs of 1,936 patients with varying stages of CKD.[23] They found that 45% had retinal microvascular abnormalities that required ophthalmic follow-up, while 3% had serious retinal vascular lesions that required urgent treatment.

Further results from CRIC indicated that a glomerular filtration rate (GFR) of <30mL/min per 1.73m^2 was associated with a three-times greater risk of developing retinopathy compared to patients with normal GFR.[24,25] Retinal vascular abnormalities may indicate the concurrent presence of macrovascular damage, such as cardiovascular disease (CVD), even after adjustment for renal dysfunction and traditional CVD risk factors.[25]

There was a strong association between the severity of retinopathy and kidney function. This association remained after adjustment for risk factors for CKD, suggesting that the retinal vascular changes reflect renal disease.[24,25] When we see rapid progression of retinal microvascular abnormalities on a dilated fundus examination, this suggests that renal function may be compromised and the patient needs referral to a nephrologist for assessment and management of the diabetic nephropathy. What is going on in

Photo: Jenny Terrell, OD

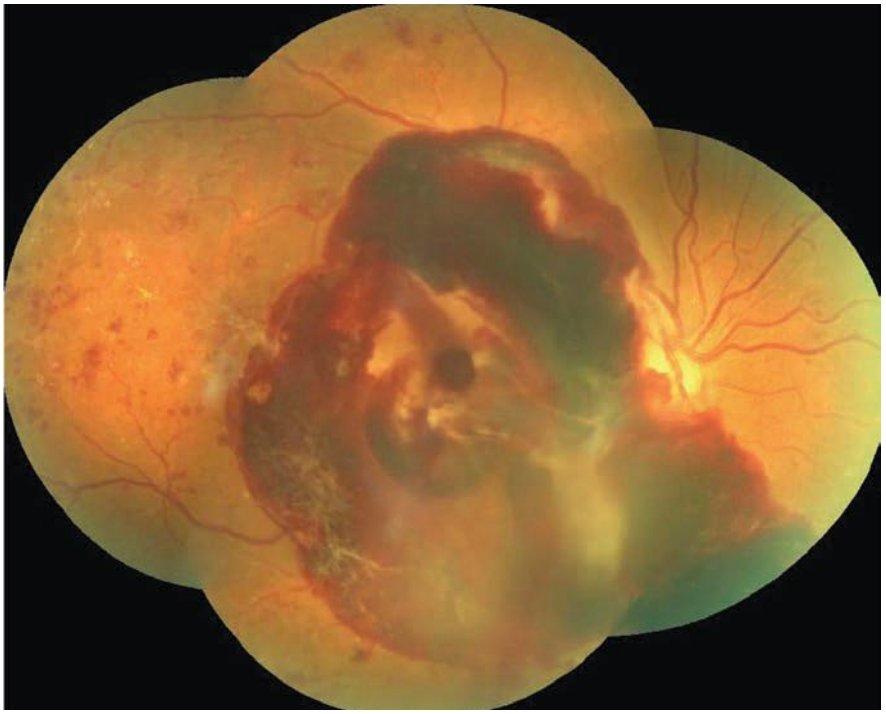

Fig. 3. Severe vision loss due to PDR more common in diabetic patients with chronic kidney disease.

the retinal ecosystem is likely also happening in the glomerulus. The assessment of retinal morphology using fundus imaging and OCT/OCT-angiography may prove valuable in future studies of CVD in those with CKD.

Severe vision loss due to proliferative diabetic retinopathy (PDR) or diabetic macular edema (DME) is about five times more common in diabetic patients with a glomerulopathy compared with non-albuminuric patients (*Figure 3*).[26] Hypertensive retinopathy can be particularly severe in renal failure, and accelerated hypertension may result in bilateral optic disc edema. Aggressive treatment for diabetic kidney disease might help prevent the progression of DR. Alternatively, patients treated with maintenance hemodialysis may experience systemic hypotension, a common side effect of ultrafiltration.[26,27] Bansal reported on two dialyzed patients who developed severe longstanding hypotension that suffered bilateral non-arteritic anterior ischemic optic neuropathy.[27]

Diabetic retinopathy patients with CKD require closer surveillance and more frequent or aggressive retinal treatment than those without. These treatments include intravitreal anti-VEGF agents, laser photocoagulation and, when necessary, surgical retina management.

One of the tests that may be of great value in patients with CKD and retinal vasculopathy is intravenous fluorescein angiography. Although generally considered safe for patients receiving dialysis, one manufacturer of fluorescein suggests using half the normal dose in dialyzed patients.[28]

In some types of transplantation (such as lung and liver), post-transplant malignancies tend to occur in the transplanted organ. In kidney transplantation, this does not appear to be the case. Patients who have undergone kidney transplantation are at a higher risk for squamous cell carcinoma (SCC), owing to postoperative immune suppression. The risk of developing SCC is about 100 times higher after a transplant. SCC and other post-kidney transplant malignancies may affect the various tissues of the eye, orbit and adnexa.[29]

Renal cell carcinoma metastasizing to the eye and orbit are rare but should be considered in differential diagnosis of mass lesions. In patients presenting with atypical orbital or ocular masses, the possibility of renal cell carcinoma metastasis should be examined, especially if there is a history of previous renal disorder.[30]

Diagnosis of Renal Disease

The National Kidney Foundation (NKF) and the National Kidney Disease Education Program (NKDEP) recommend that people at high risk be screened for kidney disease. The NKF recommends that everyone with diabetes between the ages of 12 and 70 be screened at least once a year. In addition to blood pressure and glycemic testing (HbA1c), serology and urinalysis may be performed to detect kidney disease. Specific tests of renal anatomic integrity and function include:

• ***Glomerular filtration rate (GFR):*** A common blood test that checks for CKD. The GFR indicates how well the kidneys are filtering. There are several methods to test GFR. Most commonly, the rate is estimated by measuring another substance. Many estimated glomerular filtration rate (eGFR) tests use a formula based on the levels of creatinine, a waste product produced by the body's muscles, in the blood. The eGFR result should be displayed in milliliters per minute per body surface area. In adults, a normal eGFR number is more than 90 milliliters per minute per 1.73 square meters of body surface area (mL/min/1/.73mm^2).

• ***Blood urea nitrogen (BUN) and creatinine testing:*** Frequently ordered together as part of a basic or comprehensive metabolic panel (BMP or CMP), these are groups of tests done to evaluate the health of major organs.

– *BUN:* This measures the amount of urea in a sample of blood. Urea is a waste product that forms as part of the body's natural process of breaking down proteins. It is also referred

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

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

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

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Use with Contact Lenses

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

ADVERSE REACTIONS

Clinical Trials Experience

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

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

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

USE IN SPECIFIC POPULATIONS

Pregnancy

Risk Summary

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

Data

Animal Data

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

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

Lactation

Risk Summary

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

Pediatric Use

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

Geriatric Use

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

PATIENT COUNSELING INFORMATION

Handling the Vial

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

Use with Contact Lenses

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

Administration

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

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to as urea nitrogen and is filtered out of the blood by the kidneys. When BUN levels are too high, it can be an indication that the kidneys are not functioning properly. In general, around 6mg/dL to 24mg/dL (2.1mmol/L to 8.5mmol/L) is considered normal.

– *Creatinine (blood and urine):* This test measures the amount of creatinine in the blood and/or urine. Reference range varies by sex and age. An increased creatinine level in the blood may mean that the kidneys are not working as they should.

• ***Urine albumin and albumin-creatinine ratio:*** This is used for screening and diagnosis of kidney disease. They can also help track the progression of disease and how well the kidneys respond to treatment.

– The protein albumin can pass into the urine if the glomerular capillaries suffer damage. This is similar to the pathogenesis of retinal exudate formation in diabetic retinopathy.

– For an albumin-to-creatinine ratio test, the result will generally be listed in milligrams of albumin per gram of creatinine (mg/g). For a 24-hour urine sample, the total grams of albumin in the full day's sample will be shown (g/day or g/24 hours).

– Abnormally high values on this pair of tests are an indication of renal microvascular disease.

• ***Imaging***

– Ultrasonography

– CT, MRI or CT urography

• ***Renal biopsy***

Management of Renal Disease

Depending on the underlying cause, some types of renal disease can be treated. Management usually consists of measures to control signs and symptoms, reduce complications and slow progression. Medications to lower the blood pressure—such as angiotensin-converting enzyme (ACE) inhibitors or angiotensin II receptor blockers—also help preserve kidney function.[20] Care should be taken, however, as these pharmacotherapies can initially change electrolyte levels and decrease kidney function.

A diuretic agent and low-salt diet may be recommended, especially if there is fluid retention. Statins may be prescribed, as people with CKD often experience dyslipidemia.[20] If the patient becomes anemic, the nephrologist may recommend supplements of the hormone erythropoietin, sometimes with added iron.[3,20] These will aid in production of more red blood cells, which may relieve the fatigue and weakness associated with anemia.

Treatment for end-stage kidney disease requires dialysis or a kidney transplant. Transplanted kidneys can come from deceased or living donors. Without such intervention, patients in complete or near-complete kidney failure have a life expectancy of only a few months.

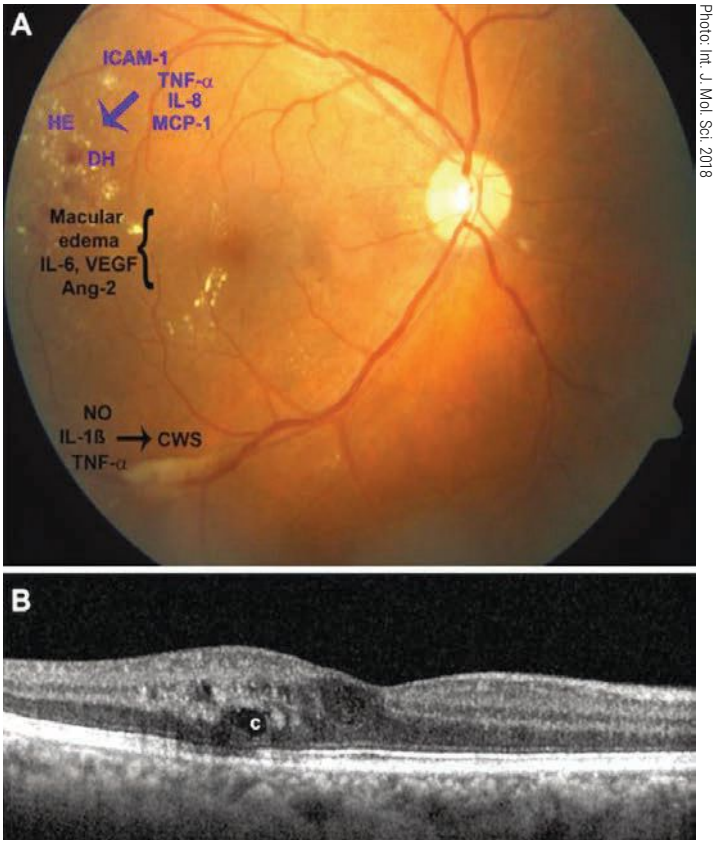

Photo: Int. J. Mol. Sci. 2018

Fig. 4. Interferon retinopathy clinical appearance and pro-inflammatory cytokines (A). Exudates and cystic edema on OCT (B).

The OD's Role

Optometrists can greatly contribute to the ocular and overall wellness of patients living with renal disease by following some essential tenets of care.

1. An annual comprehensive eye examination should be performed in patients with any renal condition, especially those with chronic or end-stage kidney disease. Examine the patient for signs of the oculorenal syndromes and retinal vascular disease.

2. Retinal vascular findings may reflect not only cardiovascular problems but also renal disease. The severity of retinal vasculopathy appears to vary with kidney function. Similar risk factors may be affecting the progression of both retinal and chronic renal disease.[2]

3. Chronic kidney disease impacts diabetic and hypertensive retinopathies. Aggressive treatment for diabetic kidney disease might prevent or slow the progression of diabetic and hypertensive retinopathies.[20,21] Facilitate a consultation with nephrology in these cases.

4. Intensive therapy of proteinuria may protect the eye. One study reported that remission of microalbuminuria with intensive therapy appeared to be a significant protective factor for the development of PDR and DME.[2]

5. Patients with chronic renal disease can lose vision. Researchers recently found a high prevalence of visual impairment and major eye diseases in patients with CKD and a strong association between the two.[22] Best-corrected vision of 20/40 or below

> **Optometrists can greatly contribute to the ocular and overall wellness of patients living with renal disease by following some essential tenets of care.**

was two to seven times more common in such patients. These findings highlight the importance of comprehensive eye examinations in CKD patients. There is a potential common pathogenesis underlying these conditions.[25,3]

6. Drugs being taken for renal disease have adverse ocular side effects. Diuretics have a strong link with progression of glaucoma. Calcium channel blockers have also been linked to progression of glaucoma. However, the ACE inhibitors may be neuroprotective as well as renoprotective.[31]

Interferon is used to treat various conditions including renal cell carcinoma. Interferon can lead to retinal damage appearing two to 12 weeks after the start of treatment (*Figure 4*). The retinopathy usually presents as cotton wool spots and retinal hemorrhages in the posterior pole.[32,33]

Most patients with early to moderate interferon retinopathy are asymptomatic. With that said, functional vision loss may occur and can be irreversible in some patients even after discontinuation of therapy. Branch retinal artery and vein occlusion, central retinal vein occlusion, cystoid macular edema and optic disc edema have all been associated with interferon therapy.[33] Patients with diabetes or hypertension or who are taking interferon are more likely to experience interferon retinopathy and should be closely monitored.

7. Drugs being taken for ocular diseases may require renal evaluation and monitoring. Most drugs and their active metabolites are eliminated through the kidneys. Therefore, all medications must be considered systemically potent in patients with poor renal function. Before prescribing a systemic agent, clinicians should question the patient about a history of renal disease. Systemic nonsteroidal anti-inflammatory drugs, steroids and antiviral agents are just some of the agents used in eye care that warrant further history and assessment of drug clearance ability.

Antiviral drugs cause renal failure through a variety of mechanisms. Direct renal tubular toxicity has been described with a number of medications with unique effects on epithelial cells of the kidney. Dosage adjustment according to renal function is indicated for many drugs.

The Bottom Line

To summarize, there exists significant evidence of a close association between renal and ocular wellness—and illness. Recognizing and understanding the links may ultimately lead to the development of new diagnostic and management strategies for both types of diseases. ■

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CRYSTALLINE KERATOPATHY: Etiologies, Ocular Manifestations and Treatment Protocols

Learn to distinguish the various clinical signs and perform the proper testing to uncover the underlying issue and initiate treatment.

BY LUANNE CHUBB, OD
PHILADELPHIA

A wide range of systemic diseases have ocular manifestations, sometimes presenting as the first or only sign of underlying illness. Crystalline keratopathy, though rare, is a corneal condition that could be signaling the presence of a number of systemic diseases, infections, disorders, adverse medication effects, cases of plant toxicity, ocular issues or other related causations (*Table 1*). The condition is defined as the deposition of crystals in the corneal epithelium, stroma and/or endothelium.[1]

The appearance of crystalline keratopathy in the cornea ranges from needle-shaped refractile polychromic crystals to dust-like, glittering granules. Imaging and observation techniques alone will not reveal the condition's culprit; rather, identification of the underlying cause of crystalline keratopathy will require a medical workup, including laboratory testing, biopsies and/or elimination of etiologies. This case report explains how to detect some of the condition's possible etiologies by describing the unique clinical signs, ocular manifestations and management options available for affected patients.

Case Report

An 84-year-old African-American male presented for a comprehensive examination. His previous ocular history was remarkable for bilateral uncomplicated cataract surgery five years ago, and his best-corrected visual acuities measured 20/20 in both eyes at distance and near. His external examination was normal and there was no afferent defect. His refraction revealed stable hyperopia with presbyopia. Biomicroscopy uncovered dense, bilateral, iridescent and polychromatic deposits within the full thickness of the corneal optic section from limbus to limbus. There was a greater concentration centrally. Intraocular pressures measured 16mm Hg, OU. Dilated fundus examination revealed normal posterior pole findings with no peripheral pathologies.

A cornea specialist on staff at Scheie Eye Institute (Stephen Orlin, MD) advised contacting the primary immediately to request the patient be worked up for multiple myeloma based on his age and previous laboratory results. The other differentials (*e.g.*, infection, toxic reaction, genetic inherited diseases) did not fit his age and he had no recent ocular surgery.

The crystalline keratopathy was photo-documented, and the patient was informed that laboratory work would be ordered through correspondence with his medical provider. Correspondence was sent explaining the finding of crystalline keratopathy and its common etiologies with recommendations for systemic laboratory work. A one-month follow-up ophthalmic appointment was scheduled to ensure follow through and complete a reevaluation ensuring condition stability.

The laboratory testing was positive for anemia, hypercalcemia, elevated creatinine and elevated serum total protein. Urinalysis results demonstrated elevated levels of total urine

About the author

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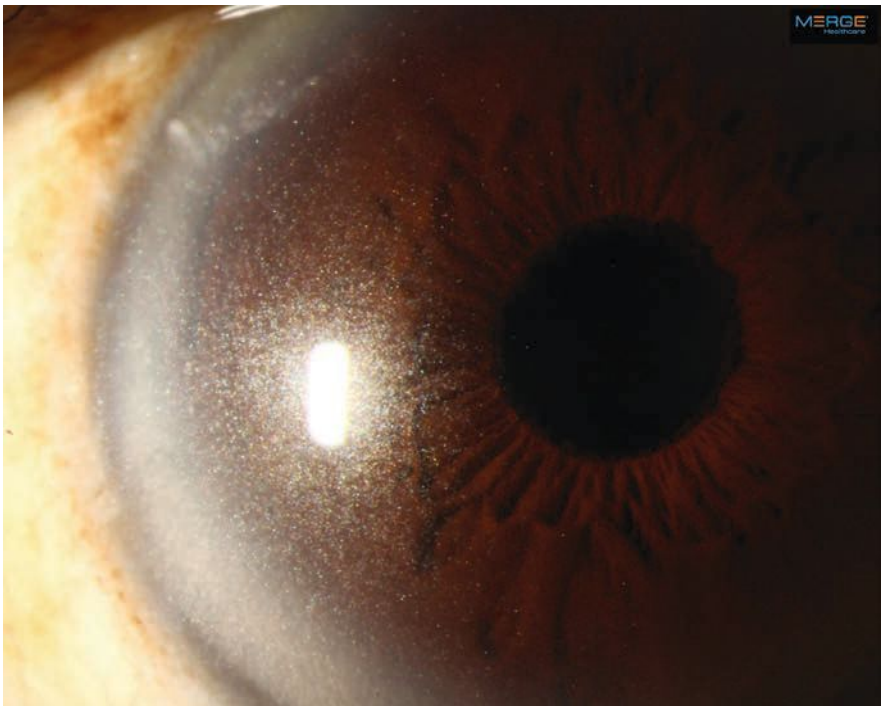
protein, urine creatinine, urine microalbumin and microalbumin/creatinine levels. Based on the laboratory results, the general practitioner, in collaboration with a hospital oncologist, arranged for hospital admission with scheduling of neuroimaging and a bone marrow biopsy. The bone marrow biopsy revealed IgG/kappa plasma cell myeloma. Overall test results were consistent with the diagnosis of stage III IgG kappa multiple myeloma with normal cytogenetics.

The patient was enrolled for a 28-day cycle of treatment with cyclophosphamide, bortezomib and dexamethasone (CyBorD). Acyclovir was added prophylactically for herpes zoster protection. The oncologist deferred the use of plasmapheresis to reduce the kappa light chains based on the patient's frail condition. Chemotherapy successfully reduced kappa light chains, and his vision and ocular comfort remained unaffected. Over the course of his systemic treatment, biomicroscopy revealed a reduction in the density of corneal crystal deposition.



The appearance of crystalline keratopathy in the cornea can range from looking like needles to small crystals or dust-like particles.

Discussion

Multiple myeloma (MM) is a malignant plasma cell dyscrasia in which clonal proliferation of B lymphocytes causes excessive levels of a single immunoglobulin referred to as a monoclonal or M-protein.[18-20] MM is the most common plasma cell malignancy, accounting for approximately 10% of hematological malignancies and 1% of all cancers.[19-22] The annual incidence is four per 100,000 people, with 85% diagnosed over age 65. It is more common in men than women and twice as common in the African race compared with Cacausians.[18-23] In fact, MM is the most common hematologic malignancy among Blacks in the United States, with a mean age of diagnosis four years earlier than that of whites.[23]

Monoclonal gammopathy of undetermined significance (MGUS) is an asymptomatic premalignant variation of MM defined by elevated serum or urine protein.[22] MGUS occurs in 3% to 4% of the population over age 50, with over 50% having the condition for as many as 10 years prior to diagnosis.[22] MGUS can be diagnosed with routine laboratory testing. Its occurrence is two times higher in African-Americans than in Caucasians.[20-23] Patients with MGUS progress to MM at a rate of 1% per year.[20-23,26] Twenty percent of patients diagnosed with MGUS convert to myeloma, amyloid or lymphoma within 20 years, and 70% of those patients monitored with periodic laboratory testing develop MM.[21,22,25-27]

'Smoldering MM' is a designated term for cases in the advanced asymptomatic premalignant phase.[22,28] It has a variable rate of progression to malignancy; 10% per year for the first five years, 3% per year for the next five years and 1.5% per year after that.[21,22,25,28] Those identified with high-risk cytogenetic abnormalities progress more rapidly, having up to a 50% risk of malignancy within two years.[22,28]

TABLE 1. KNOWN ETIOLOGIES FOR CRYSTALLINE KERATOPATHY [1,18]

Lipid metabolism disorders	Schnyder's corneal dystrophy, Tangier's disease and familial lecithin-cholesterol acyltransferase deficiency
Amino acid protein metabolism disorders	Tyrosinemia, cystinosis
Metabolic disorders	Hyperuricemia, gout, Bietti corneal dystrophy
Plasma cell dyscrasias	Monoclonal gammopathy, multiple myeloma, Waldenström macroglobulinemia, solitary plasmacytoma, systemic amyloid light chain amyloidosis and POEMS syndrome
Infections	Bacteria, fungi
Toxic reactions	Certain plants
Adverse effects from medication	Amiodarone, aminoquinolines, antibiotics, nonsteroidal anti-inflammatory agents, phenothiazines, antineoplastic drugs, rifabutin, gold salts and antibody-drug conjugates

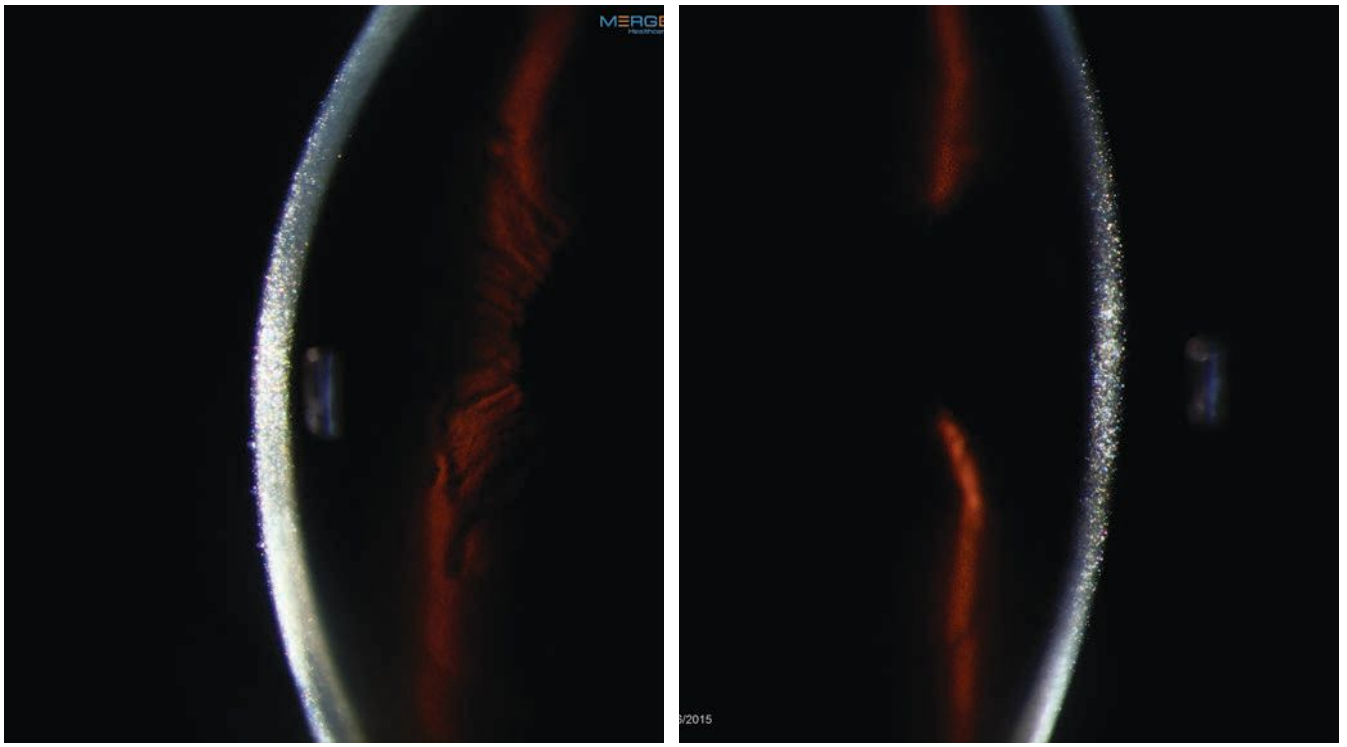


Photo: Scheie Eye Institute, University of Pennsylvania

Biomicroscopy revealed dense, bilateral, iridescent and polychromatic deposits within the full thickness of the corneal optic section.

Symptoms of MM include bone pain, fatigue, weakness and weight loss, with characteristic clinical findings of hypercalcemia, anemia, renal insufficiency and lytic bone lesions with increased risk of infection.[19,21,22,25,27] Imaging studies show diffuse osteoporosis and/or lytic lesions of the red marrow-bearing bones including the skull, spine, ribs, proximal extremities and pelvis. This increases the risk of pathological fractures in these bones.[18,19,22] Increased numbers of plasma cells can also be observed upon bone marrow biopsy.[18,19,22] Experts use fluorescent *in situ* hybridization (FISH) probes for cytogenetic classification to identify those with high-risk abnormalities.[18,19,22]

Ocular manifestations of MM are rare but have been reported in every ocular structure except the lens.[18,19,29-31] Crystalline keratopathy occurs in less than 1% of gammopathy cases and results from the crystallization of the M-protein within the cornea.[19,30,31] The deposits are predominantly from IgG kappa light chains.[19,30-32] The crystals themselves are benign but have the potential, as they amass, to cause decreased vision by altering the optics of the cornea, producing pain and photophobia through inflammatory pathways. They may also remain without causing any symptoms.[27,32-34] Their widely variable presentation of color, shape, appearance, layer of cornea involved and pattern of distribution can mimic common corneal dystrophies inciting ineffective clinical managements, delaying medical testing capable of identifying the proper systemic diagnosis.[2,5,16,18,30]

Though the etiology of the depositions is not certain, proposed mechanisms include immunoprotein transport via the tear film, diffusion from aqueous humor, increased permeability of limbal vessels and synthesis within the keratocytes.[26,34,35]

Confocal microscopy studies have described the crystal shapes as granular, globular and needle-shaped in appearance.[17,18,32,33] The typical shape identified on electron microscopy has been hexagonal with central cores and tubulars with a central lucent core.[34,35]

The immunoglobulin deposits have a strong affinity to draw copper into the cornea, further contributing to corneal irregularities and visual degradation.[30,31] Central circular yellow-brown discoloration of the cornea results from pigmentation of Descemet's membrane, and the copper deposition may extend to the lens capsule. This deposition pattern is different from the annular copper deposits found peripherally in Descemet's membrane in those with Wilson's Disease.[31]

Ciliary body cysts are associated with MM and occur in 33% to 50% of patients.[19,29,30] Most are not detected on clinical examination due to location and transparency.[19,29,30]

Retinal findings associated with MM include microaneurysms, cotton wool spots, dilated veins, intraretinal hemorrhages, Roth spots, vein occlusions and optic disc edema (all of which should demand an order for laboratory work to screen for diabetes, hypertension, coagulopathy, hyperviscosity, dyslipidemia, cardiac, carotid and giant cell etiologies).[19,29,30]

Neuro-ophthalmic manifestations include diplopia from plasma cell infiltration of the ocular motor nerves, proptosis resulting from plasmacytomas in the soft tissue or orbital bones and vision loss from optic nerve involvement.[19,30]

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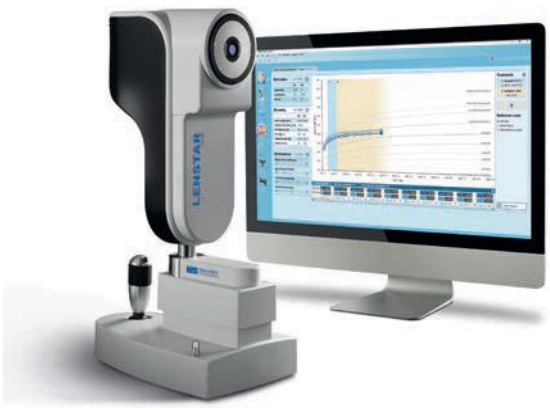

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Differential Diagnosis

Since crystalline keratopathies may originate from other etiologies, these must be ruled out.[1-5,36] Infectious crystalline keratopathy (ICK) is a slowly progressive discrete keratitis characterized by needle-like branching opacities. ICK does not occur as a primary disease; it results as a complication after an epithelial defect from injury or surgery and is always symptomatic. ICK has been reported from all of the following: *Streptococcus viridans*, *Staphylococcus epidermis*, *Streptococcus pneumoniae*, *Haemophilus*, *Enterococcus*, *Candida tropicalis* and *Acanthamoeba*.[2,4,5,13,37,38,39]

Corneal granular dystrophies are a group of bilateral non-inflammatory genetic corneal diseases, the majority having no systemic association.[2,3,36] Their crumb-like opacities are often mistaken for crystalline deposits.[36]

Progressive inherited systemic disorders may also cause the precipitation of corneal crystals, but these diseases occur earlier in life.[2,3,7] Schnyder crystalline keratopathy is an autosomal-dominant, slowly progressing dystrophy that occurs in the first to second decade of life. Genetic abnormalities in lipid metabolism may cause high levels of cholesterol and phospholipid corneal deposits which increase the risk of corneal erosion.[2,3,5,17,40-42]

Bietti corneoretinal dystrophy is an autosomal-recessive disorder that typically occurs after the second decade of life.[2,11,12,17] Presentation is characterized by bilateral crystalline deposits in the cornea and retina with progressive chorioretinal degeneration, night blindness and visual field loss.[2,11,12,17] Gout is the most common inflammatory joint disease in men over 40. Monosodium urate crystals deposit in the joints and tendons from longstanding hyperuricemia.[3,10] Ocular inflammation is rare but can occur during an acute attack. Uric acid crystal deposits may occur in any ocular tissue or adnexa, causing symptoms such as burning pain, redness and decreased vision.[3,10,13,17] Crystal deposits may be found in the corneal epithelium, stroma and Bowman's layer and appear as a orange-brown band keratopathy.[3,10]

Familial lecithin-cholesterol acyltransferase deficiency is an autosomal recessive disorder that causes unesterified cholesterol to accumulate resulting in atherosclerosis, renal insufficiency and crystalline keratopathy.[3,17] Peripheral corneal arcus with stromal haze is often a concurrent finding.[3,17]

Cystinosis is another rare metabolic disease that causes the accumulation of the amino acid cystine within the lysosomes of cells. Crystal deposition occurs in many tissues with the kidneys and eyes the most vulnerable.

There are three clinic presentations of cystinosis based on age and severity of renal disease. *Nephropathic cystinosis*, the most common and severe form, occurs in infancy and leads to renal tubular Fanconi syndrome by six to 12 months of age and failure to thrive.[7,8] *Late-onset, or intermediate, cystinosis* occurs in late childhood or early adolescence. Renal disease progression is slower with less effect on growth. *Non-nephrotic cystinosis* presents in adulthood with severe photophobia secondary to crystal deposition in the cornea. Other organs are not affected and kidney function is not compromised.[7,8] Severe photophobia and recurrent corneal erosions are notable in this disease and often lead to the need for a corneal transplant.[3,7,9]

TABLE 2. DIFFERENTIALS OF CRYSTALLINE KERATOPATHY [3,13-15,18,24,32]

Infectious	· Bacterial · Viral · Fungal
Drugs	· Amiodarone · Aminoquinolines · Chlorpromazine · Clarithromycin · Clofazimine · Gold salts · NSAIDs · Phenothiazines · Retinoids · Rifabutin · Suramin · Tamoxifen · Topical fluoroquinolones
Toxic reaction	· *Dieffenbachia* houseplant
Lipid and lipoprotein metabolism disorders	· Schnyder's corneal dystrophy · Tangier disease · Lecithin-cholesterol acyltransferase · Familial lipoprotein disorder
Amino acid metabolism disorders	· Tyrosinemia · Cystinosis
Miscellaneous dystrophy and metabolic abnormalities	· Bietti corneal dystrophy · Posterior crystalline corneal dystrophy hyperuricemia · Gout · Calcium deposits · Porphyria · Oxalosis
Plasma cell dyscrasias	· Monoclonal gammopathy of undetermined significance · Multiple myeloma · Waldenström macroglobulinemia · Cryoglobulinemia · Solitary plasmacytoma · Systemic amyloid light chain amyloidosis · POEMS syndrome

Management

Multiple myeloma (MM) is not curable; it is a treatable malignancy with a median survival rate of five to eight

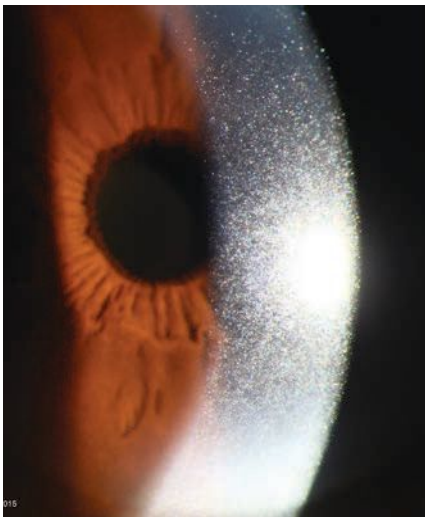
years.[21,22,25,43,45] The survival rate of MM depends on tumor stage, cytogenic abnormalities and response to therapy.[21,25]

Autologous stem cell transplantation (ASCT) increases median survival rate, but eligibility is based on age and comorbidities.[20-22,43] The four-year survival rate for this intervention is as high as 80% for those eligible patients, with a median overall survival rate of eight years.[45] In the United States, the age limit for ASCT is flexible based upon physiological rather than chronological age, so long as few comorbidities are present.[20,21] Most European countries have an age limit of less than 65 years.[21,43]

Chemotherapy for MM is tailored by the eligibility for autologous stem cell transplantation, and survival rates have improved over the last decade. Eligibility has also increased, as the FDA has approved new chemotherapeutic agents.[21,22,25]

In response to chemotherapy and/or plasma exchange, a decrease in crystal density and improvement of ocular symptoms will occur as the serum immunoglobulin level decreases. Not all patients will have improvement or reduction in the corneal crystals. One study reported continued increased deposition despite systemic treatment.[33-35] There are no topical ophthalmic agents that provide treatment. Topical ophthalmic agents such as artificial tears are palliative only.[35,44] Confocal microscopy can be used to monitor the density of crystals as chemotherapy proceeds.[33]

Severely affected patients with functional vision reductions or chronic pain may require penetrating keratoplasty; unfortunately, these cases have a high risk of graft failure due to recurrence of stromal corneal opacities.[35] A small number of patients may require a keratoprosthesis. Chiang et al. reported on the primary use of the Boston Type 1 keratoprosthesis, bilaterally, for a patient with severe crystalline keratopathy with vision loss.[35] Visual acuity was maintained at one year of follow-up.[3] Further studies using keratoprosthesis over penetrating keratoplasty are limited due to few patient numbers, low frequency of severe corneal disease and risk of complications secondary to the keratoprosthesis.[35]



Treatment options for CK vary depending on the underlying cause. In some cases, such as with MM, the condition may only be treatable and not curable.

Conclusion

Crystalline keratopathy has multiple etiologies, some of which are benign, and others which are linked to potentially fatal systemic diseases. The condition itself often goes unnoticed to the patient until it progresses and produces symptoms and pain that require intervention, which can range from supportive lubricants and advanced keratoprosthesis to corneal replacement procedures. Promptly once the condition identified, a systemic workup for general illness and coagulopathy/hyperviscosity disease is warranted. Multiple myeloma is the most common of the plasma cell malignancies, as it accounts for approximately 10% of hematological malignancies, 1% of all cancers and is associated with crystalline keratopathy. It is more common in those of African descent compared with Caucasians. Although MM is not curable, early diagnosis and treatment improves quality of life and increases overall survival rate.

Any instance of crystalline keratopathy you come across in your practice calls for an immediate protocol of monitoring, testing and elimination of possible causes to identify which systemic disease, infection or other underlying issue is behind the corneal manifestation. Initiating the appropriate treatment as soon an etiology is recognized will undoubtedly improve patient outcomes. ■

TABLE 3. DIAGNOSTIC CRITERIA FOR MULTIPLE MYELOMA [21,22,45]

From the International Myeloma Working Group

Non-IgM monoclonal gammopathy of undetermined significance (MGUS)	All three criteria must be met: - Serum monoclonal protein (non-IgM type) <3gm/dL - Clonal bone marrow plasma cells <10% - Absence of end-organ damage such as hypercalcemia, renal insufficiency, anemia, bone lesions (CRAB) that can be attributed to the plasma cell proliferative disorder
Smoldering multiple myeloma	Both criteria must be met: - Serum monoclonal protein (IgG or IgA) ≥3g/dL, or urinary monoclonal protein ≥500mg per 24 hours and/or clonal bone marrow plasma cells 10%-60% - Absence of myeloma defining events or amyloidosis
Multiple myeloma	Both criteria must be met: - Clonal bone marrow plasma cells ≥10% or biopsy-proven bony or extramedullary plasmacytoma - Any one or more of the following myeloma-defining events: · Evidence of end organ damage that can be attributed to an underlying plasma cell proliferative disorder, specifically: · Hypercalcemia: serum calcium >0.25mmol/L (>1mg/dL) higher than the upper limit of normal or >2.75mmol/L (>11mg/dL) · Renal insufficiency: creatinine clearance <40mL per minute or serum creatinine >177µmol/L (>2mg/dL) · Anemia: hemoglobin value of >2g/dL below the lower limit of normal, or a hemoglobin value <10 g/dL · Bone lesions: one or more osteolytic lesions on skeletal radiography, computed tomography (CT), or positron emission tomography-CT (PET-CT) · Clonal bone marrow plasma cell percentage ≥60% · Involved: uninvolved serum free light chain (FLC) ratio ≥100 (involved free light chain level must be ≥100mg/L) · >1 focal lesion on magnetic resonance imaging (MRI) studies (at least 5mm in size)

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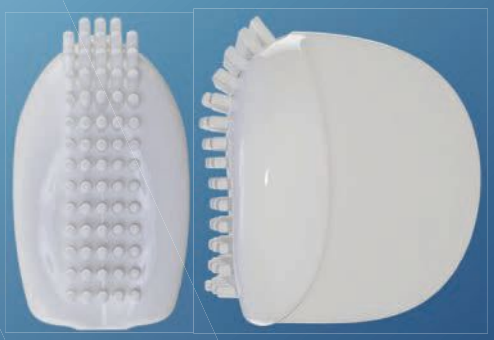

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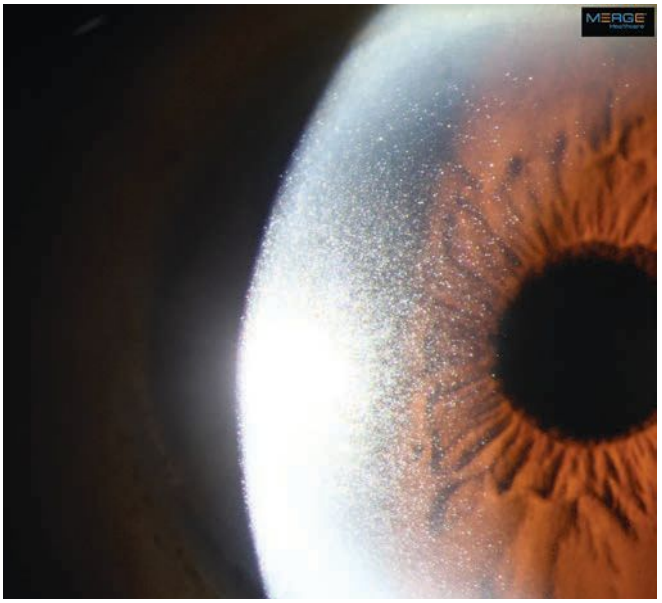

Cases of crystalline keratopathy caught early may save patients from enduring further health consequences down the line as the result of an undiagnosed systemic disease.

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WHEN YOUR PATIENT COMPLAINS OF OCULAR PAIN

Asking the right questions is key to successfully sizing up this common symptom.

BY ALISON BOZUNG, OD
MIAMI

Discomfort in and around the eye is one of the most common complaints we receive in clinical practice. However, it's also one of the least useful in terms of diagnostic guidance. How can we localize the cause? Which instances might be more troubling than others? How can we filter out the patient's subjective experience—some are far more pain-tolerant than others—when determining the urgency of our response?

The process begins by understanding what the term "pain" actually entails. Pain is defined as "a localized or generalized unpleasant bodily sensation or complex of sensations that causes mild to severe physical discomfort and emotional distress and typically results from bodily disorder (such as injury or disease)."[1] It is a type of somatic sensation that is recognized when a stimulus supersedes a particular "pain threshold."

When a stimulus is determined to be of a severity that could cause tissue damage, an afferent protective reaction is triggered. This reaction, for example, could include a person pulling their arm away from an abrasive surface or blinking their eyes when discomfort is felt. In the body, nociceptors are the specialized, unencapsulated nerve endings responsible for receiving and relaying information that is thought to be potentially harmful to the body. These stimuli include high and low temperatures, mechanical touch and exposure to chemical or inflammatory mediators.[2]

The first division of the trigeminal nerve, the ophthalmic branch (CN V1), is responsible for transmitting sensory information from the conjunctiva, sclera, cornea and upper eyelid. The ocular surface specifically detects noxious stimuli through mechanoreceptors, polymodal nociceptors and thermoreceptors.[3]

CN V1, however, is not limited to the ocular structures. It also provides innervation for the scalp, dura of the anterior cranial fossa, tentorium cerebelli, falx cerebri, superior sagittal sinus and some intracranial vessels. Therefore, inflammation or dysregulation in any of these associated areas can be referred and thereby perceived by an individual as eye pain.[4,5]

Due to the non-localizing symptom of eye pain, our examination of the patient becomes critical in determining the trigger. This article will help us answer the perennial question, "Doc, can you explain my eye pain?"

Categorizing and Qualifying Ocular Pain

When evaluating a patient who presents with eye pain, qualifying their symptoms should guide the practitioner's examination. For example, a patient presenting with eye pain after working on the computer for eight hours is likely experiencing dry eye. If the patient describes pain that is significantly worse when exposed to

About the author **Dr. Bozung** practices at the Bascom Palmer Eye Institute in Miami, where she primarily sees patients in the hospital's 24/7 ophthalmic emergency department. She serves as the associate director of the optometric residency program and is the clinical site director of the optometric student externship program. She has no financial interests to disclose.

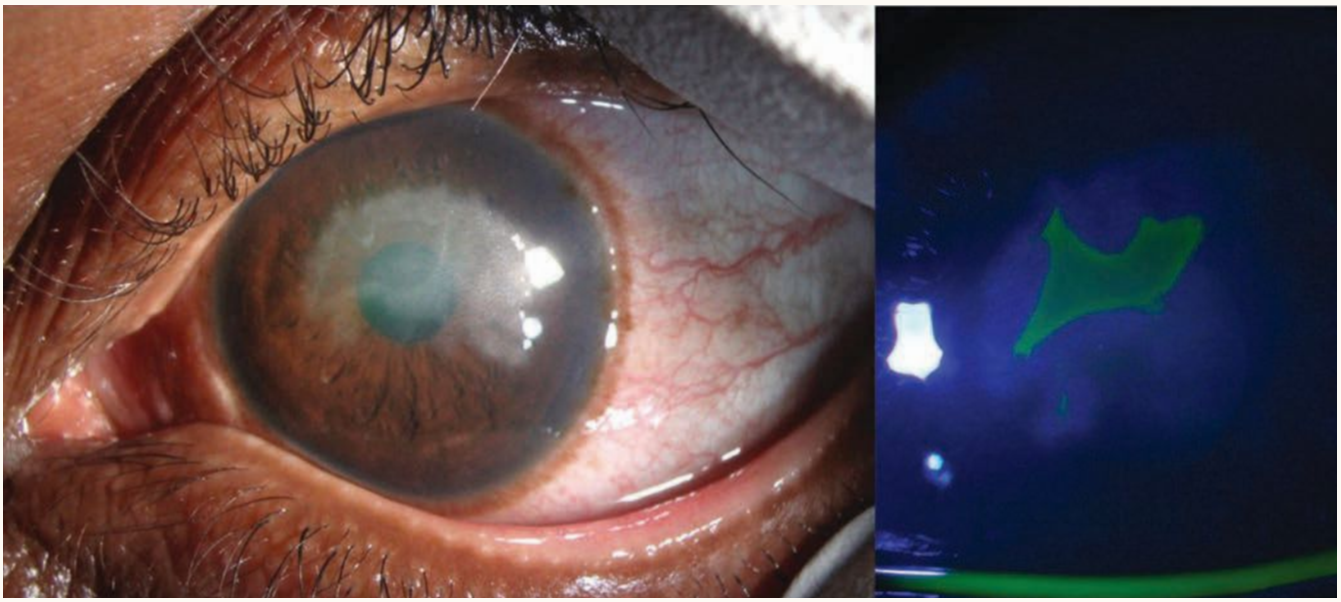

Fig. 1. This patient presented with blurred vision but did not complain of significant pain. His examination revealed a geographic area of corneal haze with an overlying epithelial defect. He has a past medical history of recent irradiation to the ipsilateral jaw for adenoid cystic carcinoma of the salivary gland. Due to the geographic appearance of the defect, this patient was started on an oral antiviral along with intense lubrication of the eye.

bright light, the doctor should assess them for corneal ulceration or abrasion or uveitis.

The patient's ophthalmic history is also a critical piece of the puzzle. If this patient recently experienced ophthalmic trauma, what was the mechanism of said injury? Did they recently have cataract surgery and are they experiencing discomfort after tapering corticosteroid drops too quickly? Additionally, ask qualifying questions regarding the pain to determine the length of time for which it has been present and if there are any relieving factors.

We can mentally categorize ocular discomfort into external or internal ocular, orbital or referred pain. External ocular pain includes causes such as corneal abrasions, dry eye and corneal ulcers. Internal ocular pain refers to conditions such as acute angle closure, uveitis and acutely elevated intraocular pressure (IOP). Orbital pain can result from periocular trauma, inflammation or a retrobulbar mass.

Referred ocular pain does not originate within the periocular structures; rather, it is caused by a stimulus from somewhere within the ophthalmic division of the trigeminal nerve's innervation territory.[4] Below, we will introduce and discuss common causes of ocular pain in relation to the structure of the eye and adnexa.

External Causes of Ocular Pain

The first category we will explore is external pain, which can be associated with a number of causes:

Release Date: September 15, 2021

Expiration Date: September 15, 2024

Estimated Time to Complete Activity: 2 hours

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

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

- Recognize the various ocular conditions that can present with pain.
- Understand how to localize the source of the pain.
- Ask the right questions when a patient complains of ocular pain.
- Narrow the diagnosis through clinical exam findings.

Target Audience: This activity is intended for optometrists engaged in ocular pain management.

Accreditation Statement: In support of improving patient care, this activity has been planned and implemented by the Postgraduate Institute for Medicine and Review Education Group. Postgraduate Institute for Medicine is jointly accredited by the Accreditation Council for Continuing Medical Education (ACCME), the Accreditation Council for Pharmacy Education (ACPE) and the American Nurses Credentialing Center (ANCC) to provide continuing education for the healthcare team. Postgraduate Institute for Medicine is accredited by COPE to provide continuing education to optometrists.

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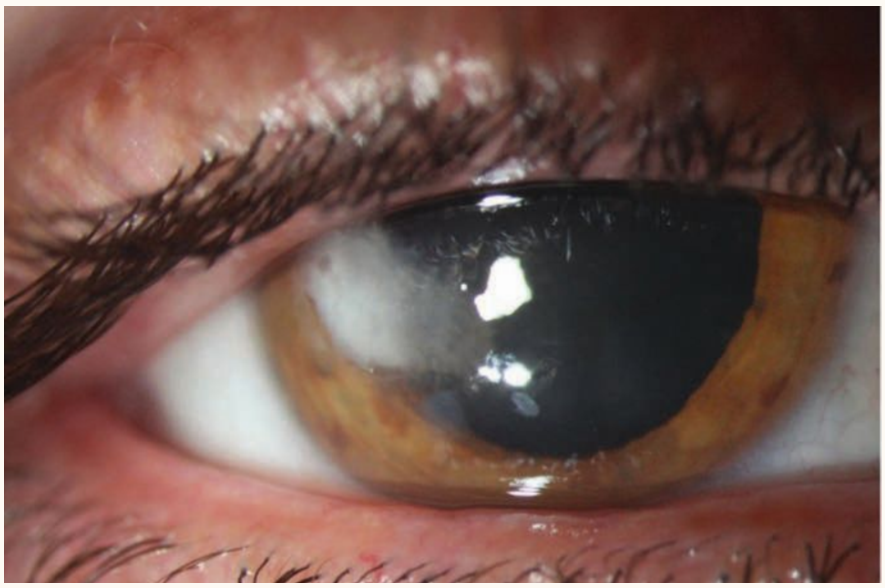

Fig. 2. This patient presented with a history of recent ocular trauma. He denied significant pain but eventually sought eye care due to blurred vision. Despite the relative "quiet" appearance of his eye, the corneal culture revealed *Fusarium* species.

Corneal epithelial defect. The cornea is the most densely innervated tissue in the body. There are an estimated 7,000 nociceptors per square millimeter, meaning the cornea is an impressive 300 to 600 times more sensitive to pain than the skin.[6] It is therefore no surprise that even a very small defect of the cornea can lead to immense discomfort.

Corneal epithelial defects can be seen with white light but are more easily visualized by sodium fluorescein under the illumination of a blue light. A careful history may also elucidate recent trauma to the ocular surface.

Intense eye pain upon awakening is commonly present in patients suffering from recurrent corneal erosion syndrome. In these cases, assessment of the cornea may reveal clinical evidence of prior trauma such as a scar, epithelial basement membrane dystrophy or a small area of nascent epithelium in erosions that have already begun to heal.

When an epithelial defect is present without pain, the physician should be suspicious of a neurotrophic component. The most common causes of a neurotrophic or desensitized cornea include herpetic keratitis, corneal nerve damage from trauma or surgery, chronic diabetes, chronic contact lens wear, chemical burns or cases such as head and neck radiation (*Figure 1*).[7] When neurotrophic keratitis is suspected, corneal sensation should be assessed prior to anesthetic drop instillation.

Dry eye and neuropathic pain. Patients with dry eye frequently experience ocular discomfort. In fact, dry eye has been reported as the number one ophthalmic cause of ocular pain that presents to an ophthalmologist.[4]

Dry eye is an over-arching term to describe a very heterogenous disease. In many settings, the clinician can visualize obvious signs of dry eye. These may include decreased tear breakup time, low tear prism height, meibomian gland atrophy, eyelid ectropion and punctate staining of the cornea and conjunctiva.

Pain, though experienced by all, is highly subjective. Pain modulation is a complex, higher-order process that is impacted by various individualized factors such as psychological stress, anticipation, past experiences and genetics, to name a few.[8,9] Furthermore, central sensitization is a maladaptive process thought to begin after an initial insult to peripheral (in our case, usually corneal) nerves. This insult can "prime" the pain pathways in the brain, decreasing the threshold needed to elicit a sensation of pain.

When a patient is experiencing significant symptoms of ocular discomfort or pain, yet there is a paucity of clinical findings to support the diagnosis of dry eye, neuropathic ocular pain (NOP) must be on our radar. Often, the discomfort is not relieved by topical anesthetics as one would anticipate with classic dry eye, making this a useful clinical probe in diagnosing this condition.[10] Allodynia, a term used to describe pain experienced from a stimulus that is not typically painful, is a common finding in patients with NOP.

NOP is challenging to treat in that it typically does not respond as well to classic dry eye therapies and can be triggered by weak stimuli such as a light breeze. There continues to be significant ongoing research investigating the pathologic underpinnings and best treatment strategies for this condition.

Infectious keratitis. The estimated incidence of corneal ulcers in the United States is between 30,000 and 75,000 annually.[11] Risks include but are not limited to contact lens wear, ocular trauma, dry eye, ocular surface disease, trichiasis and ophthalmic surgery. Clinical signs of corneal infections include an epithelial defect over an inflammatory stromal infiltrate, stromal edema, conjunctival injection and anterior chamber reaction.

Ocular pain from infectious keratitis is often coincident with photophobia, reduced visual acuity, redness and tearing. When evaluating a suspected corneal infection, the level of pain is a key component. A patient's level of pain over the course of treatment for infectious keratitis can be helpful, as reduced pain typically signifies a positive response to treatment. In addition, though not always a reliable tell, pain may also provide some clue as to the pathogen.

Classically speaking, there are some infections that tend to cause less pain than one may anticipate. As mentioned previously, herpes simplex and herpes zoster are two viruses often implicated in the pathogenesis of neurotrophic corneal disease. Interestingly, fungal keratitis has also been frequently associated with decreased pain sensation, though the reason for this is not yet fully understood. One study of patients with hyphal fungal keratitis suggests this could be the result of decreased corneal nerve density and branching (*Figure 2*).[12] Regardless of the mechanism, this reduction in pain detection can lead to a delay in seeking care and, ultimately, receiving a proper diagnosis for these indolent infections. Longer time to detection and progression of disease can lead to a worse diagnosis in these difficult-to-manage infections.

On the other side of the coin are corneal infections that create pain out of proportion to the clinical examination. The most recognizable of these infections is *Acanthamoeba* keratitis (AK). The clinician must always have this in mind when evaluating a patient with a non-specific keratitis. It should be a red flag when the patient in question has a history of contact lens wear, especially while swimming or showering in fresh water. Early clinical signs include diffuse epithelial roughness, pseudodendrites and perineural inflammation (*Figure 3*). This perineural inflammation is likely the reason for increased pain levels. Perineuritis, which presents as whitish haze along stromal corneal nerves, can be seen in up to 63% of cases at six weeks, though its existence is easily overlooked without the provider's specific intention to find it.[13]

A "classic" finding of AK is a ring infiltrate, but this is not pathognomonic for AK alone and, when present, denotes an advanced stage of disease. Due to this infection's ability to persist despite treatment, the best outcomes occur when it is diagnosed early by corneal culture or *in vivo* confocal microscopy.

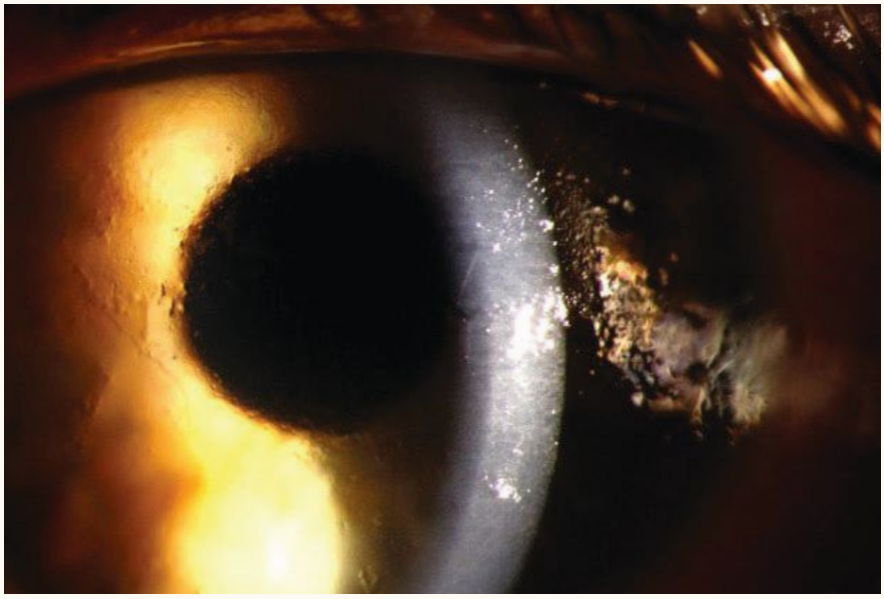

Fig. 3. This young contact lens wearer presented with significant eye pain and had diffuse corneal epithelial irregularity. There was no infiltrate or anterior chamber reaction. Due to clinical suspicion for *Acanthamoeba*, she was monitored closely. At her two-day follow-up, early perineuritis was visible and the patient was treated with antiamoebic therapy. Her symptoms resolved quickly.

Internal Causes of Ocular Pain

A variety of conditions and issues can be a source of internal ocular discomfort:

Uveitis. Photophobia is a close acquaintance of ocular pain, and one of the most common etiologies of these joint symptoms is uveitis. This condition refers to inflammation of the uveal tract and can be classified as anterior, intermediate or posterior depending on the structures involved. Pain is secondary to ciliary spasm, and therefore, cases of posterior uveitis involving primarily the retina and choroid may not be as painful as anterior uveitis. Since the iris and ciliary body are innervated by V1, the pain can "radiate," or be felt in a larger area than the eye itself. Many patients with anterior uveitis will also complain of an ipsilateral frontal headache, for example.

A thorough front-to-back ophthalmic examination is necessary to determine the level of involvement, as the treatment strategy will vary depending on the involved tissues. Even in first-time occurrences, a broad review of systems should investigate recent ophthalmic surgeries, ocular trauma, systemic autoimmune or inflammatory conditions and general systemic symptoms.

Treatment ranges from instillation of topical corticosteroid and cycloplegic agents to initiation of systemic immunosuppressive agents in chronic or non-responsive cases. Any underlying systemic disease should also be addressed.

Elevated IOP. Acutely elevated IOP, as seen in conditions such as angle closure or hyphema, can cause significant eye pain, bulbar injection, blurry vision from acute corneal edema and headache. Interestingly, there is also an association of evelated IOP with nausea and vomiting. The oculocardiac reflex, in which extraocular muscle manipulation leads to nausea and bradycardia, has been well documented in cases of strabismus surgery and trap-door orbital fractures.[14]

The understanding between acutely elevated IOP and nausea, however, is not well understood. It is thought that rapidly increasing IOP

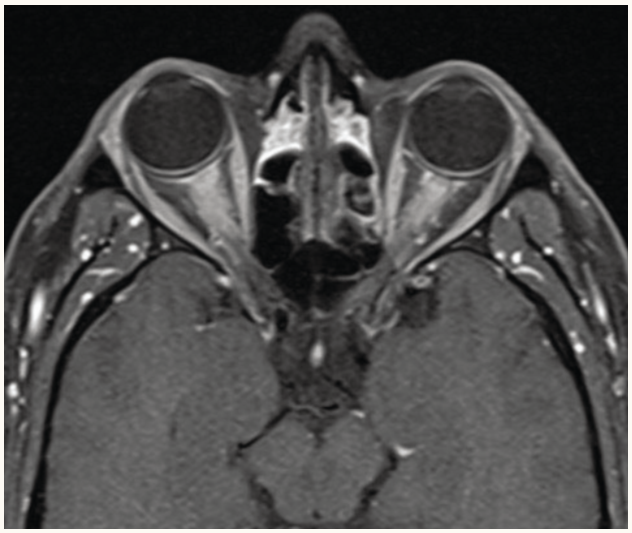

Fig. 4. This male in his mid-30s presented with severe eye pain bilaterally that was worse on eye movement. A CT scan was completed during the initial visit due to suspected myositis but was unremarkable. The patient returned to the emergency department three days later with a sudden and drastic decline in his visual acuity from 20/15 in each eye at the initial visit to no light perception in the right eye and 20/400 in the left eye. An MRI obtained that day (seen here) revealed bilateral ON. Labs eventually returned with elevated anti-MOG antibody titers.

can act as a noxious stimulus to the ophthalmic division of the trigeminal nerve, perhaps by way of swiftly altered corneoscleral stretch. Because rapid IOP changes are not associated with cardiac complications, it has been postulated that there may exist a direct "oculo abdominal reflex" arc involving the trigeminal nerve and specific vagus nerve branches to the visceral organs.[15] Our examination should ultimately focus on determining the cause of the elevated IOP and treating comorbid conditions. Ocular pain will subside significantly with normalization of the IOP in these cases.

Orbital (and Cavernous Sinus) Causes of Ocular Pain

Orbital—and cavernous sinus—pain can present as a result of a number of different issues:

Optic neuritis (ON). A hallmark of this condition is optic nerve inflammation. Though it can occur at any age and can be secondary to infectious or autoimmune conditions, the most common subset of ON is demyelinating disease that affects middle-aged females. The inflammation is most often located along the retrobulbar/intraorbital segment but may also be visible anteriorly as optic disc edema in about a third of cases. ON is associated with pain in over 90% of cases, and this typically intensifies with eye movement.[16] Though all four recti muscles attach to the annulus of Zinn near the orbital apex, the superior and medial recti muscles share close attachments with the optic nerve sheath itself. Eye movement is thought to cause tractional forces on the inflamed nerve sheath and meninges, worsening symptoms.[17]

Visual symptoms in ON tend to follow the onset of pain within a matter of days. At that point, our clinical exam will often reveal an afferent pupillary defect, mildly decreased visual acuity, a central scotoma and dyschromatopsia of the affected eye. A high index of suspicion must be maintained in early presentations, as there may be a lack of objective findings to support the diagnosis initially. It is also important to note that our differential list should include atypical forms of ON, such as those related to infections, neuromyelitis optica spectrum disorder or myelin oligodendrocyte glycoprotein (MOG) syndromes if the condition presents bilaterally or is associated with severe visual deficits (*Figure 4*).

Extraocular muscle injury. Damage to the extraocular muscles occurs mainly via blunt orbital trauma or a sharp, penetrating object. In these cases, patients usually experience diplopia and pain, especially when moving their eye. The eye itself often manifests some evidence of trauma, such as a subconjunctival hemorrhage, traumatic iritis or commotio retinae. When evaluating a patient for orbital trauma, pay close attention to motility deficits, eye misalignment, enophthalmos and maxillary hypoesthesia, as these can all be red flags for an orbital fracture.

If you suspect an orbital fracture with or without extraocular muscle entrapment, obtaining computed tomography (CT) imaging is helpful to ascertain the extent of involvement (*Figure 5*). CT imaging is quicker, less expensive and better at bone imaging when compared with magnetic resonance imaging (MRI), making it the method of choice for these cases. Pain after orbital trauma arises from muscle contusion, hematoma or entrapment. Discomfort from a hematoma will likely resolve as inflammation subsides, but patients with entrapped tissue should be referred to oculoplastics for a surgical evaluation.

Orbital inflammation. Orbital pain, though not necessarily specific to the orbit for reasons discussed above, is a salient feature of orbital inflammation, which can occur secondary to an underlying systemic pathology such as thyroid dysfunction, granulomatosis with polyangiitis, sarcoidosis, IgG-4-related disease, Sjögren's syndrome, metastatic disease or other lymphoproliferative disorders.[18] Orbital inflammation can be caused by an infection such as orbital cellulitis, which should be ruled out by a careful history and imaging of the adjacent sinuses.

When no underlying pathology can be deemed responsible, the condition is aptly termed idiopathic orbital inflammation (IOI). Under the umbrella category of IOI, inflammation most commonly involves the extraocular muscles (myositis), lacrimal gland (dacryoadenitis) or orbital fat. IOI is the second most common inflammatory orbital disorder, after thyroid eye disease.[19] Patients with

IOI are more likely to be female and in their fourth decade of life. Most commonly, when IOI affects muscles, the horizontal recti are the most commonly involved.[20] The most common symptom, therefore, is pain on eye movement, typically in the gaze directed away from the affected muscle. These cases will also likely exhibit motility restriction and conjunctival injection over the recti insertion.

When clinical suspicion supports this diagnosis, additional imaging with orbital ultrasonography, CT or MRI can be helpful, though some suggest MRI is the most sensitive diagnostic study.[21] A careful review of systems and medications should be performed, and inflammatory and autoimmune labs are often completed to rule out a systemic etiology. Depending on the severity, oral non-steroidal anti-inflammatory drugs may be a first-line therapy, but most cases will require the use of oral corticosteroids. For non-responsive cases, orbital biopsy and steroid-sparing agents are considered and often comanaged with rheumatology.

Cavernous sinus pathology. The cavernous sinuses are paired sinuses located directly posteromedial to the superior orbital fissures. The sensory ophthalmic (V1) and maxillary divisions (V2) of the trigeminal nerve pass through the cavernous sinus. Therefore, pathology in the cavernous sinus can cause pain or headache. Some examples of cases presenting with periocular pain are those secondary to carcinoma with perineural spread through the cavernous sinus, carotid-cavernous fistula, cavernous sinus aneurysms or thromboses and Tolosa-Hunt syndrome.[22-24]

Since the oculomotor, trochlear and abducens cranial nerves as well as a portion of the internal carotid artery pass through the cavernous sinus with such close proximity, it is common to see multiple cranial nerve palsies co-present when there is a mass or inflammation in the cavernous sinus. Neuroimaging is vital and should be completed on an emergent basis for an ultimate diagnosis in these cases.

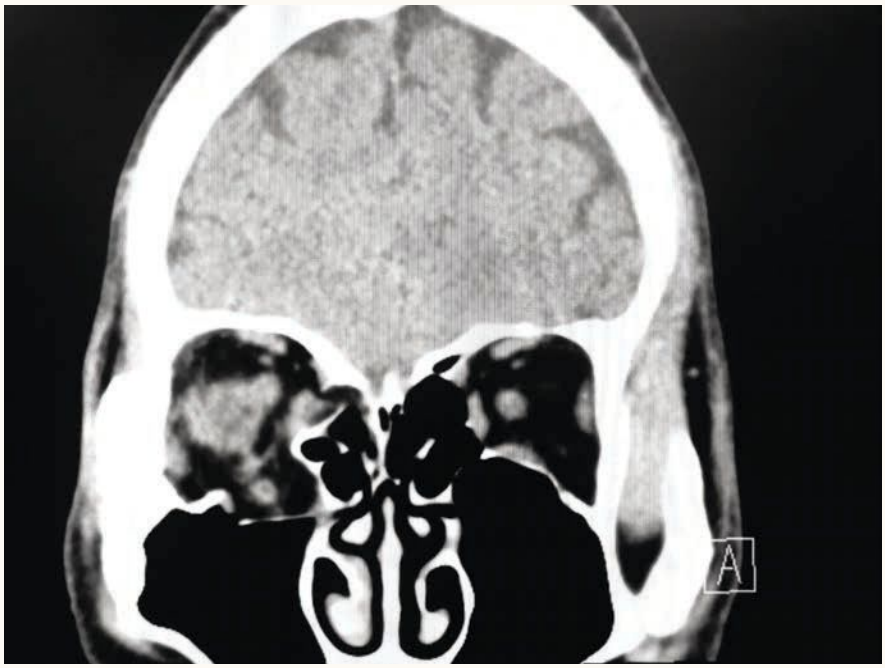

Fig. 5. This patient sustained blunt ocular trauma to the right side of his eye and developed eye pain and diplopia afterward. CT imaging revealed right medial wall and floor fractures with extraocular muscle deviation. He underwent surgical repair with a floor implant.

What We Gain from Pain

Ocular pain can be an onerous symptom, but as primary eye care providers, we are well-equipped to assess these complaints. Starting with a comprehensive history, we can focus our evaluation to determine the cause of pain for the patient in our chair. When we can help a patient in pain, there is a lot to gain for us as well as our patients. ■

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To obtain continuing education credit through the Optometric Study Center, complete the test form on the following page and return it with the $35 fee to: Jobson Healthcare Information, LLC, Attn.: CE Processing, 395 Hudson Street, 3rd Floor New York, New York 10014. To be eligible, please return the card within three years of publication. You can also access the test form and submit your answers and payment via credit card online at revieweducationgroup.com. You must achieve a score of 70 or higher to receive credit. Allow four weeks for processing. For each Optometric Study Center course you pass, you earn 2 hours of credit. Please check with your state licensing board to see if this approval counts toward your CE requirement for relicensure.

1. Nociceptor stimuli include all of the following except _____.
a. Exposure to mechanical touch.
b. Exposure to high or low temperatures.
c. Exposure to chemicals.
d. Exposure to sound.

2. The first division of the trigeminal nerve innervates _____.
a. The upper row of teeth/jaw.
b. The anterior 1/3 of the tongue.
c. Anterior cranial fossa dura.
d. The temporal scalp.

3. How many corneal nociceptors are estimated per square millimeter?
a. 50.
b. 900.
c. 7,000.
d. 15,000.

4. Herpetic keratitis, corneal surgery, ocular trauma, diabetes and head and neck radiation can all be implicated in which of the following conditions?
a. Neurotrophic keratitis.
b. Optic neuritis.
c. Uveitis.
d. Retinal detachment.

5. Based on a retrospective review, the most common ophthalmic cause of ocular pain presenting to the ophthalmologist is _____.
a. Corneal abrasion.
b. Dry eye.
c. Uveitis.
d. Post-herpetic neuralgia.

6. All of the following factors except _____ are thought to be involved in the pain-modulation process.
a. Psychological stress.
b. Genetic etiology.
c. Visual acuity.
d. Anticipation.

7. _____ is a term used to describe pain due to a stimulus that does not typically provoke pain.
a. Allodynia.
b. Hyperalgesia.
c. Hypoalgesia.
d. Dysesthesia.

8. Infectious keratitis would be more likely in a patient with a history of _____.
a. Ptosis.
b. Trichiasis.
c. Ophthalmoplegia.
d. Enophthalmos.

9. Why might hyphal fungal keratitis be less painful than one may expect?
a. Increased time to diagnosis.
b. Increased rate of coinfection with other microbes.
c. Decreased corneal nerve density and branching.
d. Decreased extent of stromal involvement.

10. Which of the following is not an early clinical sign of *Acanthamoeba* keratitis?
a. Diffuse corneal epitheliopathy.
b. Pseudodendrite lesions.
c. Perineuritis.
d. Ring infiltrate.

11. What is the name of the reflex that causes nausea and bradycardia when extraocular muscles are manipulated?
a. Oculocardiac reflex.
b. Oculogyric crisis.
c. Oculoemetic reflex.
d. Oculomotor carditis.

12. Who is amongst the most likely demographic for a demyelinating disease-related optic neuritis?
a. Male child.
b. Elderly female.
c. Middle aged female.
d. Middle aged male.

13. What percent of optic neuritis cases present with pain?
a. 100%.
b. 90%.
c. 75%.
d. 50%.

14. What is a clinical feature that could suggest MOG-related as opposed to demyelinating disease-related optic neuritis?
a. Bilateral disease.
b. Lack of pain on eye movement.
c. 20/30 visual acuity in the affected eye.
d. Negative neuroimaging results.

15. Which of the following is not a sign of an orbital fracture?
a. Extraocular muscle motility deficit.
b. Periocular ecchymosis.
c. Maxillary hypoesthesia.
d. Enophthalmos.

16. Why is a CT scan more reasonable to obtain than an MRI in cases of suspected orbital fractures?
a. It images soft tissue better.
b. It images the optic nerve better.
c. It images the globe better.
d. It images the bone better.

17. What is not a typical cause of orbital inflammation?
a. Sarcoidosis.
b. Dry eye disease.
c. IgG-4-related disease.
d. Granulomatosis with polyangiitis.

18. In cases of IOI syndrome, which statement below is accurate?
a. It never requires use of oral corticosteroids.
b. All cases require a biopsy.
c. It can masquerade as an orbital tumor.
d. All cases require long-term immunosuppressants.

19. Which of the following is true about idiopathic orbital inflammation syndrome?
a. Males are more affected than females.
b. The optic nerve is commonly compromised.
c. The incidence increases in the elderly.
d. Dacryoadenitis is a common manifestation.

20. What does not pass through the cavernous sinus?
a. CN II.
b. CN III.
c. CN IV.
d. CN VI.

Examination Answer Sheet

When Your Patient Complains of Ocular Pain

Valid for credit through September 15, 2024

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

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Answers to CE exam:

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

Post-activity evaluation questions:

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

21. Recognize the various ocular conditions that can present with pain. ① ② ③ ④ ⑤
22. Understand how to localize the source of the pain. ① ② ③ ④ ⑤
23. Ask the right questions when a patient complains of ocular pain. ① ② ③ ④ ⑤
24. Narrow the diagnosis through clinical exam findings. ① ② ③ ④ ⑤

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

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

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

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

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

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

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

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

Ⓐ Very confident Ⓑ Somewhat confident Ⓒ Unsure Ⓓ Not confident

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

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

30. Additional comments on this course: _____

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Rate the quality of the material provided:

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

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

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

Signature _____ Date _____ Lesson 121624 RO-OSC-0921

MRI Contact Lens Considerations

Material impurities may interact with magnetic fields and cause problems for practitioners and patients alike.

Q Tattoos can interfere with MRI, as studies report cases of local tissue irritation and burns. Since cosmetic and prosthetic contact lenses can share some of the same chemicals used in tattoo ink, are they safe to wear during an MRI?

A "It is safe to wear most contact lenses for an MRI, but cosmetic and prosthetic contact lenses are a potential exception; these lenses usually contain metallic oxides," says Brian Chou, OD, of ReVision Optometry in San Diego. He notes that while metallic oxides are inorganic salts without ferromagnetic activity, associated impurities such as metallic fragments can interact with MRIs.

Adverse Effects

A 2013 case report out of Japan described a 28-year-old woman wearing "circle" cosmetic contact lenses who underwent a head MRI.[1] Imaging showed a magnetic susceptibility artifact, or loss of signal or distortions. The patient did not experience discomfort, pain or heat during the imaging. The authors subsequently ran an *in vitro* MRI with "circle" contact lenses on a gelatin phantom. They confirmed the susceptibility artifact and measured a 1°C rise in temperature of the lens.

For patients with tattoos undergoing MRIs, skin burns are a rare complication.[2] In a survey of 135 patients who underwent an MRI after permanent cosmetics were applied (*e.g.*, tattooed eyeliner, eyebrows), just 1.5% reported transient problems, including slight tingling and burning.[3] In correspondence with *The New England Journal of Medicine*, researchers estimated the probability of tattoo-related MRI reactions to be between 0.17% and 0.30%.[4]

Cosmetic contact lenses contain metallic oxides that can negatively affect MRIs.

Despite the low risk of tattoo-related MRI related adverse reactions, due to the shared use of metallic oxides in both tattoos and certain contact lenses, the conservative guidance is for patients not to wear cosmetic or prosthetic contact lenses during an MRI. Susceptibility artifacts may interfere with MRI interpretation, particularly if the structures of interest are around the eye or orbit.

In contrast to tattoos, where pigments are in direct contact with tissue, the pigments in most if not all cosmetic lenses are near the front surface of a contact lens and are reasonably isolated from corneal tissue. Still, there is the remote potential for minimal thermal damage of the ocular surface. Indeed, the package insert for the Air Optix Colors contact lenses (Alcon) states that the lenses contain iron oxide, a metallic-based colorant, and cautions wearers to "remove the lenses before undergoing an MRI."[5]

There are other considerations, too. For patients with disfiguring corneal scars, cornea tattoos can improve the cosmetic appearance.[6] Corneal tattoos can also have other applications, such as blocking out glare due to a peripheral iridotomy. Inform patients with corneal tattoos about the rare adverse events associated with a head MRI.

Finally, there are several efforts underway to develop "smart" contact lenses for augmented reality and other advanced functions.[7] If these lenses have embedded ferromagnetic components, they may also require removal prior to an MRI. The same considerations would apply to future intraocular lenses with "smart" capability.

Takeaways

Due to the properties of cosmetic and prosthetic contacts, patients should remove these lenses prior to having an MRI, as imaging outcomes and patient reactions may be adversely affected. ■

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About Dr. Shovlin

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

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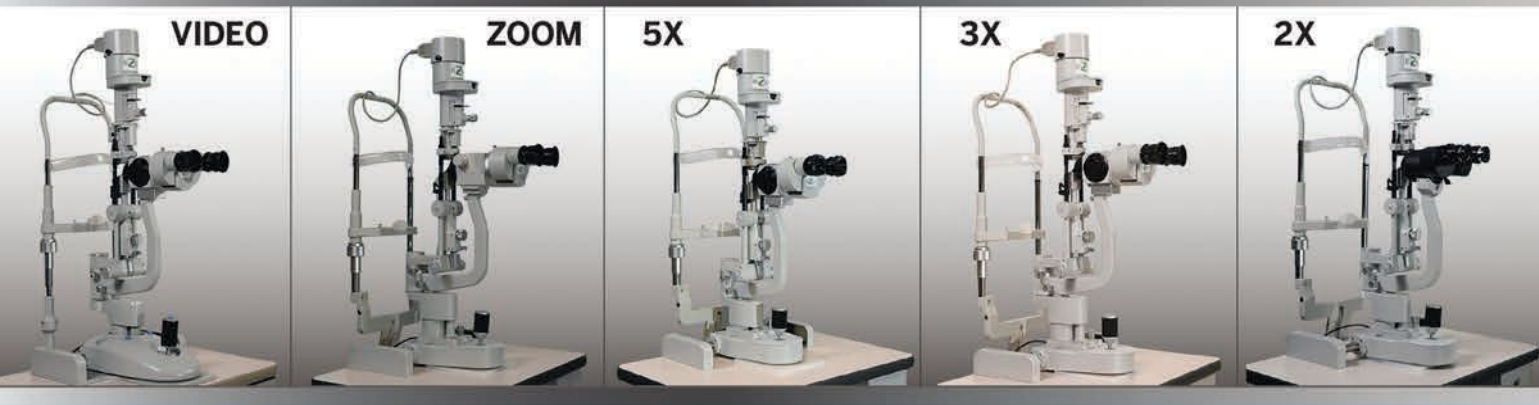

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Tread Lightly

A traumatic tire explosion in a patient's place of work was to blame for hundreds of corneal foreign bodies.

BY RACHAEL LLOYD, OD
SAINT CLOUD, MN

A 71-year-old man was referred from the local hospital after a 125-pound cement truck tire exploded while he was attempting a repair at his tire shop. The explosion catapulted the patient 10 to 15 yards in the air before he landed on cement and lost consciousness. The patient was airlifted to the local hospital due to life-threatening injuries, including a sternum fracture, left ulna fracture, traumatic brain injury and numerous foreign bodies embedded in his chest, face and eyes. The patient was hospitalized for three days, during which he complained of severe eye pain, light sensitivity and reduced vision in both eyes. He was given erythromycin ointment to be used once daily OU by the attending hospital staff. An eye examination was never completed while the patient was admitted.

Examination

One-week post-accident, the patient presented complaining of eye pain OS>OD and blurry vision. He stated that the erythromycin ointment was not helpful but that his eyes did feel better.

The patient's entering visual acuities were 20/30 OD and 20/25 OS uncorrected. His pupils were equal and round without an afferent pupillary defect. Intraocular pressures were 13mm Hg OD and 16mm Hg OS. During testing, the patient was extremely photophobic. A drop of proparacaine was instilled OU to help with pain control.

Upon anterior segment evaluation, the eyelids were noted to have mild edema of the superior lids. There were numerous embedded bulbar conjunctival foreign bodies, and the conjunctiva was diffusely injected 360°. No foreign bodies were noted in the palpebral conjunctiva. The cornea showed trace edema OD>OS, and numerous corneal foreign bodies of differing material were noted OU, OS>OD. Some foreign bodies were embedded past Bowman's membrane, and some were superficial. None of the foreign bodies were embedded past the posterior stroma/Descemet's membrane, nor were any full stromal thickness. There was no apparent iris or lens damage. The foreign bodies were only present in the exposed tissues within the palpebral aperture due to the explosive force embedding the foreign bodies before a blink could occur.

The patient was extremely symptomatic due to hundreds of open corneal epithelial wounds as a result of foreign bodies that were beginning to move anteriorly. Some had already fallen out on their own, which was apparent due to positive sodium fluorescein staining over abrasions without any foreign bodies present.

The foreign bodies were of differing materials, including metallic, organic, glass and others of unknown origin, due to the various substances that originated from the tire tread. This encompassed everything that the tire had driven over and trapped in its tread.

All corneal defects were Seidel-negative OU and non-penetrating. The anterior chamber was deep and quiet OU. The iris was flat, even and intact OU. Upon dilation, no foreign bodies were noted in the posterior chamber.

An initial lavage with saline eye wash was done to remove any superficial foreign bodies OU. Once complete, a foreign body spud was used to remove all surface-level foreign bodies and an Alger brush with 5mm burr was used to remove rust rings where necessary OU.

Discussion

Corneal foreign bodies are the second most common form of ocular trauma. Traditionally, they are superficial and can be very uncomfortable for the patient. Rarely, they may embed in and even penetrate the cornea.

These injuries commonly happen in the workplace and occur most frequently in occupations that involve metalwork. The best protection against corneal foreign bodies is protective eyewear, such as safety shields and goggles. Most workers do not wear eye protection consistently and, because of this, are at a much higher risk of serious ocular harm.[1]

In the unfortunate event that a foreign body makes its way into the cornea, the origin of the foreign body must first be determined. Removal should then happen as quickly as possible.

Removal can occur using many different techniques. Irrigation can remove superficial foreign material quite well and is less invasive than other methods. However, if the foreign body is deeply embedded, a 25- to 30-gauge beveled needle or a stainless steel spud can be used under slit lamp to assist in removal. While an Alger brush with burr can also be used

About Dr. Mangan

Dr. Mangan is a board-certified consultative optometrist from Boulder, CO, and a fellow of the American Academy of Optometry. He is an assistant professor in the department of ophthalmology at the University of Colorado School of Medicine. His focus is on ocular disease and surgical comanagement. He has no financial interests to disclose.

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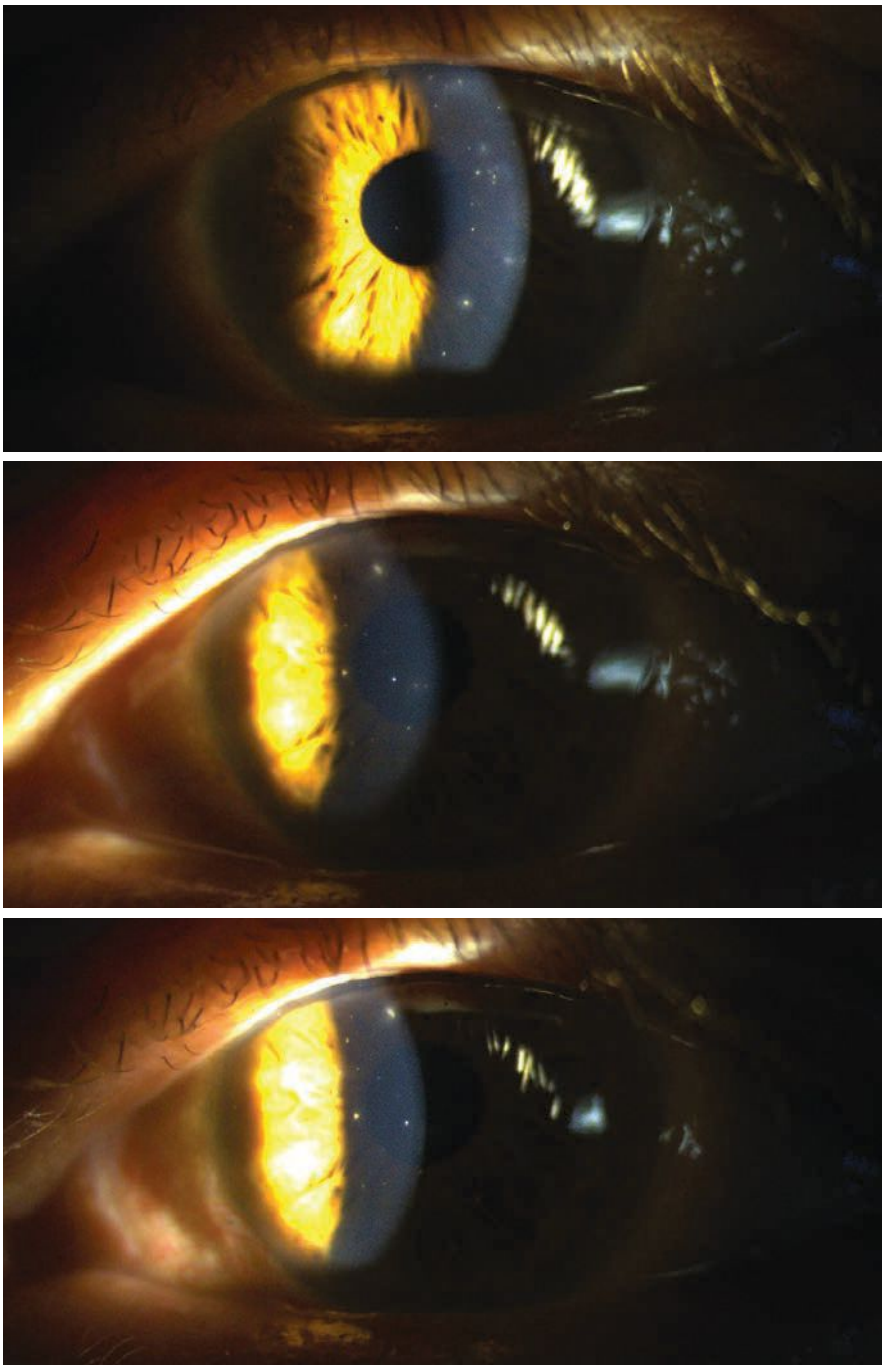
REVIEW of OPTOMETRY



Embedded corneal foreign bodies of differing material scattered throughout the left cornea.

to eradicate a foreign body, it is most frequently used to remove rust rings that are commonly the result of metallic foreign bodies.

Metallic foreign bodies can be removed with a low risk of infection; however, they have a higher risk of scarring after removal. If the abrasion post-foreign body removal is large or central in nature, it is common to issue topical prophylactic antibiotic coverage.

If a foreign body is organic in nature, risk of infection is higher, and the patient should be monitored closely for development of bacterial or fungal keratitis post-foreign body removal. It would also be appropriate to issue a topical broad-spectrum prophylactic antibiotic in this case.

Patching has been shown to provide no improvement in healing time or pain control in those who have undergone foreign body removal and is therefore not a commonly used method.[2]

If a foreign body is posterior to the posterior stroma or Descemet's membrane, or full stromal thickness, emergent surgical removal by an ophthalmologist is recommended.

Wrap-up

Once the foreign bodies that were able to be removed were taken care of, the patient was given one more saline lavage OU. Ofloxacin drops were issued for use QID OU for seven days. The patient was not patched, and, due to the small size of the patient's corneal epithelial defects and residual embedded foreign bodies, a bandage contact lens was not placed.

Upon follow-up two days later, the patient's symptoms were greatly reduced and his vision had begun improving. A few superficial foreign bodies were starting to emerge, so removal was assisted with a corneal foreign body spud.

At the final follow-up two days later, all remaining foreign bodies were deep in the corneal epithelium, and the cornea was smooth without sodium fluorescein staining. No additional abrasions were present. The patient was asymptomatic.

Since the final follow-up, the patient was seen two additional times. He remained without symptoms, and no further foreign body removal was necessary. ■

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Persistent Problems

Poor ocular history contributed to this recent diagnosis.

BY TANIA PATEL, OD
LEXINGTON, KY

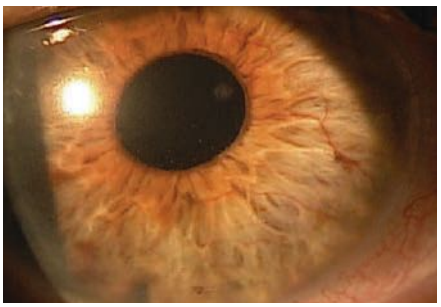
An 81-year-old Caucasian male was referred for a YAG capsulotomy evaluation. He complained of constant blurry vision, which started two years ago and was more prevalent in the right eye. Because of this, he had difficulty distinguishing road signs, seeing his cell phone and watching TV. He reported that his vision was "like looking through wax paper" and it had been very difficult to function because he is hard of hearing and depends heavily on his vision.

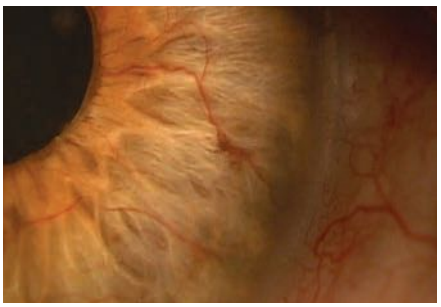
His medical history consisted of thyroid disease, heart murmurs, hypertension and COPD. His ocular history included cataract surgery OU and a horseshoe tear (HST) with vitreous hemorrhage OD that was treated with argon laser approximately two years ago. He developed an inferior retinal detachment (RD) three months after the HST repair and was treated with pars plan vitrectomy and silicone oil. Postoperative instructions included maintaining face-down positioning; however, the patient was unable to position his head correctly. He subsequently developed proliferative vitreoretinopathy (PVR) with inferior fluid and required a retinectomy.

Upon exam, his best-corrected vision was 20/200 OD and 20/80 OS. The motility was full in both eyes, and confrontation visual fields were restricted 360 OD and full to careful finger counting OS. Pupil was round but nonreactive to light in OD; the one OS was reactive to light. There was an afferent pupillary defect in the OD by reverse testing.

Slit lamp exam of the anterior segment examination showed a central corneal scar OD and mild punctate epithelial erosion. Significant neovascularization of iris (NVI) OD was seen (*Figures 1 and 2*). Also noted was PCIOL, s/p YAG capsulotomy in OD. A PCIOL was present in OS with moderate posterior capsular opacification OS. IOPs was 28mm Hg OD and 10mm Hg OS. No apparent neovascularization of the angle (NVA) or peripheral anterior synechia was visible during gonioscopy.

Evaluation of the posterior segment showed CD ratio larger in OD compared to OS. Right eye nerve appeared shallow, and macula showed subretinal fluid (SRF). The area of retinectomy was visible. It also showed chronic anterior inferior retinal detachment (*Figure 3*). OCT of the right eye confirmed SRF (*Figure 4*). Left nerve had cup to disc ratio of 0.40, and the rest of the posterior segment was unremarkable.





Figs. 1 and 2. Neovascularization of iris (NVI) was found during the slit lamp exam.

Take the Retina Quiz

1. Which is not a cause of NVI?
a. Central retinal vein occlusion (CRVO).
b. Chronic RD.
c. Proliferative diabetic retinopathy (DR).
d. Exudative macular degeneration.

2. What duration of time defines an RD as "chronic?"
a. Greater than one week.
b. Greater than two weeks.
c. Greater than one month.
d. Three months.

3. Which is not considered a risk factor for development of PVR in a RD?
a. Vitreous hemorrhage.
b. Chronic RD.
c. Smoking history.
d. DR.

4. What is the surgical failure rate in RD repair due to PVR?
a. 1% to 5%.
b. 5 to 10%.
c. 10% to 20%.
d. Over 25%.

5. Which statement is false?
a. NVI can occur before NVA.
b. NVA can occur before NVI.
c. Prostaglandin analogs should be used to manage neovascular glaucoma (NVG).
d. Prostaglandin analogs should not be used to manage NVG.

For answers, see page 106.

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

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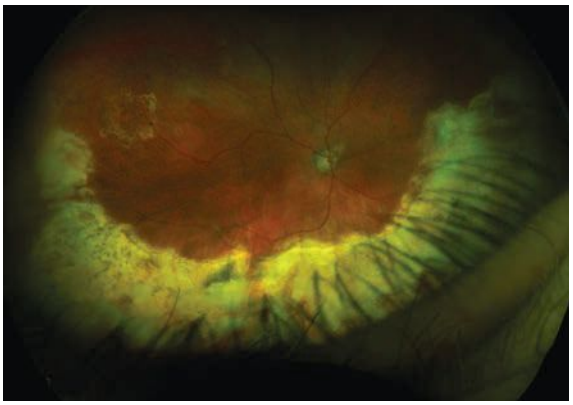

Fig. 3. Inferior retinal detachment was found in the right eye.

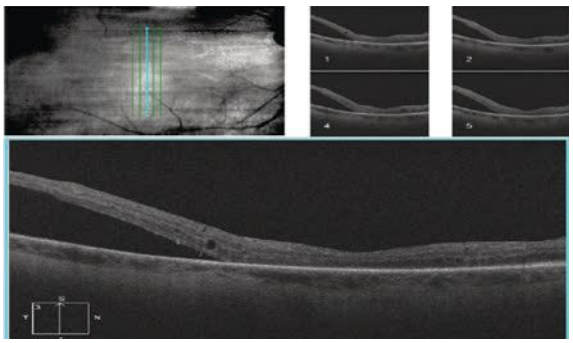

Fig. 4. OCT of the right eye confirmed SRF.

Diagnosis

This patient was diagnosed with an old rhegmatogenous retinal detachment (RRD), macular atrophy from presumed multiple macula-off RDs, active NVI and ischemic status of the retina (suspected ocular ischemic syndrome) OD, and ocular hypertension from impending NVG. He also had some detached retina inferiorly, which was believed to represent the anterior portion of the retinectomy.

The previous carotid Doppler—done 10 years prior—was found to have 80% occlusion, which prompted a new Doppler to be ordered. After reviewing the results, the retina specialist did not think repair of the patient's chronic RD would abate the anterior segment neovascularization. He was started on Combigan (brimonidine/timolol, Allergan) BID OD to help lower the intraocular pressure (IOP).

Discussion

Historically, the most common causes of NVI include DR and CRVO. His retina did not show any signs of these conditions and he denied having diabetes or any history of venous occlusion. Other differential diagnoses included retinal ischemia, tumors, ocular inflammation and RD.

This patient had a history of RRD, PVR and retinectomy. PVR is thought to be an abnormal wound healing process where proliferative, contractile cellular membranes form in the vitreous and the retina, causing tractional RD. Membranes are formed due to cytokines and inflammatory mediators from tissue damage or anatomic disruption caused by the RRD.[1]

The surgical failure rate of RRD repair due to PVR in RRD is believed to be between 5% and 10%. Risk factors for PVR development include retinal tear size, presence of vitreous hemorrhage, chronic intraocular inflammation, longstanding untreated RRD, unsuccessful RRD repair, history of smoking and penetrating ocular trauma.[2]

Retinectomy is the treatment of choice to relax the tension on the retina by peeling the tractional membrane and removing the tissue—the anterior flap of a retinal tear or remove the fibrotic, contracted retina from PVR.[2,3] It is performed in up to 64% of the PVR cases. Studies have shown that retinectomy with use of silicone in eyes with recurrent RD or PVR shows good prognosis and low risk of recurrence. Three types of retinectomy are performed: inferior 180° (as seen in this patient), 360° and focal posterior—of which the first is most common.[3]

The severe neovascularization of the patient's anterior segment was thought to be secondary to carotid occlusive disease or from hypoxia due to chronically detached retina. Carotid artery occlusion with elevated IOP in the ipsilateral eye is suspicious for ocular ischemic syndrome.[4] However, his current carotid Doppler was reported to be normal.

Retinal ischemia and subsequent hypoxia can occur due to chronic separation of the sensory retina from the choroidal blood flow.[1] This can trigger neovascularization. In this patient, chronic RD caused retinal ischemia and hypoxia, which stimulated the production of VEGF and triggered the NVI.

Preserving the remaining vision, preventing NVG and secondary angle closure glaucoma OD and improving his vision OS by performing YAG capsulotomy are the primary goals for this patient.

NVG can be managed with topical alpha-2 agonists, beta blockers and topical or oral carbonic anhydrase inhibitors. Prostaglandin analogs should be avoided to prevent further breakdown of the blood-aqueous barrier.[4] The patient's IOP remained high on Combigan BID, so he was referred to a glaucoma surgeon for possible surgical intervention. ■

Special thanks to Taylor Brooke Runyon and Hannah Collier, both fourth-year students at University of Pikeville, Kentucky College of Optometry.

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Dr. Patel received her doctor of optometry degree at Nova Southeastern University College of Optometry and practices at Commonwealth Eye Surgery in Lexington, KY. She is a member of the Kentucky Optomeric Association and the American Optometric Association. She has no financial interests to disclose.

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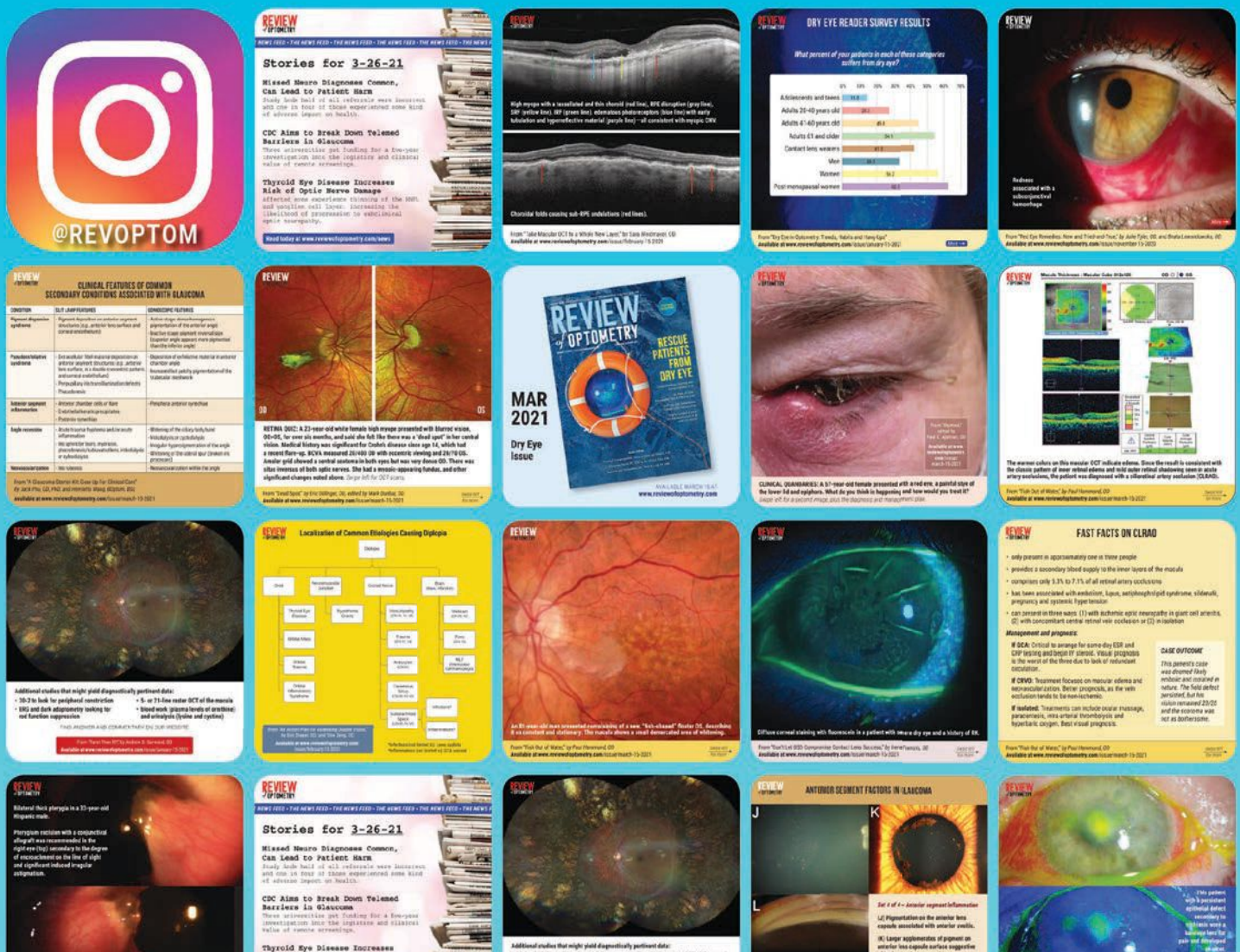

BY BISANT A. LABIB, OD

Tear Film Fighters

While often underappreciated, these important components serve to protect our eyes.

An intact and stable tear film is necessary for many vital functions. Among other things, it provides comfort and clarity that is often reduced in dry eye disease. While the relationship between tear composition and dryness is well-established, we often neglect the other microscopic yet essential components of the tear film that serve to protect our eyes from infection and damage. Besides comfort and a smooth refractive surface, the tear film is also our eye's first line of defense, protecting us from pathogenic invasion.[1]

Schirmer's test can help detect reduced aqueous production, indicating the need for clinical management.

Parts of the Whole

The tear film is a heterogenous, clear covering over the anterior surface of the eye. It serves as a barrier between the cornea and conjunctiva and the external environment.[1] The tear film is composed of three distinct layers, which all contribute to its various functions.

The first, most outermost layer is the lipid layer, which is secreted by meibomian glands. This portion of the tear film provides surface tension necessary to prevent tear evaporation and keep the ocular surface smooth and well lubricated, while also preventing spillover onto the lid margin.[2,3]

The middle portion is the aqueous layer, which is produced by the lacrimal glands. This is a critical layer in that it is not only responsible for lubrication, hydration and nourishment of the underlying avascular cornea, but it also contains several molecules that serve as antimicrobials, protecting the eye from various external pathogens.

Finally, the innermost layer is the mucin layer of goblet cells, which assists in wettability and spreads tears with each blink.[3]

Since the eye is readily exposed to the external environment on a daily basis, it is subject to the invasion of many pathogens, including bacteria and fungi. Some of these common microorganisms of the ocular surface include the bacteria *Staphylococcus aureus* and *epidermis*, *Streptococcus pneumonia* and *Pseudomonas aeruginosa*. Fungal infections are usually caused by *Aspergillus*, *Candida* and *Fusarium* organisms. These pathogens are often implicated in bacterial or fungal keratitis associated with trauma or poor contact lens hygiene. In defense of these potential infections, blinking and reflex tearing flush out the tear film, while the tear film itself contains various antimicrobial defense proteins.[2]

One of the most common antimicrobial substances in the tear film is lysozyme.[2,4,5] This is secreted by the lacrimal glands and found in the aqueous layer, functioning to destroy bacterial cells through the breakdown of peptidoglycan within the cell wall.[5] It is mainly effective against gram-positive bacteria and constitutes approximately 20% to 30% of the total proteins found in tears.[2] Lysozyme exhibits antifungal properties as well, as it is able to break down chitin, a component in fungal cell walls.[2,5] One study also described its anti-HIV activity.[2] In addition, decreased levels of lysozyme in the tear film have been reported in dry eye disease, herpetic disease and beta-blocker usage.[4]

Another abundant and important protein in the tear film is lactoferrin.[2,4,5] This substance is also found in the aqueous layer and secreted by the lacrimal glands. Lactoferrin indirectly works as an antimicrobial by binding iron and undermining bacterial metabolism and growth.[2,5]

Similarly, lipocalin also binds molecules that transport iron and are produced by many types of bacteria and fungi, interfering with these harmful organisms' iron uptake abilities. It is abundant in reflex tears and produced by the lacrimal glands.[2]

While the origin and exact mechanism is unknown, alpha-lysin is found in tears in higher quantities

About Dr. Labib

Dr. Labib graduated from Pennsylvania College of Optometry, where she now works as an associate professor. She completed her residency in primary care/ocular disease and is a fellow of the American Academy of Optometry and a diplomate in the Comprehensive Eye Care section. She has no financial interests to disclose.

Tear Film Properties

Layer	Production	Function
Lipid	Meibomian glands	Facilitate surface tension to prevent evaporation and spillover
Aqueous	Lacrimal glands	Facilitate hydration to nourish the underlying cornea and produce antimicrobials to protect the eye
Mucin	Goblet cells	Facilitate tear spreading

than those that are present in the aqueous and plasma, functioning to damage bacterial cell membranes.[5]

In addition to these proteins, there are immunoglobulins and antioxidants present in the tear film, both of which contribute to the antimicrobial activity of the ocular surface. Namely, secretory immunoglobulin A (sIgA) is the most copious and multifactorial in purpose. Its presumed mechanism of action is to coat microbes, preventing adherence to host cells and further neutralizing and lysing bacteria, viruses and parasites.[2,5]

As the eye is also subject to oxidative stress and damage due to constant environmental exposure, antioxidants such as ascorbic acid, uric acid, L-cysteine and L-tyrosine also protect the ocular surface by acting as free radical scavengers.[1]

Takeaways

The functions of the tear film are many, and this covering's antimicrobial properties are often underappreciated. All of these microscopic components serve to combat harmful pathogens every day. A disruption in this composition is seen in many ocular surface disease processes, which should be considered when assessing the risk of potential ocular infection. ■

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▶ DIAGNOSTIC EQUIPMENT

Eye Tracking Device Detects Oculomotor Dysfunction

If you want to improve the accuracy of your EOM exams, reduce the time it takes to perform them and create better documentation of results, a new product from RightEye aims to help, the company says. Called RightEye Sensorimotor, the device is a tablet with an eye tracker and custom software that administers oculomotor tests and then produces documentation of the results.

Because the test can be run with minimal input from an operator, doctors can remove this element from their exams and delegate it to a tech in the pretest area, the company suggests. The tech can conduct a screening in less than 30 seconds; then, if deemed medically necessary by the doctor, a more comprehensive sensorimotor exam can be completed in under five minutes, RightEye explains. Available tests include nine-point motor function, near point of convergence, cardinal gaze assessment, Worth 4-dot, fixation stability, randot testing, saccades/pursuits and other measures of binocular function. The company says the device meets CPT requirements as a billable test of sensorimotor function.

Virtual Reality Headset For Visual Field Testing

A new virtual reality (VR) headset can complete visual field testing in under three minutes per eye and in multiple settings around the practice, according to manufacturer M&S Technologies. The Smart System VR Headset can perform 10-2, 24-2 and 30-2 visual field and contour stereo testing, and features an eye tracking function for fixation monitoring, the company says.

The programmable headset is lightweight, portable and does not require an internet connection to operate, says M&S. It features an adjustable head strap and is cushioned for a comfortable fit, the company adds. The device comes with a handheld controller for the patient and a tablet PC for the clinician that monitors and records test progression. In order to ensure accurate vision testing, the headset must be used in a fully illuminated room, and it includes an option to pause or restart the test as needed, says M&S. Results can be printed or exported immediately once the test concludes.

Three-in-One Device for Myopia Assessment

A new device for myopia management may reduce chair time and save space in your practice by performing three ocular measurements at once, manufacturer Oculus says. The Myopia Master can measure axial length, refraction and central corneal radii, eliminating the need to have a separate device for axial length measurement as is typical now.

The device uses interferometry to perform the various measurements. While traditional refraction tests are prone to fluctuation from day-to-day changes in acuity and measurement variability, the Myopia Master is not influenced by eye accommodation status, the company says. It also records lifestyle and other patient-specific risk factors (*e.g.*, number of myopic parents, time spent outdoors, near-work activity) and combines this information with the measurement data to produce a report of the individual's myopia profile, company literature explains. The Brien Holden Vision Institute collaborated with Oculus to develop the patient questionnaire and the risk assessment algorithm.

▶ PRESCRIPTION DRUG RESOURCE

Online Drug Database Helps Inform Prescribers

To help guide prescribing decisions, a company called EyeMedsNow has created a large, searchable database of information on over 230 ophthalmic medications that can be accessed at no cost by anyone who registers.

A search tool lets you filter results based on over 100 ocular conditions, dozens of drug classifications, patient age, route of administration, custom keywords, OTC/Rx designation and whether payment assistance is available. Results appear in the form of "drug cards" that summarize key facts about the product, including typical usage and dosing, container sizes, preservatives, proper storage, and important warnings and precautions. Each drug card also includes links for any payment assistance services, as well as for GoodRx, to give you insight on local pharmacy pricing, the company says.

The site also features a dosing calculator and additional databases for OTC tears and eyelid hygiene products. ■

That's Swell of You

A complex medical history complicates assessment of a recent-onset red eye.

BY SAURIN PATEL, OD
PHILADELPHIA

A 65-year-old Black male presented with acute redness, tearing, soreness and pain OS of five days' duration. He did not complain of reduced vision. He said the pain was 5 out of 10 and that he had similar symptoms in right eye, which spontaneously resolved one week prior. His past ocular history included blunt trauma OS with epiretinal membrane formation and an episode of non-granulomatous uveitis of unknown etiology some 10 years ago.

Medical history included discoid lupus, antiphospholipid antibody syndrome, Raynaud's, osteomyelitis of the extremities and left hip, anemia, peripheral neuropathy, renal failure, cerebral vascular accident and rehabilitated substance dependence. His medications included Plaquenil 200mg BID since 2006, metoprolol, nifedipine, warfarin and acetaminophen.

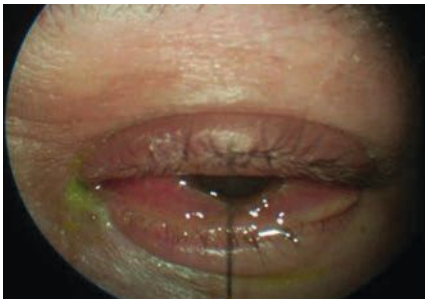
Diagnostic Data

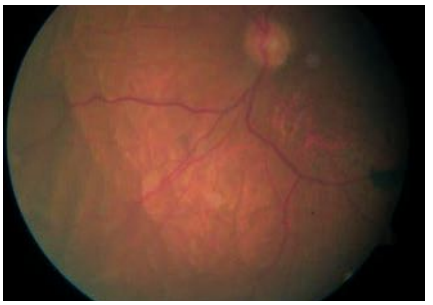
His best-corrected visual acuities were 20/25-2 OD and 20/40 OS at distance and near. His pertinent external findings by gross observation are demonstrated in *Figure 1*. Pupils and ocular motilities were normal. Confrontation visual fields were constricted. Refraction was not completed due to the emergent nature of the presentation.

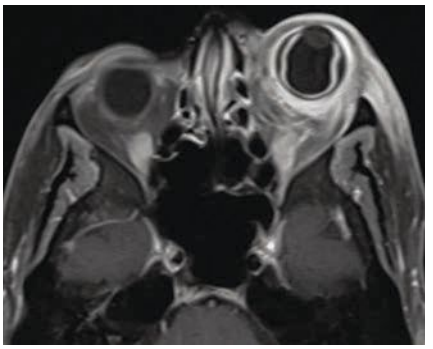
Biomicroscopy OD revealed normal and healthy anterior segment structures. Examination OS uncovered diffuse injection with tenderness of the external lids, palpebral and bulbar conjunctiva. There were no cells in the anterior chambers of either eye, the lenses were well centered and there was no evidence of vitritis, OU. Goldmann applanation tonometry measured 10mm Hg OU. The dilated fundus exam was normal OD. The left eye (*Figure 2*) demonstrated an epiretinal membrane and pigment disruption of the macula secondary to previous trauma. There was no evidence or neurosensory retinal detachment or frank choroidal folds in either eye. Orbital MRI is also available (*Figure 3*).

Your Diagnosis

What would be your diagnosis in this case? What is the patient's likely prognosis? To find out, please read the online version of this article at www.reviewofoptometry.com. ■







Figs. 1-3. What do these images suggest about the nature of this patient's condition?

ABOUT THE AUTHOR

Dr. Patel is an attending optometrist at the Philadelphia Veterans Affairs Medical Center, a clinical educator at Salus University and a private practice owner. He is also a Lt. Col. of the U.S. Air Force. He has no financial interests to disclose.

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

Retina Quiz Answers (from page 98)—Q1: d, Q2: b, Q3: d, Q4: b, Q5: c

NEXT MONTH IN THE MAG

In October, we present a series on the theme of strengthening your practice. Articles will include:

- Protect Yourself Against a Malpractice Claim
- Skill-building for the Busy Clinician: How to Add New Services
- Take Advantage of the Changing Scope of Practice Landscape
- Empower Your Staff—and Free Yourself Up

Also included in October:

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Micaela Crowley, OD
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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, Nelson JL, Pruitt JD. Loading and release of a phospholipid from contact lenses. Optom Vis Sci. 2011;88(4)502-506.

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

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