Clear Up Cataract Concerns, *P. 40* • A Results-Oriented Neuro Work-up, *P. 46*

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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.

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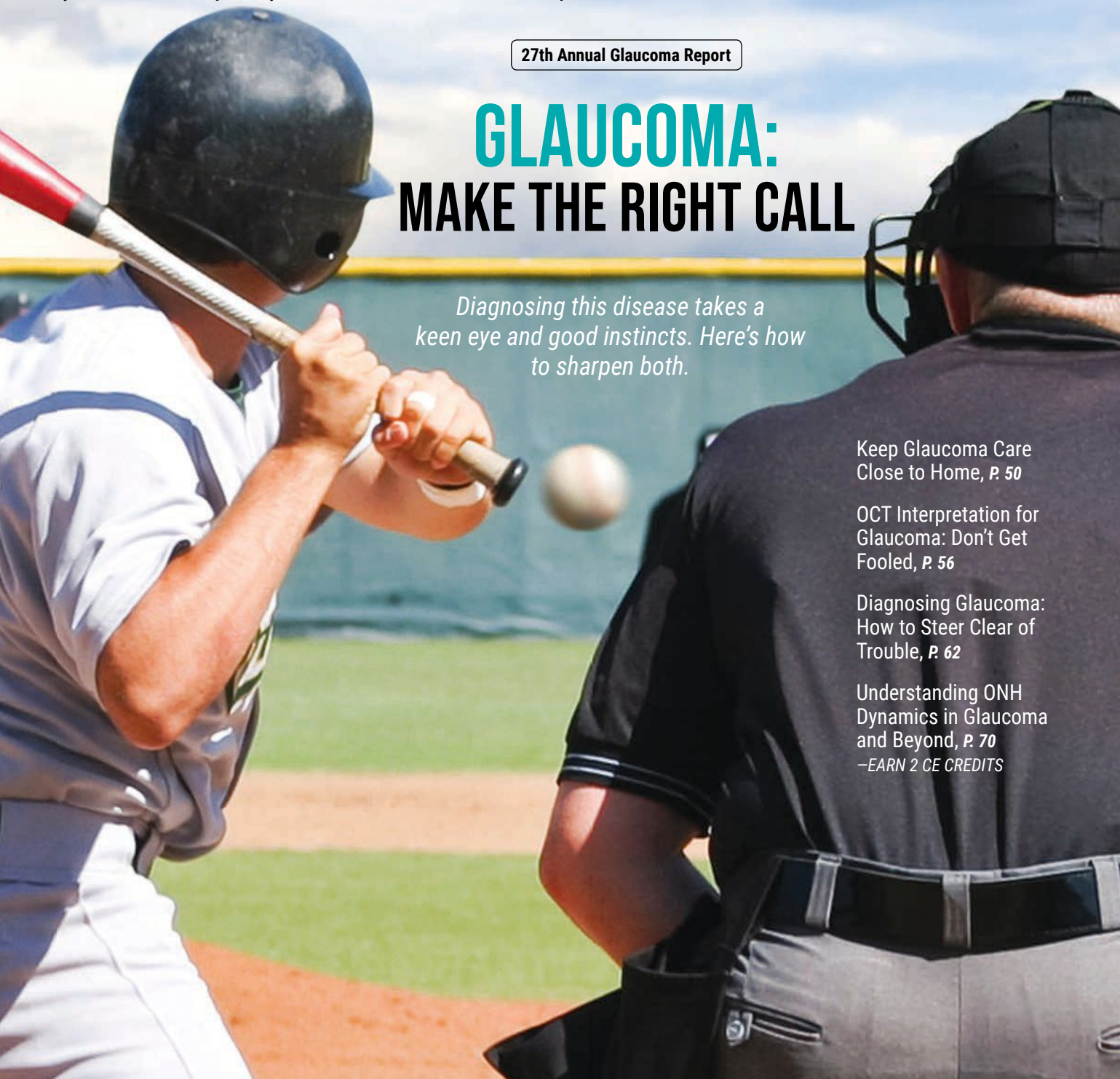
27th Annual Glaucoma Report

GLAUCOMA: MAKE THE RIGHT CALL

Diagnosing this disease takes a keen eye and good instincts. Here's how to sharpen both.



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Alcohol Exacerbates Dry Eye in Women

Sex-specific mechanisms highlight hormonal influences on the condition.

Researchers investigated the association between dry eye and alcohol consumption and found that alcohol's effect on dry eye disease (DED) is very sex-specific.

The population-based study included 77,145 participants between the ages of 19 and 94 (59% female) from the Dutch Lifelines cohort. All participants were cross-sectionally assessed for DED with the Women's Health Study dry eye questionnaire. The researchers assessed alcohol consumption with a self-reporting food frequency questionnaire. Analyses were adjusted for age, sex, BMI, smoking status, education, income and 55 potentially confounding comorbidities.

They found that 30% of participants had symptomatic dry eye, with alcohol significantly increasing the risk in females but not in males. Curiously, the researchers found that increasing alcohol intake had a protective effect on symptomatic dry eye among males. Alcohol had sex-specific effects on all DED outcomes assessed, including symptomatic dry eye, highly symptomatic dry eye, clinical diagnosis and the Women's Health Study's definition of dry eye.

> **"Differences in sex hormones may play a role, as androgens—more prevalent in males—are thought to help maintain ocular surface and adnexal health.**

"The influence of sex on the association between alcohol use and all outcome measures of dry eye found in this study is particularly intriguing," the researchers wrote in their paper. "Past studies have not investigated this relationship. Stratification by sex is crucial as alcohol has been found to have different physiological and pathological effects on males and females. In addition, the prevalence, clinical characteristics, pathophysiology and treatment response of dry eye are all well-known to be substantially different in females and males."

The researchers proposed several mechanisms to explain female response. One theory suggests that differences in sex hormones may play a role. Androgens—more prevalent in males than females—are thought to help maintain ocular surface and adnexal health. Additionally, androgen deficiency has been linked via an autoimmune process to tear deficiency, corneal and conjunctival damage, lacrimal gland inflammation and meibomian gland dysfunction. On the other hand, estrogen stimulates immune responses; its effects on the human ocular surface remain inconclusive.

The team concluded that in this large, population-based study, alcohol was a risk factor for symptomatic DED only in females. "This adds to the evidence of sex-specific pathophysiological mechanisms of dry eye and illustrates the importance of sex stratification in studies investigating DED," the researchers wrote. "The mild protective effect of increased alcohol intake in male drinkers is advised to be interpreted with caution, as alcohol's other health effects might be of greater clinical significance."

Magno MS, Daniel T, Morthen MK, et al. The relationship between alcohol consumption and dry eye. Ocul Surf. May 21, 2021. [Epub ahead of print].

CORRECTION

The June Coding Connection column "Bring The Retina Into View" stated that CPT code 92284 is a "true bilateral test requiring that it be performed bilaterally." That is incorrect. Here, Dr. Rumpakis provides additional information on the code and clarifies its use:

In 2012, a *CPT Assistant* article addressed the use of code 92284 in unilateral or bilateral ophthalmological procedures and the need for modifiers to distinguish between procedures performed on one eye or both eyes.

CPT 92284 is considered, per AMA/CPT and CMS guidelines, an inherently unilateral/bilateral code that can be reported once per session when performed in one eye or both eyes, without the use of code modifiers for laterality.

The *CPT Assistant* article clarified that CPT 92284 did not require the use of modifiers to differentiate between unilateral and bilateral procedures. Specifically, the article stated "Some ophthalmological procedures (CPT 92284 included) may not necessitate the use of modifier 50 or 52. For example, the procedure may routinely require the performance of multiple elements that may be repeated, as indicated in the code descriptor. However, the CPT code is reported only once for the session, regardless if the procedure is performed unilaterally or bilaterally.""

Modifiers 50 and 52: special ophthalmological services. CPT Assistant, Oct. 2012. American Medical Association. All rights reserved.

UV Index Misses Key Ocular Aspects

Protecting your eyes from the sun can go a long way toward preventing acute ocular diseases such as photokeratitis and snow blindness, and chronic ones like cataracts and pterygia. But, while people often take precautions to protect their skin, they rarely protect their eyes with the same fastidiousness. A new study noted that the UV index isn't a great measure for the amount of UV damage to the eyes and proposed a new instrument to help the public better pinpoint ocular UV exposure.

The instrument includes a rotating model head with UVB sensors that record intensity at the crown and eye area. The sensors span eight azimuths from sunrise to sunset during multiple climatic conditions throughout the year. The researchers obtained UV dose intensities from their instrument to create an ocular UV index and compared it with the UV index.

They found that UV exposure to the crown of the head increases with the sun's altitude, whereas UV exposure to the eyes is greater at lower solar altitudes. "The ocular UV index levels were higher than recorded UV index levels in the summer under low solar altitude in the early morning and mid-to-late afternoon and were markedly higher all day in winter when solar altitude remains low," the researchers wrote.

"This may be because the UV sensor on the crown mainly measured overhead solar UV and those on the eyes were influenced by the horizontal effects of the scattering component of UVB in winter when the sun altitude was lower," the researchers explained. "When the mannequin [modeled after a Japanese female] faced the sun (summer, spring and autumn), ocular exposure slightly decreased around the time of maximum solar culmination but then increased again, so there were two peak times of high irradiance." They noted that previous studies have found steep bimodal peaks of UVA (315nm to 400nm). The present study measured only 280nm to 310nm of UVB.

They concluded that the UV index doesn't provide a good warning system when it comes to ocular solar exposure. They say their proposed ocular UV index will be useful for warning the public about ocular UV exposure in addition to providing another means of researching UV-induced ocular diseases.

Hatsusaka N, Seki Y, Mita N, et al. UV index does not predict ocular ultraviolet exposure. Trans Vis Sci Tech. 2021;10:1.

Glaucomatous Disc Hemorrhage Affects Central VF

Optic disc hemorrhages strongly correlate to the development and progression of glaucoma, yet the pathophysiology of this associated finding remains unknown. Looking to bridge this gap, a study recently reported that central visual field (VF) loss is accelerated in glaucomatous eyes with disc hemorrhage and corresponds topographically to the location of the hemorrhage. The researchers recommend supplementing 10-2 visual fields with 24-2 fields in these patients.

The prospective study evaluated 343 eyes of 220 subjects who had at least three years of follow-up with a minimum of five visits that included 10-2 and 24-2 VF testing. Of the total eyes, only 39 had experienced a disc hemorrhage.

The team found that eyes with disc hemorrhage had rates of 10-2 mean deviation (MD) loss that were three times faster than controls (-0.36 dB/year) and were 3.7 times more likely to progress. In early glaucoma, the rate of 10-2 MD loss increased to 5.5 times faster. Disc hemorrhage eyes also had a faster rate of 24-2 MD deterioration, but it did not reach statistical significance.

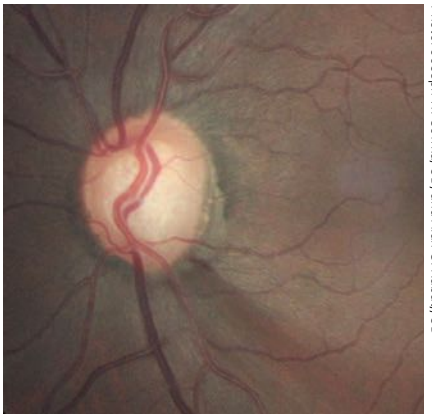

Photo: Joseph W. Sowka, OD, and Alan G. Kabat, OD

Disc hemorrhages predict accelerated loss, especially in early stages of glaucoma.

The researchers found that a larger proportion of glaucomatous eyes showed central VF progression rather than peripheral VF progression in the disc hemorrhage group (30.8% vs. 20.5%, respectively) compared with the non-hemorrhage group (10.9% vs. 9.2%, respectively). Superonasal and superotemporal central VF regions progressed more rapidly than other regions, especially in eyes with disc hemorrhage.

"Disc hemorrhages are an independent predictor for more accelerated central VF loss in glaucoma, especially in early stages of the disease," the study authors concluded. "Therefore, examination of the central VF using a 10-2 strategy should be considered in glaucoma patients with a history of disc hemorrhage for sensitive detection of disease progression."

David RCC, Moghimi S, Do JL, et al. Characteristics of central visual field progression in eyes with optic disc hemorrhage. Am J Ophthalmol. June 6, 2021. [Epub ahead of print].

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Broccoli, Saffron Could Fight Ocular Diseases

Diet and nutritional supplements have long been considered a resource in fighting ocular diseases and conditions such as cataracts, glaucoma, diabetic retinopathy and age-related macular degeneration (AMD). Now, new research suggests the addition of certain natural foods, along with a Mediterranean diet and nutraceutical supplements, can further benefit patients and reduce the risk of vision-impairing conditions.

"Broccoli, nuts, saffron and tiger nuts can help prevent/manage ocular diseases and also can help fight against certain risk factors related to visual impairment," the researchers wrote in their paper. "These foods display anti-inflammatory, detoxicating, anti-angiogenic, anti-apoptotic, photo-protective, antioxidant and neuroprotective effects to some extent."

In addition to their own investigations, the research team from Spain analyzed 177 studies conducted from 1983 to 2021 that reviewed the benefits of natural food, the Mediterranean diet and supplements for ocular health.

Broccoli's Benefits

The study touts this vegetable as an important source of micronutrients and fiber, carotenes (beta-carotene and lutein), vitamins (A, B, C and E), isothiocyanates, fatty acids (linoleic acid and palmitic acid) and minerals (calcium, iron, magnesium, potassium, phosphorus and sodium), in addition to amino acids.

The investigative team conducted a pilot intervention study involving 14 age- and sex-matched individuals. Half the participants consumed a daily amount of 375g of broccoli, equivalent to 10g of lutein, for four weeks. The researchers found a significant increase in the macular pigment optical density in the retinographies of the right eyes in the broccoli group.

"Broccoli is a dark, leafy green vegetable, and those all have good lutein," says optometrist and ocular nutrition expert Jeffrey Anshel, of Encinitas, CA.

In mice, saffron prevented retinal ganglion cell death common in chronic hypertensive eyes.

Saffron and AMD

This spice harvested from crocus flowers contains crocin isomers, zeaxanthin, lycopene and vitamin B12, the investigators explained. In early stages of AMD, research has shown, saffron can improve visual function by reversing the damage to photoreceptors and bipolar cells caused by oxidative stress. In another investigation, daily saffron intake improved retinal changes in patients with both dry and wet AMD.

Considering glaucoma, the researchers analyzed saffron in a mouse model of chronic ocular hypertension and found that saffron extract resulted in a reduction in both the number and signs of microglial cell activation as well as a down-regulation of the purinergic receptor P2RY12a, a marker of inflammation-related non-activated microglia. Saffron also prevented the retinal ganglion cell death that occurred in chronic hypertensive eyes, suggesting the neuroprotective effect of saffron could be due to its anti-inflammatory and antioxidant properties.

Chufa de Valencia

The authors conducted a recent study about the role daily intake of tiger nuts has on dry eye disease (DED). The pilot study included 20 women aged 45 to 70 who worked daily at computers. After adding tiger nuts to their diets, the women in this group showed a noticeable reduction of the signs, symptoms and subjective sensations of DED, including reduced blink frequency and higher tear break-up time and Schirmer test results.

Walnuts' Retinal Impact

Recent experimental evidence suggests that the main polyphenols of walnuts—ellagitannins and their metabolites—have beneficial properties against the oxidation processes of cellular components and in the inflammation pathways, in addition to positively influencing the intestinal microbiome, the authors noted. Still, Dr. Anshel cautions that vegetable omega-3s have their limitations.

"Plant sources of omega-3s aren't the best when it comes to metabolism compared with long-chain ones such as EPA and DPA, since conversion of the shorter-chain lipids aren't efficient in men and are barely efficient in women," he explains.

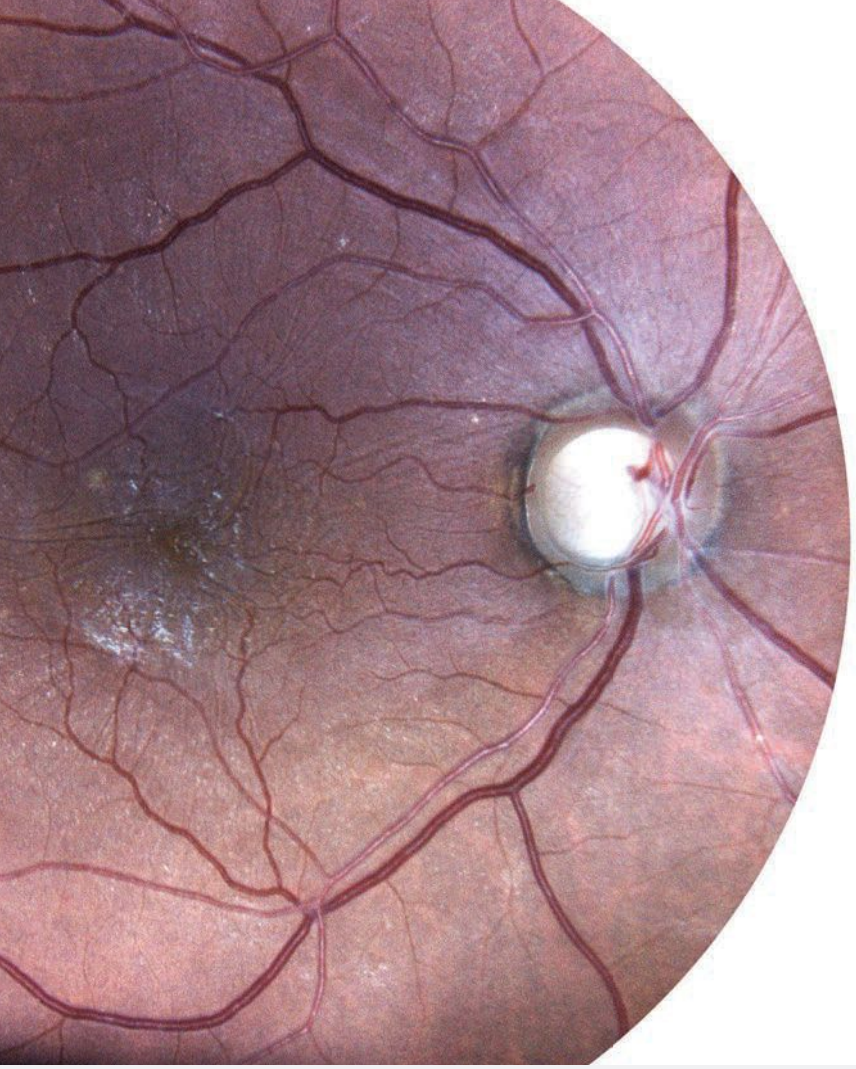
Takeaways

The authors suggest the potential antioxidant, anti-inflammatory and neuroprotective effects of natural foods, the Mediterranean diet and nutraceutical supplements could provide a promising, affordable option for patients at risk of vision loss.

When asked about the numerous supplements, diets and food recommendations to improve ocular health, Dr. Anshel says, "The bottom line is: there is no bottom line. There is no single magic bullet, and you can't just do one thing and affect all the different diseases in the body." Rather, it's a combination, he asserts.

"In my lectures, I ask the audience, 'How many people think they have the perfect diet?' No hands go up, except for a weightlifter who keeps track of all the food he eats. But that's rare."

Valero-Vello M, Peris-Martínez C, García-Medina JJ, et al. Searching for the antioxidant, anti-inflammatory and neuroprotective potential of natural food and nutritional supplements for ocular health in the Mediterranean population. Foods. 2021;10(6):1231.



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Stress, Lack of Sleep Can Trigger Uveitis

A recent study set out to determine which factors trigger inflammatory episodes of recurrent acute anterior uveitis (RAAU), a disease responsible for 10% to 15% of blindness in the United States. Stress and inadequate sleep were shown to be the two biggest predictors of future uveitis episodes.

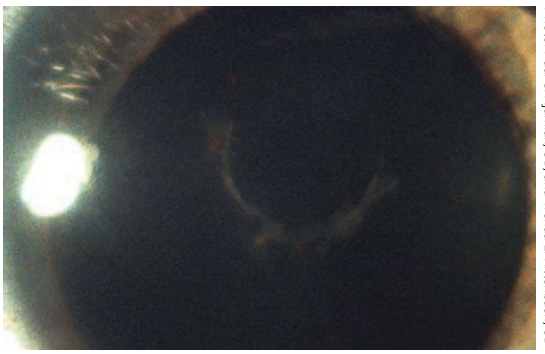

Photo: Paul C. Ajamian, OD, and Trenda L. Rittenbach, OD

These two factors can contribute to future attacks of RAAU.

This case-control study, conducted in Bangkok, included 78 RAAU patients over the age of 18. Participants completed an interview questionnaire asking them about potential factors associated with a uveitis attack, such as average sleep and exercise time, smoke and alcohol consumption, prodromal symptoms, low back pain, anxiety, life events, financial problems, eye trauma, accidents, history of an illness and stress. Patients reported on the presence of these factors during the month before their most recent attack. They also underwent a stress test.

Higher levels of stress and lack of sleep were associated with impending uveitis attacks in RAAU patients. "The odds of uveitis attack within the following month were about nine times in those with stress, and 12 times with sleep deprivation," the researchers wrote in their paper. "It appeared that other suspected factors, including exercise time, smoking, joint pain and alcohol consumption, were not significantly correlated with the uveitis attack." No life events were reported by patients in the questionnaire, so it's not known whether one could trigger a uveitis episode.

Lack of sleep was defined as getting less than the nightly seven hours recommended by the National Sleep Foundation. Sleep deprivation has been shown to alter immune system function over time, as well as increase C-reactive protein levels and upregulation of interleukin-6 and tumor necrosis factor. The upsurge of inflammatory cytokines may be what triggers the inflammatory attack in RAAU patients. As for stress, it may also activate the hypothalamic pituitary adrenal axis, leading to an altered immune response.

Repetitive episodes of intraocular inflammation pose the risk of tissue damage, glaucoma, cystoid macular edema, cataract and permanent visual disability. Inform patients that keeping stress levels low and getting seven to nine hours of sleep each night may help offset the frequency of uveitis episodes.

Neti N, Pimsri A, Boonsopon S, et al. Triggering factors associated with a new episode of recurrent acute anterior uveitis. Sci Rep. 2021;11:12156.

Sclerals Show Promise Against Infectious Keratitis

Recent years have seen newfound interest in scleral contact lens prescribing for more patients beyond those with corneal irregularities, and researchers are also exploring whether this lens modality could be used as a vehicle to deliver antibiotics in severe infectious keratitis cases.

The investigative team from Mexico added moxifloxacin to the fluid reservoir of sclerals and reported this alternative approach was both an effective and comfortable option to treat infectious keratitis. Additionally, they observed no complications or side effects in the 12 eyes that were treated for the condition .

The study used a scleral lens filled with 0.5% moxifloxacin as a reservoir and replaced it every 24 hours until epithelization was complete or the culture report and/or antibiogram demonstrated either a microorganism not susceptible or resistant to moxifloxacin. All patients completed at least one month of follow-up.

Of the 12 eyes, seven had culture-positive bacterial infection, two were mycotic and three had no culture growth. The researchers discontinued treatment in three eyes due to lack of response in one eye and the presence of mycotic infection in the other two. All infections resolved favorably at the final follow-up.

This novel approach may provide an equally effective therapy and avoid the need for frequent instillation of eye drops, the researchers noted.

"The main advantage was having the lens full of antibiotics, thereby avoiding the need to apply drops every hour or to have to wake up at night to apply drops. The protection provided by the scleral lens could also help improve keratitis symptoms," the investigators wrote in their paper for the *Cornea* journal.

Although the wait time was almost the same between scleral lens adaptation and saturation time, the number of drops required was reduced from 37 to five, which could also be a cost-effective positive aspect of the approach, the authors added.

Polania-Baron E, Santana-Cruz O, Lichtinger A, et al. Treatment of severe infectious keratitis with scleral contact lenses as a reservoir of moxifloxacin 0.5%. Cornea. 2021;40(7):831-6.

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Orbital Fracture Linked to Risk of Dry Eye

New research suggests that patients with orbital fracture are at higher risk of dry eye compared with the general population.

The investigation included patients from the Taiwan National Health Insurance Research Database. Overall, 46,179 and 184,716 participants comprised the study and control groups, respectively. Each patient in the case group was age- and gender-matched to four controls without orbital fracture.

The team found that during the follow-up period, the case group was more likely to develop incident dry eye (0.17%) than the control group (0.11%). Statistical analysis demonstrated that the case group had an almost fivefold increased risk of dry eye compared with controls.

When stratified by age group, the data showed that orbital fracture was most common among patients aged 18 to 29 years. Specifically, patients with orbital roof fracture had the greatest risk of developing dry eye. Regardless of receiving surgery or not, the patients with orbital fracture had a higher risk of developing subsequent dry eye.

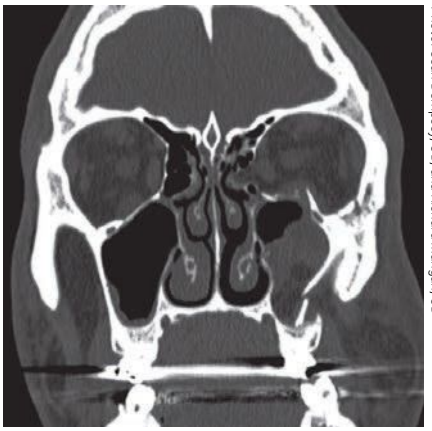

Photo: Sean Dempsey, OD, and Richard Mangan, OD

Mechanical compression from orbital trauma contributed to lacrimal gland injury.

"One plausible explanation for our observation was that the rate of dry eye occurrence following the orbital fracture was attributed to lacrimal gland injury," the authors wrote in their study. Given that the lacrimal gland is located in the superolateral aspect of the orbit, "this suggests that the anatomical disruption and mechanical compression from orbital trauma contributed to the resulting injury of the lacrimal gland, including hematoma, edema and vascular insufficiency."

Early recognition of orbital fractures with lacrimal gland involvement with raised awareness in the clinical setting "could preserve visual function and prevent further complications," the study authors concluded.

Hsu CYY, Tu JCY, Chung CH, et al. Risk of dry eye syndrome in patients with orbital fracture: a nationwide population-based cohort study. Healthcare (Basel). 2021;9(5):605.

Contrast Acuity Detects MCI in Alzheimer's Patients

Patients with Alzheimer's disease (AD) have been shown to exhibit retinal changes as well as impaired contrast sensitivity, color vision, visual attention and saccadic eye movements. However, a new study is among the first to focus specifically on cases of mild cognitive impairment (MCI) that represent prodromal AD, which may be more difficult to diagnose. These patients too may be identifiable by ocular examination, the study finds.

Fourteen subjects with MCI caused by AD and 16 controls without visual impairment were observed to determine performance variances. The average participant age was 75, and each had a best-corrected binocular visual acuity between 20/20 and 20/25. Participants underwent the following ocular tests:

- High- and low-contrast letter acuity (LCLA) testing
- Vision-specific quality of life scales, including the 25-Item National Eye Institute Visual Function Questionnaire and 10-Item Neuro-Ophthalmic Supplement
- OCT scans

The investigators also used a picture naming test common in cognitive assessment called the Mobile Universal Lexicon Evaluation System (MULES).

Both the MULES test of rapid image naming and binocular LCLA at 1.25% contrast were found to distinguish patients with MCI due to AD from participants without visual impairment. MCI patients were only able to identify one-third the number of letters seen by the controls, a difference of 10 letters in binocular LCLA. The MCI group's test times were 1.6 times longer, and they also committed three times as many errors.

"Lower (worse) binocular LCLA scores at 1.25% contrast, longer (worse) MULES test times and greater numbers of MULES errors were significant predictors of MCI vs. control status, accounting simultaneously for ocular pathology," the researchers said in their paper.

"These visual measures are showing early promise as future markers to identify disease states and to potentially follow patients in clinical trials and observational research. Notably, both MULES and LCLA were stronger predictors of the MCI disease status than hippocampal atrophy on MRI," the researchers said.

Rapid image naming and binocular LCLA could one day become diagnostic tools for prodromal AD. Larger and longer studies will help model the protocol's effectiveness and that of other visual tests as biomarkers.

Wu SZ, Nolan-Kenney R, Moehringer NJ, et al. Exploration of rapid automatized naming and standard visual tests in prodromal Alzheimer disease detection. J Neuro-Ophthalmol. 2021;00:1-9.

BRIEF SUMMARY OF PRESCRIBING INFORMATION

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

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

Initial U.S. Approval: 2017

1 INDICATIONS AND USAGE

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

4 CONTRAINDICATIONS

None

5 WARNINGS AND PRECAUTIONS

5.1 Pigmentation

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

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

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

5.2 Eyelash Changes

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

5.3 Intraocular Inflammation

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

5.4 Macular Edema

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

5.5 Bacterial Keratitis

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

5.6 Use with Contact Lens

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

6 ADVERSE REACTIONS

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

6.1 Clinical Trials Experience

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

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

8 USE IN SPECIFIC POPULATIONS

8.1 Pregnancy

Risk Summary

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

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

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

Data

Animal Data

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

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

8.2 Lactation

Risk Summary

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

8.4 Pediatric Use

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

8.5 Geriatric Use

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

13 NONCLINICAL TOXICOLOGY

13.1 Carcinogenesis, Mutagenesis, Impairment of Fertility

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

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

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

13.2 Animal Toxicology and/or Pharmacology

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

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

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VYZ.0109.USA.20 Issued: 5/2020

VYZULTA®
(latanoprostene bunod ophthalmic solution), 0.024%

THE HORSEPOWER YOU NEED TO LOWER IOP

Powerful IOP reduction with excellent tolerability[1,2]

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

TAKE A TEST RIDE AT VYZULTAHCP.COM

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

INDICATION

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

IMPORTANT SAFETY INFORMATION

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

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

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

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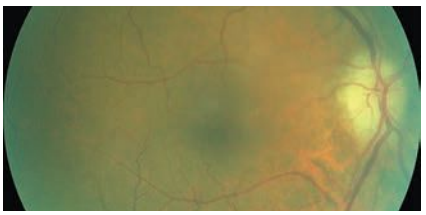

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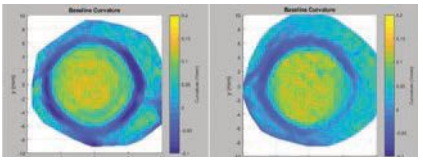

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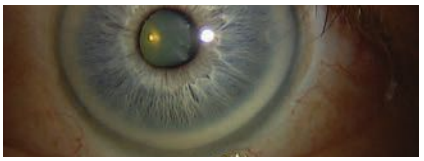

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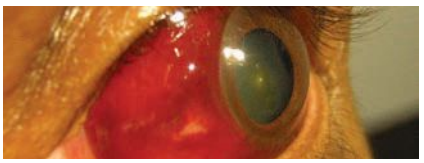

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IN THE BATTLEGROUND OF DRY EYE...

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

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

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

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

INDICATION

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

IMPORTANT SAFETY INFORMATION

Contraindication:

EYSUVIS, as with other ophthalmic corticosteroids, is contraindicated in most viral diseases of the cornea and conjunctiva including epithelial herpes simplex keratitis (dendritic keratitis), vaccinia, and varicella, and also in mycobacterial infection of the eye and fungal diseases of ocular structures.

Warnings and Precautions:

Delayed Healing and Corneal Perforation: Topical corticosteroids have been known to delay healing and cause corneal and scleral thinning. Use of topical corticosteroids in the presence of thin corneal or scleral tissue may lead to perforation. The initial prescription and each renewal of the medication order should be made by a physician only after examination of the patient with the aid of magnification, such as slit lamp biomicroscopy, and, where appropriate, fluorescein staining.

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

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

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

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

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

Adverse Reactions:

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

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

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

kala PHARMACEUTICALS

US-EYS-2100022 www.EYSUVIS.com

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

BRIEF SUMMARY OF FULL PRESCRIBING INFORMATION

INDICATIONS AND USAGE

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

CONTRAINDICATIONS

EYSUVIS, as with other ophthalmic corticosteroids, is contraindicated in most viral diseases of the cornea and conjunctiva including epithelial herpes simplex keratitis (dendritic keratitis), vaccinia, and varicella, and also in mycobacterial infection of the eye and fungal diseases of ocular structures.

WARNINGS AND PRECAUTIONS

Delayed Healing and Corneal Perforation—Topical corticosteroids have been known to delay healing and cause corneal and scleral thinning. Use of topical corticosteroids in the presence of thin corneal or scleral tissue may lead to perforation. The initial prescription and each renewal of the medication order should be made by a physician only after examination of the patient with the aid of magnification, such as slit lamp biomicroscopy, and, where appropriate, fluorescein staining.

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

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

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

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

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

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

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

ADVERSE REACTIONS

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

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

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

USE IN SPECIFIC POPULATIONS

Pregnancy—Risk Summary: There are no adequate and well controlled studies with loteprednol etabonate in pregnant women. Loteprednol etabonate produced teratogenicity at clinically relevant doses in the rabbit and rat when administered orally during pregnancy. Loteprednol etabonate produced malformations when administered orally to pregnant rabbits at doses 1.4 times the recommended human ophthalmic dose (RHOD) and to pregnant rats at doses 34 times the RHOD. In pregnant rats receiving oral doses of loteprednol etabonate during the period equivalent to the last trimester of pregnancy through lactation in humans, survival of offspring was reduced at doses 3.4 times the RHOD. Maternal toxicity was observed in rats at doses 347 times the RHOD, and a maternal no observed adverse effect level (NOAEL) was established at 34 times the RHOD.

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

Data—*Animal Data:* Embryofetal studies were conducted in pregnant rabbits administered loteprednol etabonate by oral gavage on gestation days 6 to 18, to target the period of organogenesis. Loteprednol etabonate produced fetal malformations at 0.1 mg/kg (1.4 times the recommended human ophthalmic dose (RHOD) based on body surface area, assuming 100% absorption). Spina bifida (including meningocele) was observed at 0.1 mg/kg, and exencephaly and craniofacial malformations were observed at 0.4 mg/kg (5.6 times the RHOD). At 3 mg/kg (41 times the RHOD), loteprednol etabonate was associated with increased incidences of abnormal left common carotid artery, limb flexures, umbilical hernia, scoliosis, and delayed ossification. Abortion and embryofetal lethality (resorption) occurred at 6 mg/kg (83 times the RHOD). A NOAEL for developmental toxicity was not established in this study. The NOAEL for maternal toxicity in rabbits was 3 mg/kg/day.

Embryofetal studies were conducted in pregnant rats administered loteprednol etabonate by oral gavage on gestation days 6 to 15, to target the period of organogenesis. Loteprednol etabonate produced fetal malformations, including absent innominate artery at 5 mg/kg (34 times the RHOD); and cleft palate, agnathia, cardiovascular defects, umbilical hernia, decreased fetal body weight and decreased skeletal ossification at 50 mg/kg (347 times the RHOD). Embryofetal lethality (resorption) was observed at 100 mg/kg (695 times the RHOD). The NOAEL for developmental toxicity in rats was 0.5 mg/kg (3.4 times the RHOD). Loteprednol etabonate was maternally toxic (reduced body weight gain) at 50 mg/kg/day. The NOAEL for maternal toxicity was 5 mg/kg.

A peri-/postnatal study was conducted in rats administered loteprednol etabonate by oral gavage from gestation day 15 (start of fetal period) to postnatal day 21 (the end of lactation period). At 0.5 mg/kg (3.4 times the clinical dose), reduced survival was observed in live-born offspring. Doses ≥ 5 mg/kg (34 times the RHOD) caused umbilical hernia/incomplete gastrointestinal tract. Doses ≥ 50 mg/kg (347 times the RHOD) produced maternal toxicity (reduced body weight gain, death), decreased number of live-born offspring, decreased birth weight, and delays in postnatal development. A developmental NOAEL was not established in this study. The NOAEL for maternal toxicity was 5 mg/kg.

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

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

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

NONCLINICAL TOXICOLOGY

Carcinogenesis, Mutagenesis, Impairment of Fertility—Long-term animal studies have not been conducted to evaluate the carcinogenic potential of loteprednol etabonate. Loteprednol etabonate was not genotoxic *in vitro* in the Ames test, the mouse lymphoma thymidine kinase (tk) assay, in a chromosome aberration test in human lymphocytes, or *in vivo* in the single dose mouse micronucleus assay. Treatment of male and female rats with 25 mg/kg/day of loteprednol etabonate (174 times the RHOD based on body surface area, assuming 100% absorption) prior to and during mating caused pre-implantation loss and decreased the number of live fetuses/live births. The NOAEL for fertility in rats was 5 mg/kg/day (34 times the RHOD).

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

Manufactured for:
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Watertown, MA 02472

Part # 2026R02

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Leadership in clinical care

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LETTERS TO THE EDITOR

Feedback and ideas from the optometric community.

SHARE YOUR THOUGHTS
Letters are welcome.
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▶ LOW MARKS FOR LOW VISION COVERAGE

I continue to be disappointed by the lack of attention given to low vision rehabilitation as a valuable component of the treatment models described in *Review of Optometry*. Two recent examples include "Blindsided" (May 2021) and "Five Questions on Dry AMD Monitoring and Management" (April 2021).

In "Blindsided," which outlines next steps in caring for a patient with newly diagnosed retinitis pigmentosa, clinical and genetic testing are discussed but no mention is made of the functional implications of this young patient's severe (and likely progressing) visual field loss.

Discussions regarding driving and other daily activities need to be had as soon as possible with this patient, with an eye toward preparation for the reality of future worsening and efforts to allow the patient to maintain functional vision. Even if the diagnosing provider is not in a position to personally provide comprehensive low vision rehabilitative care, mention should be made of timely referral as a key component in this patient's treatment plan.

In "Five Questions," the author makes mention of low vision services, but undermines this by advising that low vision referral be "considered" only in cases of advanced AMD. Referral shouldn't have to wait until a patient demonstrates geographic atrophy or a choroidal neovascular membrane. Multiple studies have concluded that low vision rehabilitation results in improved quality of life and that early referral better prepares patients with progressive conditions to handle further vision degradation.

The American Academy of Ophthalmology has defined early referral as the standard of care for patients with loss of visual acuity (<20/40), visual field or contrast sensitivity that interferes with one's activities. All optometrists should be leading the charge in making sure that any patient who meets the criteria has a comprehensive low vision rehabilitation evaluation performed, either in their own office or by referring to another optometrist experienced in performing these examinations.

When a leading publication like *Review of Optometry* continues to overlook this important treatment modality, it goes against the oath we've all taken to best care for our patients and it harms efforts by optometry and ophthalmology to solidify low vision rehabilitation as the standard of care. We've all received outstanding training in medical eye care, but we need to see past the diagnosis and take better care of the person experiencing vision loss.

—Joshua L. Robinson, OD
Director, Low Vision Rehabilitation
Vanderbilt Eye Institute
Nashville

I found the April letter by Megan Lott, OD, especially interesting. Her comments about lack of attention to vision therapy also apply to low vision, both long-time, effective services that are exclusively optometric. I agree with Dr. Lott that in taking on the medical model of eye care, we have moved away from the basics of our profession: providing patients with the clearest, most comfortable and most functional vision possible.

> **"People come to ODs because they want clear, binocular vision so they can function normally and enjoy important activities, not because they want to be tested and monitored."**

Optometry has become a profession that has focused on eye care and forgotten that we are dealing with the whole person. People come to ODs because they want clear, binocular vision so they can function normally and enjoy important activities. They do not come to us because they want to be tested and monitored. I also agree with Dr. Lott that it is astounding that our colleagues do not refer patients to other optometrists when they would benefit from low vision care or vision therapy.

I then found, four pages later, the column by Dr. Karpecki. He discusses how he feels that optometry should be the primary provider for AMD management. He mentions how exciting it is that there are new methods for early detection and monitoring of AMD. As exciting as these new techniques are to Dr. Karpecki, he should understand that what patients want is to see well. They don't want to be monitored, they want to be helped. They want to continue reading, driving, using a computer, watching their grandchildren, recognizing friends' faces and many other activities. They want to be independent. Dr. Karpecki would serve patients better by providing low vision help or referring to a low vision OD.

The medical model may represent advances for our profession, but let's not forget why the patients come to us and what they expect from us.

—David L. Armstrong, OD, low vsion optometrist
Roanoke, VA

▶ NEURO NEGLECTED

My takeaway from the April news story "Missed Neuro Diagnoses Lead to Patient Harm" was that the schools of optometry—and ophthalmology—are not keeping pace with needs of today's eyecare providers to adequately evaluate, diagnose, treat or refer ophthalmic neuro encounters. If they think they are, the statistics don't reflect this.

—Howard Levenson, OD
San Rafael, CA

Digital Health in Modern Optometry: Advanced At-Home Monitoring for Age-Related Macular Degeneration

Patients with AMD can receive comprehensive monitoring services from remote providers.

By Sean W. Smolenyak, OD

Patients with intermediate Age-Related Macular Degeneration (iAMD) may be at increased risk for converting to neovascular AMD (nAMD). Since frequent patient visits (ie, more often than every 3-6 months) to monitor for disease conversion can be impractical for clinicians and patients alike, Optometrists often turn to an Amsler grid. By instructing patients on how to use it, the hope is that patients will report signs of metamorphopsia that could suggest the presence of nAMD.

However, the Amsler grid is too simplistic a tool to screen for the worsening of such a complex disease. Incorporating digital health care models can help propel a practice into the realm of artificial intelligence (AI) and leverage the latest innovations in remote monitoring. In the case of monitoring iAMD patients for conversion to nAMD, the digital health care provider that leads the way is the Notal Vision Diagnostic Clinic.

Digital health care models have been embraced elsewhere in eye care. The iCare Home (iCare) is a tonometer designed for home-based IOP monitoring. Constant glucose monitoring, wearable heart monitors, and contact tracing technologies have become commonplace in endocrinology, cardiovascular medicine, and public health arenas, respectively. The time for embracing a digital health care model for retinal care has arrived.

NOTAL VISION DIAGNOSTIC CLINIC

Patients with iAMD who enroll in the Notal Vision Diagnostic Clinic's ForeseeHome AMD Monitoring Program perform daily at-home testing with automated data analysis for evidence of conversion to nAMD. At-home monitoring supplements routine in-person examinations, allowing clinicians to have patients continually monitored for evidence of nAMD between office visits. In the case of the ForeseeHome AMD Monitoring Program, the system uses a daily preferential hyperacuity perimetry test.

A HIPAA-compliant portal allows physician access to individual patient ForeseeHome AMD monitoring data provided by the Notal Vision Diagnostic Clinic.
Image: Notal Vision

Results from the daily at-home tests are securely uploaded to the cloud and are analyzed by an AI algorithm to detect aberrations from the patient's baseline metamorphopsia map. In the event that a change is detected, which may indicate that a patient has converted from iAMD to nAMD, an in-house eye physician at the Notal Vision Diagnostic Clinic reviews the patient's most recent visual testing.

If the in-house provider observes that nAMD activity may be present, the Notal Vision Diagnostic Clinic alerts the referring Optometrist's office via an encrypted email that contains a link to an online portal with the patient's most recent at-home diagnostic examination. If the referring clinician's office does not read the message, the Notal Vision Diagnostic Clinic will then contact the office via telephone. At this point, the referring clinician decides the best course of action, which is usually an in-person examination. The Notal Vision Diagnostic Clinic only communicates with the patient

directly if they are unable to contact the office after multiple attempts. By not interfering with the doctor-patient line of communication unless required, the relationship and trust between the patient and the Optometrist is maintained.

An Optometric practice that refers patients to the Notal Vision Diagnostic Clinic's ForeseeHome AMD Monitoring Program does not incur any costs. After an order is sent to enroll the patient in the program, the Diagnostic Clinic is responsible for confirming benefits with the patient's health insurance, shipping the device to the patient, remotely training the patient on how to set up and use the ForeseeHome platform, as well as monitoring patient compliance. With the Notal Vision Diagnostic Clinic taking on these responsibilities, the referring practice can focus on providing care to patients rather than on managing the logistics of eligibility, inventory, and setup.

If the Notal Vision Diagnostic Clinic detects that at-home monitoring has stopped, the patient is contacted directly to inquire about the patient's health and to offer troubleshooting support. Patients who wish to take their ForeseeHome device to a new location—perhaps on vacation or to another home—can coordinate with the Notal Vision Diagnostic Clinic to reconnect to the platform.

Clinicians who wish to monitor use patterns and testing results for particular patients have 24/7 access to online patient records. Other clinicians might prefer monthly summaries of patient data or may opt to review patterns and results from the time period shortly before a patient's next in-office examination.

Just as routine in-clinic monitoring alone is inadequate for the detection of nAMD conversion, home-based monitoring by itself is not the most effective method for observing evidence of disease activity. At-home monitoring should be viewed as a supplement to, not a replacement of, in-clinic examinations.

HOW THE LATEST DATA SUPPORT HOME-BASED MONITORING

Two recent studies found that early detection of nAMD may be key to preserving vision, and that use of an at-home/in-person model for detection is an effective screening method for disease conversion.

A 2020 retrospective study reviewed data of real-world patients with nAMD. The study authors found that eyes that presented with at least 20/40 VA at baseline maintained a mean VA of at least 20/40 after 1 and 2 years of anti-VEGF therapy.[1] However, among eyes that had less than 20/40 VA at baseline, mean 20/40 VA was not achieved at either 1 or 2 years. Approximately 66% of real-world patients in the study presented with VA worse than 20/40, illustrating that a majority of real-world patients lose significant vision before being detected.

A 2021 retrospective study reviewed the data of patients who used the Notal Vision Diagnostic Clinic and had at least 20/60 VA at baseline (ie, when at-home monitoring was prescribed).[2] Patients in the study were monitored for disease progression by both the device and by in-person examination conducted routinely or when symptoms presented.

Researchers identified 306 patients who converted from iAMD to nAMD. Among them, 69% of patients had disease detected via at-home monitoring. Median baseline at the time of nAMD detection was 20/32-1, which is above the 20/40 threshold described in the 2020 study above. Approximately 36% of patients who converted to nAMD had at least 20/40 VA at baseline, and 81% of those patients had 20/40 VA when nAMD disease activity was detected.

Given the findings of these two studies, Optometrists should strongly consider referring their iAMD patients to the Notal Vision Diagnostic Clinic, facilitating early detection and a qualified referral for treatment for patients who convert to nAMD.

PUSHING EYE CARE INTO THE MODERN AGE

As patients become more familiar with digital health care models, they may come to expect that their Optometrist embrace such frameworks. The Notal Vision Diagnostic Clinic uses at-home monitoring to supplement the in-person examinations that patients have come to rely on without interrupting the workflow of a practice, thus enabling maximum patient engagement with minimal clinical disruption.

In this digital health care model, at-home testing devices, AI systems, remote monitoring centers, and a patient's Optometrist combine forces to provide the most comprehensive monitoring system available to patients with iAMD. For some, it could mean the difference between early intervention and permanent vision loss.

Sean W. Smolenyak, OD, practices optometry at the Albemarle Eye Center in Edenton, North Carolina.

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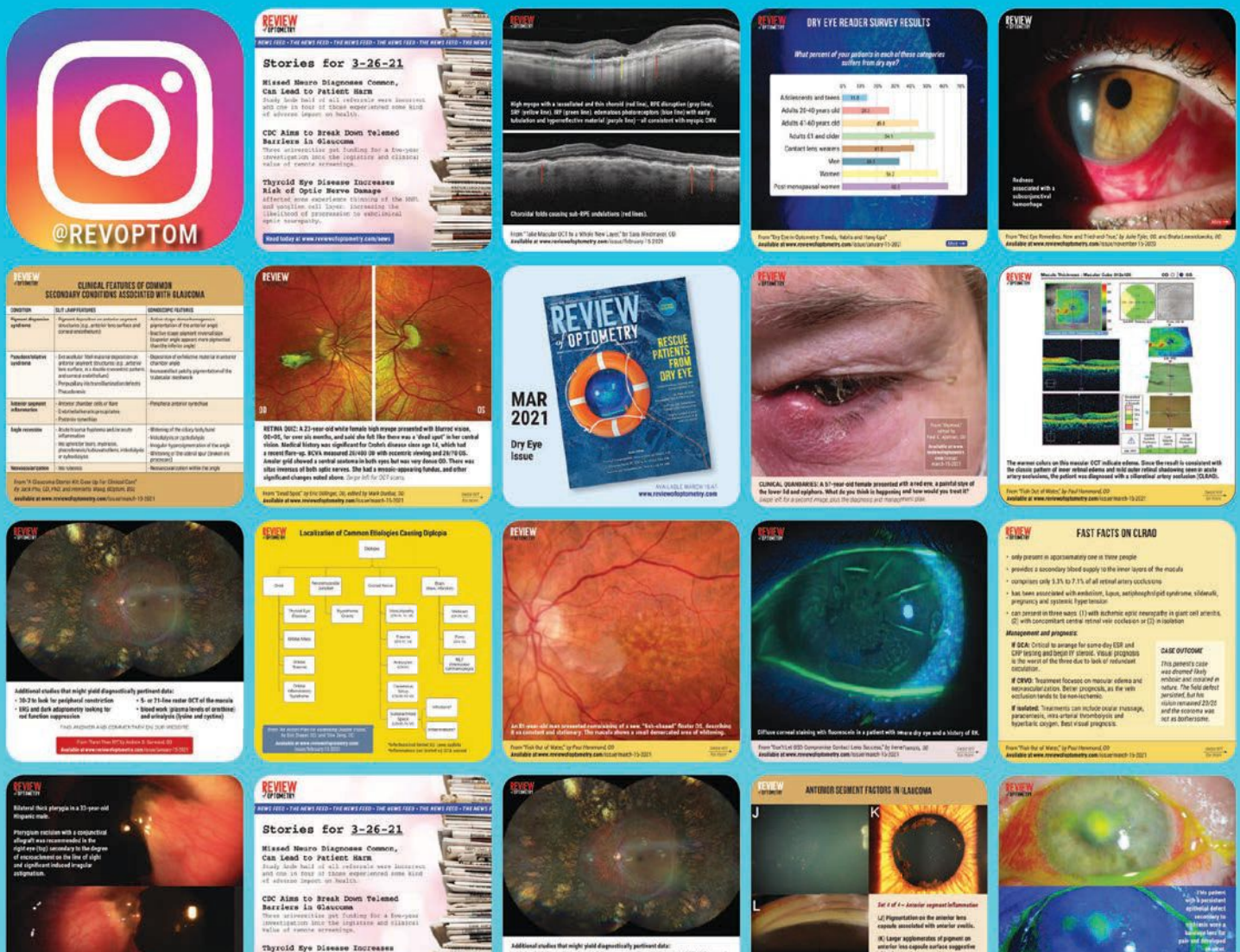

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BY JACK PERSICO
EDITOR-IN-CHIEF
OUTLOOK

Legislation With Vision

Both chambers of Congress are advocating expansion of Medicare to include eyecare services. Let's support it.

A simple pair of glasses can radically change the lives of a billion people around the world, according to a 2019 report from the World Health Organization. Here in the US, estimates say about eight to 10 million Americans need refractive correction but don't have it. Add in the untold millions who are undercorrected or still using an outdated Rx and the numbers quickly mushroom.

That millions here and a billion worldwide could be helped by such a simple thing, *but aren't*, is nothing short of a travesty. This isn't rocket science, it's refraction—the core component of optometric care for over a century.

So, it's welcome news that legislation introduced in the House of Representatives on June 28 would expand Medicare coverage to include routine eye exams and vision care materials. With the stroke of a pen, 60 million people would suddenly get these benefits.

The American Optometric Association says that 20.5 million Medicare beneficiaries have vision problems but only 57% received an eye examination during the previous year.

According to an AOA report, the House bill—called the Medicare Vision Act of 2021—would:[1]

- Expand Medicare Part B coverage to include annual refraction and contact lens fitting services.
- Ensure direct administration of the benefit by Medicare, to keep penny-pinching vision plans out of the mix.
- Provide coverage up to $100 for one pair of eyeglasses or a two-year supply of contact lenses each year.
- Require the materials benefit be administered by Medicare's Durable Medical Equipment program instead of third-party materials sellers, who are often more interested in their own outcomes than those of senior citizens.
- Provide a pathway to coverage for the low vision aids that are increasingly necessary in an aging population.

"We want to make sure that seniors can live independently for as long as possible," said House Rep. Kim Schrier (D-Wash.), one of the sponsors, in a statement. "An important factor of independent living is making sure that they can see well enough to drive to appointments, walk safely around the house and carefully read prescriptions."

Over in the Senate, a similar plan is afoot, this one to provide not just vision but dental and hearing services. Sens. Bernie Sanders (I-Vt.) and Chuck Schumer (D-NY) are champions of it.

These plans will surely meet with pushback for all the usual reasons. Let's hope something comes of it, as the need is obvious and the fix—your exam chair—is ready and waiting. The public could certainly do with more attention to their vision needs.

Vision care perhaps deserves greater attention inside the profession—and this magazine—too. We often hear from low vision specialists when our coverage of vision-impairing eye diseases gives short shrift to the help available through low vision aids and services. You can see this month's letters page for such discussions, in fact.

Point taken: medical eye care may well be where ODs' educational needs are the greatest right now, but their patients still just want to see better when they leave than when they arrived. ■

1. AOA advocacy helps shape new U.S. House bill to expand essential eye health and vision coverage for Medicare beneficiaries. www.aoa.org/advocacy. July 1, 2021.

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

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

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BY PAUL M. KARPECKI, OD
CHIEF CLINICAL EDITOR

Become Who You Are

With legal standing and abundant patients, optometrists can and should be masters of glaucoma.

With last month's announcement of Texas ODs being able to independently manage glaucoma patients and Massachusetts's law in January allowing topical glaucoma prescription authority, all 50 states now allow our profession to effectively manage this blinding condition. These legal rights mean we must become the primary providers and prescribers for glaucoma.

Optometry conducts 85% of all comprehensive eye exams in the US. You can expect that for every 100 exams you perform, about three should be glaucoma patients or suspects. Thus, you instantly have a glaucoma population to manage by simply seeing primary eye care patients, and these cases can add up. Below I'll discuss the ways to properly and effectively test and manage glaucoma patients.

Getting Started

To effectively manage glaucoma, you'll need the ability to examine the optic nerve and determine its size, estimate vertical vs. horizontal C/D ratio and recognize the presence of disc hemorrhages, pallor or notching. With time, you'll pick up nerve fiber layer defects. This, combined with IOP, visual field testing and pachymetry, will get you started. Before long, however, an OCT device will be particularly warranted.

To advance further, especially in diagnosis of borderline cases, newer technologies to consider include hysteresis, ganglion cell complex assessment on OCT, faster perimetry for patients who are reliable visual field test takers, progression analysis software and advanced pupil testing.

> **For every 100 exams you perform, about three should be glaucoma patients or suspects. Thus, you instantly have a glaucoma population simply by seeing primary eye care patients.**

Two that have helped me greatly are hysteresis with the Ocular Response Analyzer (Reichert) and advanced pupil testing with EyeKinetix (Konan). Average hysteresis is around 10.3mm Hg. A glaucoma patient with low values is more likely to show visual field progression; if they are a glaucoma suspect, they're more likely to develop glaucoma.[1,2]

Pupil testing is also quite beneficial because the vast majority of glaucoma patients have asymmetric ganglion cell loss, resulting in a positive relative afferent pupillary defect (RAPD).[3] EyeKinetix easily reveals a clinically significant RAPD. Even when C/Ds, OCT or pachymetry were borderline in many of my glaucoma suspects, those with no RAPD and hysteresis above 10.5 haven't progressed to glaucoma. It allows me peace of mind to know when to start treatment and also to identify who is likely to be a fast progressor (*e.g.*, hysteresis < 8mm Hg).

Treatment Advances

We're entering an era where focusing on the ocular surface is critical to maintaining drop adherence. We need to move beyond medical management in patients experiencing ocular surface disease. Optometrists can provide selective laser trabeculoplasty (SLT) in six states. Whether you practice in a state where you can perform the procedure or if you comanage, SLT is a great alternative to beginning a second medication. In patients with ocular surface disease, dexterity challenges or adherence issues, it may even be a better initial choice.

Minimally invasive glaucoma surgery is a low-risk option for cataract patients. This is a great opportunity for those with glaucoma, but after cataract surgery it's no longer an option. New modalities like bimatoprost SR (Durysta, Allergan) show lasting effects for more than 18 months after a single time-released implant is inserted in the anterior chamber.[4]

Glaucoma management is squarely in optometry's domain. It's most often a slowly progressive disease that gives us ample time to contemplate our next move. Patients need years of ongoing care and, with that, a lasting bond with a trusted, long-time care provider. In short we have the patients, and the patience, for it.

There is a safety net—specialists that handle advanced forms like pigmentary glaucoma and cases that progress while on treatment—but it's imperative you make glaucoma a part of your everyday practice. ■

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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

There's a Drop For That

These ocular meds are a hot commodity for both patients and practitioners.

Eye care and eye drops have been carrying on like the chicken and the rooster for centuries. The global market for ophthalmic drops is currently estimated to be $36 billion to $39 billion US. That's not as much as the government spends on protecting the snail darter and Sesame Street, but at least in my world, that's still a whole lot of billions.

The eye's protective system in our body's tear chemistry is elegant and would be virtually indestructible if we didn't eat and drink what we eat and drink, stare at screens all day and fight like dogs to live longer and longer. Cavemen didn't need drops. They cured itchy eyes by being eaten by saber-toothed tigers before they turned 16. No college funds were ever needed either.

But now, dry eye drops are just about the sexiest thing in the world to both patients and doctors. Our patients want the magic eye drop to solve all of their problems. And the manufacturers work like hell to make sure we know that their drop is, in fact, *the* end-all, be-all when it comes to ocular dryness and everything else. Want to solve the border problem? Artificial tears.

Oh, yes, we all have our favorites. Me? I personally like the ones with the prettiest labels. They seem to work the best for my patients in Texas where it only rains when there is a tornado.

I remember so well the look on many of my patients' faces through the years during conversations that went just like this:

Me: "Tell me about your eyes."

Patient: "They water all the time."

Me: "That's because they are dry."

Patient (to herself): "Great. The dumbest eye doctor in America."

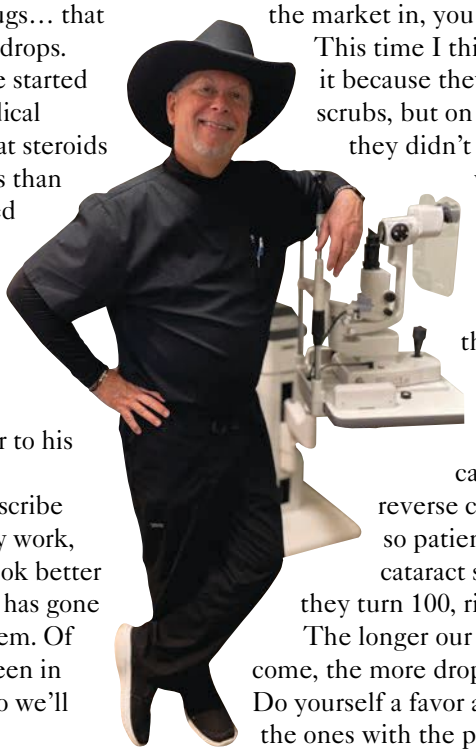
Me: "You will need to take these drops with the pretty label."

Patient (to herself): "Said the dumbest eye doctor in America."

But dryness is only the tip of the eyedropper. Now, there are drops for everything. There are drops to slow myopia, and you know what, they actually seem to work. There are drops for bacterial infection, which almost zero patients actually have when we prescribe the medications that actually create super bugs… that require even more drops.

And don't get me started on steroids. In medical school, we learn that steroids are more dangerous than razor blades wielded by an angry eight-year-old orangutan. If you prescribe steroids, you might as well be Dr. Kevorkian, except he was nicer to his patients than you.

Me? I like to prescribe steroid drops. They work, patients feel and look better and, so far, nobody has gone blind or died on them. Of course, I've only been in practice 41 years, so we'll see if this changes.



What about mydriatics, you may ask? Well, what about them? What's your question? Are they necessary? Sure, if you miss stuff in your examination that you should have easily seen and documented with widefield retinal photography. There are only two things patients hate more than dilation by drops: the puff tonometer and spending an extra $39 to get the pictures.

Did you know that a well-known over-the-counter red eye drop has been demonstrated to inactivate the coronavirus? As we all start climbing back into airplanes, shouldn't the public be told about that? Just asking…

And the drop that cures presbyopia? Patients are very excited about that. I remember a point 11 years ago when I was told it would be approved within two years. Some reps were in my office recently. They told me their drop would definitely be on the market in, you got it, two years. This time I think they meant it because they were wearing scrubs, but on the other hand, they didn't bring bagels, so who knows.

If one of the many eye drops can actually slow or reverse the hardening of the crystalline lens, then I assume it can also slow or reverse cataract formation so patients may not need cataract surgery until they turn 100, right?

The longer our lifespans become, the more drops we'll need. Do yourself a favor and recommend the ones with the pretty labels. ■

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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Don't Blow Your Top

Confrontation fields may be the only clue to this disorder.

Q An 18-year-old female presented for a routine exam with no visual complaints, but confrontation visual fields revealed an inferior defect in one eye. What tests should I perform and what is in the differential diagnosis?

A "Many causes of a visual field defect are easily recognized, such as a retinal detachment or glaucoma," says Paul C. Mitchell, OD, of Eye Care of Delaware. "Others are more elusive and require an extensive work-up." He adds that uncovering the etiology of less common conditions always requires a more deliberate evaluation. Investigating retinal, optic nerve and optic pathway causes is essential.

Start with in-office testing, including a thorough history, best-corrected vision, pupils, color vision, intraocular pressure, dilated eye exam, formal visual fields and OCT. B-scan ultrasound, fluorescein angiography and fundus autofluorescence (FAF) may also be necessary.

Element of Surprise

The patient's visual acuity was 20/20 OU. Pupillary testing showed no afferent defect. Confrontation fields showed a defect inferiorly OD. Pressures measured 16mm Hg OU. Visual fields disclosed inferior altitudinal defects greater in the right eye than left (*Figure 1*). OCT revealed superior RNFL loss OU with a single peak. Superior ganglion cell layer loss was also noted. Careful optic disc exam showed a superior entrance of the central retinal artery and vein with loss of optic nerve tissue adjacent to it. There was no disc edema or pallor (*Figure 2*). B-scan ultrasound and FAF ruled out optic nerve head drusen. An MRI was ordered and was negative. Based on the appearance of the optic nerve, OCT and visual field defects, a diagnosis of bilateral superior segmental optic nerve hypoplasia (SSOH) was made.

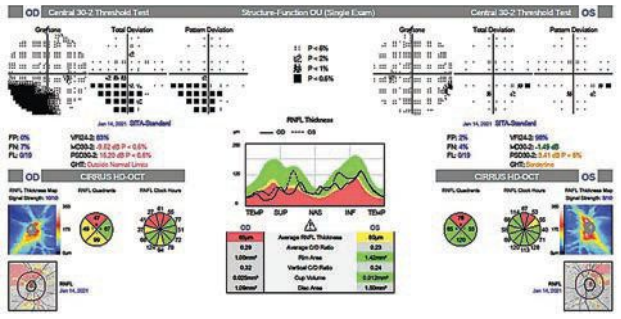
SSOH, or "topless disc syndrome," is a form of optic nerve hypoplasia. There is loss of nerve tissue superiorly on the disc with a relative superior entrance of the central retinal artery and vein. There can be superior pallor and a superior peripapillary halo adjacent to the disc. OCT findings typically show loss of the RNFL superiorly with a single peak and a corresponding inferior visual field defect. This condition tends to be nonprogressive.

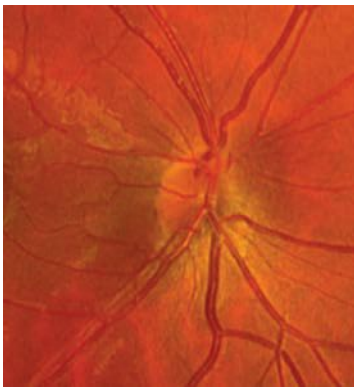
"Because this is a congenital condition, most patients are unaware of their visual field loss, and central vision remains good," Dr. Mitchell says. He notes females are more affected than males, there is an association with gestational diabetes and the condition occurs in 0.3% of Asians.[1-4]

Check for Signs

When evaluating a young patient with field loss, look at the disc for signs of drusen, optic pit or glaucoma. Consider a B-scan or FAF to detect buried drusen. A B-scan would show a highly reflective nodule and FAF would exhibit autofluorescence. A history of sudden vision loss and pain with eye movement would point toward an inflammatory event such as optic neuritis. Check color vision, pupils and order an MRI of the brain and orbits to confirm the diagnosis of optic neuritis. Look at the visual field pattern along with disc pallor or swelling that might indicate an intracranial cause. Careful dilated retinal exam is also key.

"During every eye exam you perform, routine or not, confrontation fields are critical and may be the only clue of an ocular disorder," Dr. Mitchell notes. In our patient, a careful disc exam with ancillary testing confirmed SSOH. We expect her nerves and visual fields to be stable over time. ■





Figs. 1 (above) and 2 (right). VF and OCT as well as fundus images are useful in diagnosing SSOH.

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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

Make Glaucoma Pay

Preventing claim rejections and audit exposure is key.

The other side of the coin in glaucoma management is following the rules of creating, maintaining and managing the medical record; when properly done, it can prevent claim rejections and unnecessary exposure to carrier audit. The good news is that these rules are simple, straightforward and easy to follow.

State Your Clinical Rationale

When ordering any special ophthalmic test, you will submit to a third party for payment, clearly establish why you ordered the test and why it's necessary for the patient. Each test must meet the requirement for medical necessity, which is based on a clinical finding discovered during the patient exam. Your medical record must contain a written statement of this medical necessity. If you keep in mind stating *what* you need to do, *why* you need to do it and *when* you need to do it, you will be in compliance with most medical necessity provisions of carrier rules.

Be Specific

When I do an internal audit for a practice, I frequently see in patient records what many doctors call a "glaucoma check." The chief complaint (CC) might say, "Patient returning for glaucoma check," or sometimes nothing more than, "IOP check." Simple phrases mean different things to different people. Avoid all-purpose phrases in the medical record. Rather, describe exactly why the patient is returning to the office.

The CC requirement can be fulfilled properly if you direct the patient to return to the office for a specific reason at an appropriate interval. The plan section of your medical record should include a statement such as: "Patient to return to clinic for evaluation of IOP and optic nerve Q3 months or PRN should additional symptoms arise," rather than, "RTC three months for glaucoma check." The former statement tells the record what you want the patient to do and why.

> **State *what* you need to do, *why* you need to do it and *when* you need to do it.**

When the patient returns, the CC should read: "Patient returning to clinic per doctor-directed order for evaluation of IOP and optic nerve." If you have orders pending for special ophthalmic testing, list them in both the plan and the reason for the visit.

The same patient may require different levels of examination and testing on different visits, and our records need to properly reflect that.

Complete Requirements

Each special ophthalmic test you perform requires its own interpretation and report (I/R) to be considered complete or billable, and each test and I/R must stand on its own. An I/R should contain:

a. Clinical findings.
b. Reliability of the test.
c. Comparative data.
d. Clinical management—how the test results will affect management of the condition/disease, *e.g.*:
   i. Change, increase or stop medication.
   ii. Recommendation for surgery.
   iii. Recommendation for further diagnostic testing.
   iv. Referral to a specialist or subspecialist for additional treatment.

If you haven't completed an I/R for each clinical test performed, then it is deemed that the test was never performed and was in conflict with your provider agreement or contract.

Keep an Eye on Updates

The combination of what CPT codes are allowed to be performed on the same date of service and the modifiers needed if you have to break an established rule are updated quarterly, so it is vital to use some form of technology, such as CodeSafePlus, to keep up to date with the rules. Carriers follow these rules when approving or denying claim submissions.

The various component output of code combination rules shows that:

1. The rule is *Active* or *Inactive*.
2. The code combination is *Allowed* or if there is a code *Conflict*.
3. The *Conflict* can be overridden by use of a modifier or not.
4. The *Administrative Explanation* or reason for the *Conflict* rationale.

Understanding these is essential to getting paid properly on your submitted claim and avoiding audit exposure. Improper use of modifiers is a primary trigger for carrier audits.

Providing great clinical care for your glaucoma patient is only half the battle; maintaining a proper clinical record that establishes medical necessity, clearly states your orders and follows the rules of the CPT and CCI is just as important for clinical success. ■

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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OPTOMETRY AT WORK: The How, When and Where of Who Delivers Care

Wide-ranging study finds a more balanced distribution between the genders, yet the profession falls short with minorities.

BY JANE COLE
CONTRIBUTING EDITOR

Over the next decade, the optometric workforce is projected to experience growth of 1.4% each year, a continued shift toward a female OD majority and a more limited additional capacity for the profession to expand than previously suggested, a recent national survey suggests.[1]

The investigation, based on 2017 data and published in *Optometry and Vision Science*, also found a lack of diversity in the profession. Minorities are poorly represented in optometry, which the authors cite as concerning and a barrier to care. Specifically, only 3.8% of respondents self-identified as Hispanic or Latino, 0.4% as Black/African-American and 0.2% as Native American.

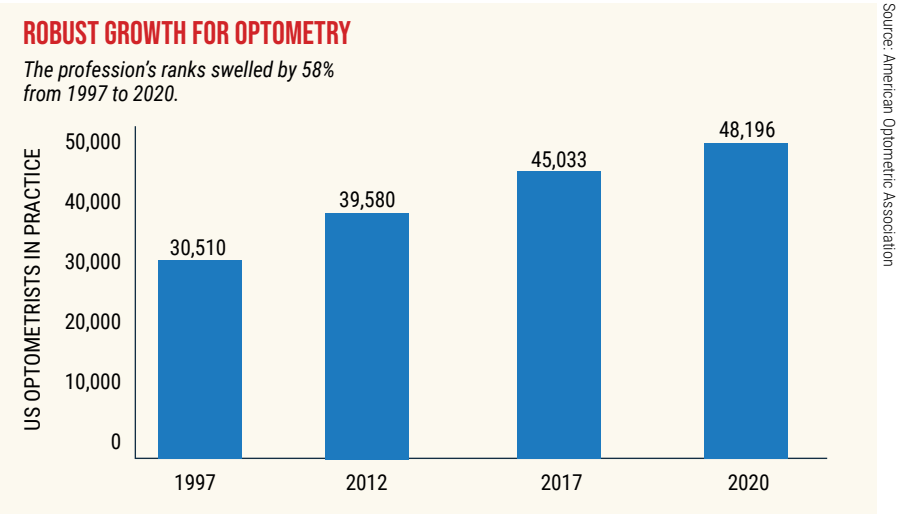
"This is clearly much lower than the overall population's makeup and hopefully is a wake-up call to our profession to encourage minority recruitment," says Brian Chou, OD, of San Diego.

Researchers distributed the 2017 National Optometry Workforce Survey to roughly 4,000 ODs from the AOA's database. The results reflected the responses of 1,158 optometrists.

The study found the optometric workforce is projected to grow annually by just 0.6% to 0.7% more than the United States population, and that trend should stay consistent over the next 10 years if no additional optometry programs open.

The investigation also reported some notable shifts compared with similar workforce data from 2012, published by the Lewin Group in 2014, that suggested a much higher potential for the then-current workforce to see more patients, as well as a significant disparity in productivity between men and women working in the profession.[2]

"We found there really weren't significant differences when we challenged the assumption that the productivity of women wasn't the same as men, and also, as a profession, we've really moved toward a more equitable and balanced workforce," says lead researcher David A. Heath, OD, EdM, president of the State University of New York College of Optometry.



Source: American Optometric Association

Growth in the optometric workforce has outpaced that of other medical professions.

Women Edge Past Men as Dominant Gender

As more women enter the profession, the workforce continues to experience a shift in the male-to-female ratio, says Dr. Heath.

Female ODs represented 43% of responding doctors in the survey. In 2017, 45% of ODs in the AOA's database were women (20,249). By January 2020, the number of female optometrists grew to 23,367 (49%). Taking into account that roughly 650 more women than men now graduate each year from optometry programs, and that most retirees currently are male, the authors predict more women than men are now actively practicing optometry in the United States.

Females Claim Majority in Optometry Schools

A few years ago, optometrist Pamela Miller of Highland, CA, lectured at her alma mater, the Southern California College of Optometry. When the longtime private practice owner looked out at the crowd of students and noticed it was predominantly female, she asked herself, "Where have all the men gone?"

Dr. Miller, who opened her practice cold 50 years ago, recalled being one of only a handful of women who attended the optometry school during the early 1970s.

After graduation, Dr. Miller was the first woman to serve on the California State Board of Optometry and the first female board member of the California Optometric Association. Still, despite being at the forefront of women entering the profession, Dr. Miller says she was denied a bank loan of $34,000 to purchase her practice initially due to her gender, despite the fact that she owned residential property and personally knew the bank loan officer.

Fast forward to today and gender discrepancies in the profession have narrowed significantly, including in optometry school enrollment.

In the 2017-18 academic year, there were 4,830 females and 2,294 males enrolled as full-time optometry students at US schools and colleges of optometry, including Puerto Rico, according to the Association of Schools and Colleges of Optometry (ASCO).[3] In fact, female optometry students have outnumbered males for at least the past decade.[3]

For optometry, the future—and much of the present—is female. Women often make up the lion's share of graduating classes, as seen here among Berkeley's 2021 grads, while retirees exiting the field skew heavily male. The 2017 National Optometry Workforce Survey documents female ODs' gains both in sheer numbers and also hours worked and patients seen.

"Throughout our profession, entering class profiles are approximately 70% female, and have been for several years now," says optometrist Chris Wroten, partner at the Bond-Wroten Eye Clinics in Louisiana and adjunct professor at Southern College of Optometry. "As a result, more and more female doctors of optometry are thankfully sharing their leadership skills by assuming prominent roles in their state associations and within the AOA and other optometric organizations.

Dr. Wroten served as an expert panel member for the 2012 AOA/ASCO Eyecare Workforce Study and is currently the moderator of the AOA Presidents' Council meetings, president of the Louisiana State Board of Optometry Examiners and Chair of SCO's Board of Trustees. He also supervises his practice's primary care and ocular disease residency program and works with student externs.

In his numerous roles in the profession, Dr. Wroten believes optometry is following the same trend as medicine and dentistry—the gender pay gap still exists but is showing signs of closing, while more and more women, and fewer men, are applying to medical, dental and optometry school.

"The women in our profession I've been blessed to know and work with are all extremely passionate about optometry, care deeply for their patients, have an unsurpassed work ethic and see every bit as many patients as their male counterparts, while still striking an appropriate work-life balance," says Dr. Wroten, who works with several female doctors of optometry at his practice, including his wife.

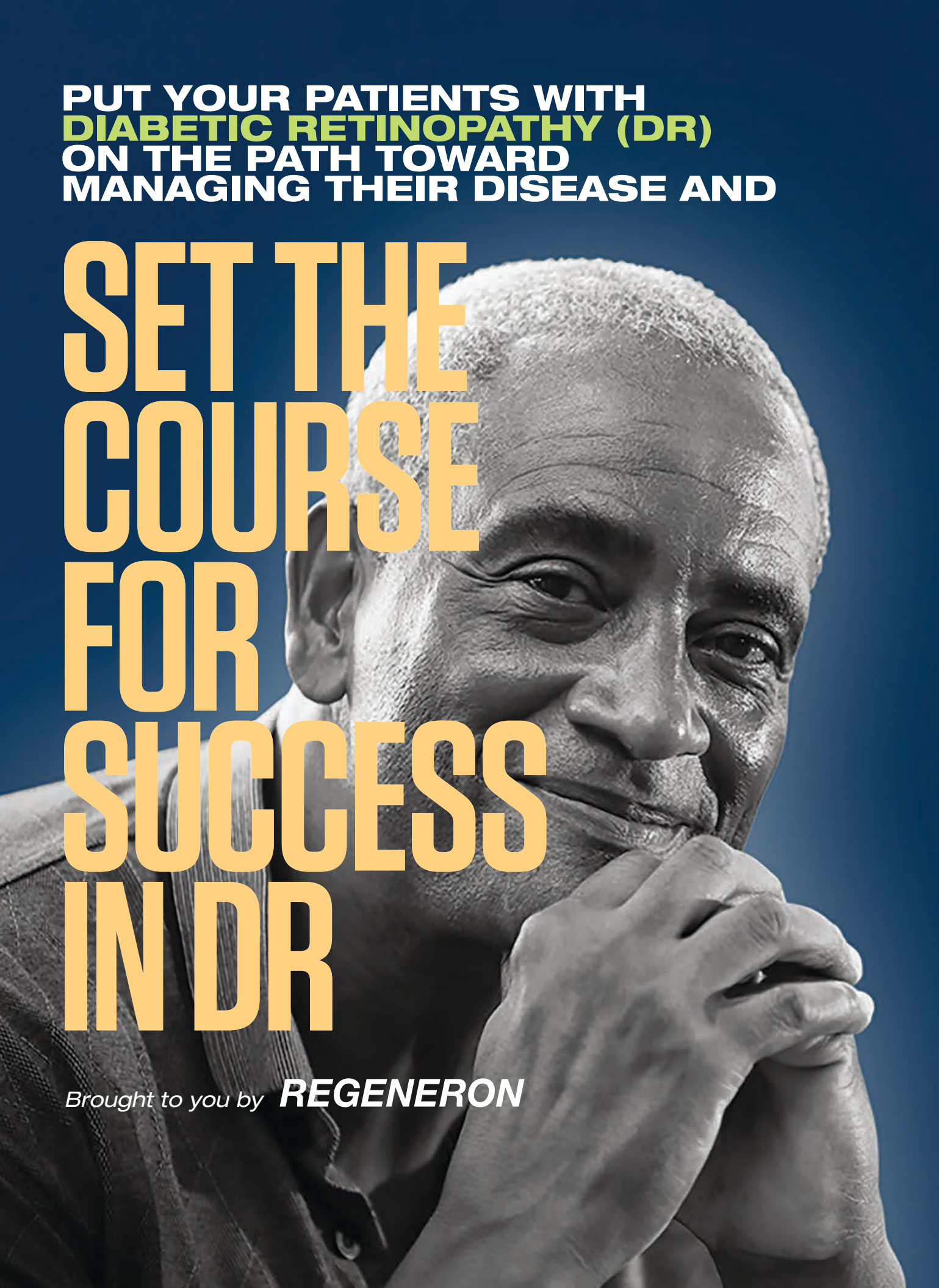
Productivity Assumptions Questioned

It has commonly been assumed that female ODs work fewer hours than men and see fewer patients; however, the current survey refutes this theory and illustrates an evolving and equitable workforce, the authors note.

Looking at weekly work schedules, women and men logged about the same number of hours: 37.5 for women vs. 38.9 for men. This difference wasn't significant and is notably smaller than the difference reported in the Lewin study (women: 38.55 hours; men: 42.15).

Similarly, the gender gap narrowed a bit in the number of weeks worked per year. In 2017, women reported working an average of 47.7 weeks while men worked 48.5 weeks annually. In the 2012 Lewin data, women reported working about 46.7 weeks a year vs. 47.8 weeks a year for men.

Considering productivity, women and men both saw essentially the same number of patients per hour: 1.97 for women vs. two for men. This is in contrast to the Lewin study, which reported lower productivity in women based on patients they saw per hour: 1.63 for women vs. 1.89 for men.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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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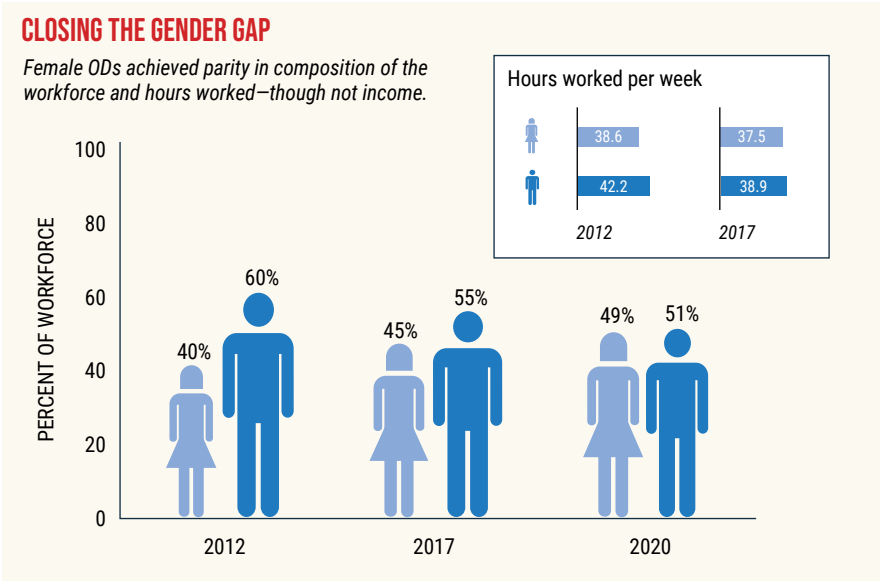

Women have made steady gains in the makeup of the optometric workforce and are on track to become the majority of practicing ODs in short order—if they haven't already.

In 2017, 77% of women and 78% of men reported they would prefer to have the same or fewer patient care hours. However, there was an increase in the number of women who desired to decrease hours, from 10% in 2012 to 25% in 2017.

Also of note in gender trends: women tended to be employed rather than self-employed and earned less as a whole.

Women Still Lag in Pay

Overall, a slight majority of optometrists reported income between $100,000 and $199,999, with more female optometrists than male making less than $100,000. Perhaps not surprisingly, those ODs who made more than $200,000 were most likely to be self-employed (23%).

One reason for the apparent salary differences between genders may simply be that male responders tended to be older, more experienced and owned their practices, Dr. Heath says.

Beyond optometry, some recent studies affirm the pay gap still exists between genders across various medical disciplines. One investigation that evaluated salaries among physicians with faculty appointments at 24 US public medical schools found an absolute annual pay gap of $51,315 between men and women.[4] After adjusting for factors such as age, years in rank and specialty, the annual disparity was $19,878.[4]

Another research paper, published in 2020 in the *New England Journal of Medicine*, found female primary care providers (PCPs) generated nearly 11% less annual visit revenue than male doctors in the same practices, yet they spent more time with patients per visit, per day and per year.[5] The revenue gap was driven entirely by differences in visit volume, which were only in small part explained by the fewer days that female PCPs saw patients.[5]

Taken together, these results suggest that the differences in time spent with patients may be a contributor to the gender pay gap, with female physicians effectively generating 87% of what male physicians generate per hour of direct patient care, the authors said.[5]

"Honestly, I think the wage gap is still there because we are women," says Jennifer Dattolo, OD, FCOVD. "I think in most professions, men make more than women do for the exact same job."

Dr. Dattolo, who is a partner at her practice in Woodstock, GA, says she feels society devalues women to some degree, and this still lingers in the profession. "I do think men and women are working close to the same hours per week and seeing the same number of patients. And hopefully the pay gap between men and women will close and become equal in the very near future," she says.

Since the majority of graduating doctors from optometry school are currently female and many new grads carry a significant debt load, Dr. Miller speculates an employed, salaried position might be a more attractive option than pursuing an ownership opportunity, which may factor into the pay discrepancy between genders.

"You govern your own future," Dr. Miller says. "There are women who want to own a practice and others who want flexibility."

Despite the pay gap, both male and female ODs also appeared to be equally happy with their jobs. Career options/professional growth satisfaction ranked in at 65% for both male and female ODs, in the current survey.

Additional "Capacity" Questioned

Respondents were also asked to assess their ability to take on more patients in their practice, which was defined as "additional capacity."

The Lewin study reported that optometrists could, on average, see about 20 more patients a week, or 32% more patients annually. In 2014, this finding fed controversy about whether there was a need for more optometrists and more optometry schools, and essentially split the profession into two camps over the issue, Dr. Heath says.

Dr. Chou remembers the impact of the Lewin study and how the results instigated a heated discussion in the profession on whether there were too many optometry schools minting too many ODs.

This latest workforce survey's growth findings of 1.4% annually (0.6% to 0.7% greater than the US population) suggests an adequate workforce supply, with a small surplus created each year, Dr. Chou suggests.

"This is contrary to the doom-and-gloom scenario of too many optometry schools and too many optometry graduates," Dr. Chou adds. However, these workforce surveys did not consider

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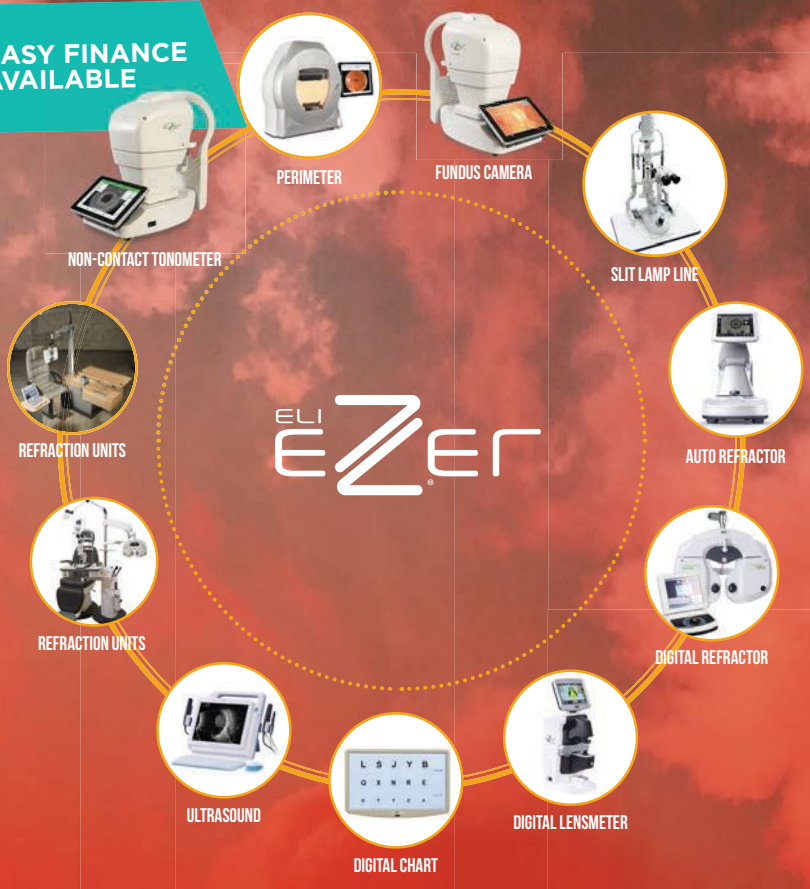

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technological advances that may reduce consumer demand of traditional optometric services, Dr. Chou suggests.

"For example, there is a proliferation of self-administered online vision tests with remote eyeglass and contact lens prescription renewal," Dr. Chou notes. "If optometric refractive services erode without the corresponding expansion into other services like medical eye care, our profession can still find itself with far too many optometrists and not enough patients seeking care, irrespective of what the latest workforce survey says."

The Lewin survey's question on this topic was effectively appointment book-based, reflecting the sum of the empty slots and the number of no-shows in an OD's appointment book, Dr. Heath explains. "Since some optometrists may add more appointments to compensate for no-shows—and there will always be no-shows—the 32% figure was an inflated estimate," he adds.

There was also no exploration as to whether the responders *wanted* to see more patients. As such, the current study asked the question differently, by querying ODs on how many more patient exams they could accommodate without changing current schedules or staffing. To further validate this, the study then queried participants about waiting times for appointments and whether ODs actually wanted to see more patients.

Overall, the mean number of additional patients that reportedly could be seen by an individual provider was pegged at 9.67 per week. This number conceptually represents the additional capacity that an optometrist could see as opposed to would likely see, researchers noted. The new data indicate a likely range of additional patient capacity of 2.29 to 2.57 patients per week (5.05 to 5.65 million annually profession-wide).

The study also considered if doctors who reported they could accommodate additional patients could do so without changing their practice patterns. A total of 65% of ODs reported a wait of at least two days for a patient to get an appointment for a comprehensive eye examination, whereas 7% reported a waiting time of two to three weeks and 5% indicated a wait time of more than three weeks.

In the 2017 data, nearly two-thirds of ODs reported that their current wait times for appointments were the same as the previous year. Only 13% of practicing optometrists reported a decrease in appointment wait times, whereas 23% reported an increase.

A final question assessing the ability of the optometrist to see additional patients asked respondents to indicate if they were unable to accommodate all patients requesting appointments, provide care to all who requested appointments but were overworked, provide care to all who requested appointments (not overworked) or could accommodate more patient appointments.

About 8% indicated they were unable to accommodate all patients asking for an appointment, whereas 12% indicated they did provide services to all patients but were overworked. Notably, only one-third (33%) of respondents selected could accommodate more patient appointments.

Most optometrists within each group indicated they wanted their patient care hours and their non–patient care hours to remain unchanged (49% and 57%, respectively). Less than a third of optometrists in each group indicated a desire to increase patient care hours.

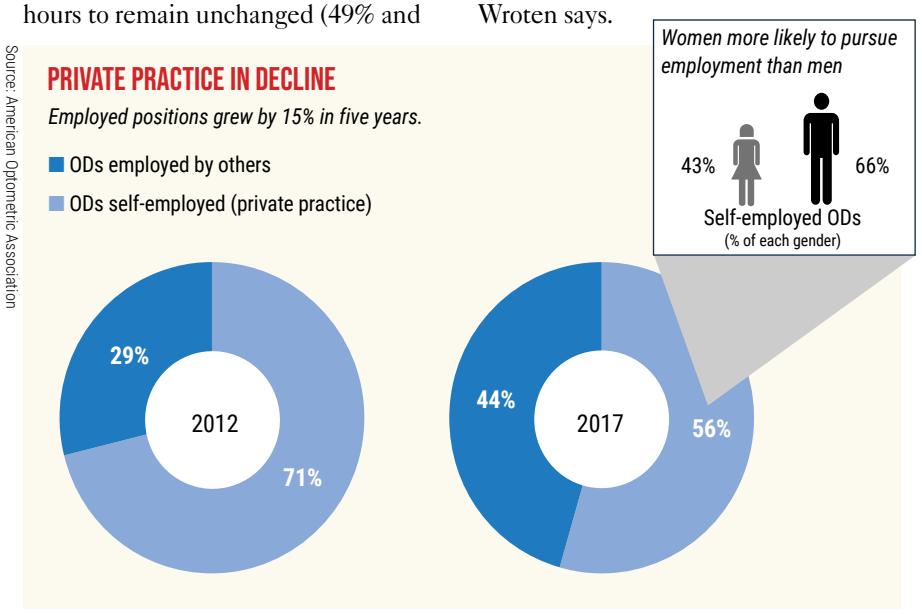
"I do think there are still opportunities to provide more medical eye care and for many of us to better use our staffs in the provision of care," Dr. Wroten suggests.

Another area to consider: new technology may enhance efficiency, but in turn, could also erode some traditional modes of practice.

"However, I don't get the impression that the demand for optometry services far exceeds the supply of optometrists, except in more rural areas where it's becoming harder and harder to recruit new graduates to practice," Dr. Wroten explains.

Additionally, the national applicant pool for optometry school has remained relatively flat for several years now, while the number of optometry school positions available has grown as some existing institutions increase class sizes and newer schools open.

"A host of factors, including the impacts of telemedicine, emerging technologies and the average retirement age for doctors of optometry, coupled with the quality and quantity of optometry school graduates, will ultimately determine whether demand exceeds supply or vice versa," Dr. Wroten says.



Experts attribute the shift toward employed positions over private practice ownership to many factors, including the high cost of student loan debt for new grads and the appeal of private equity.

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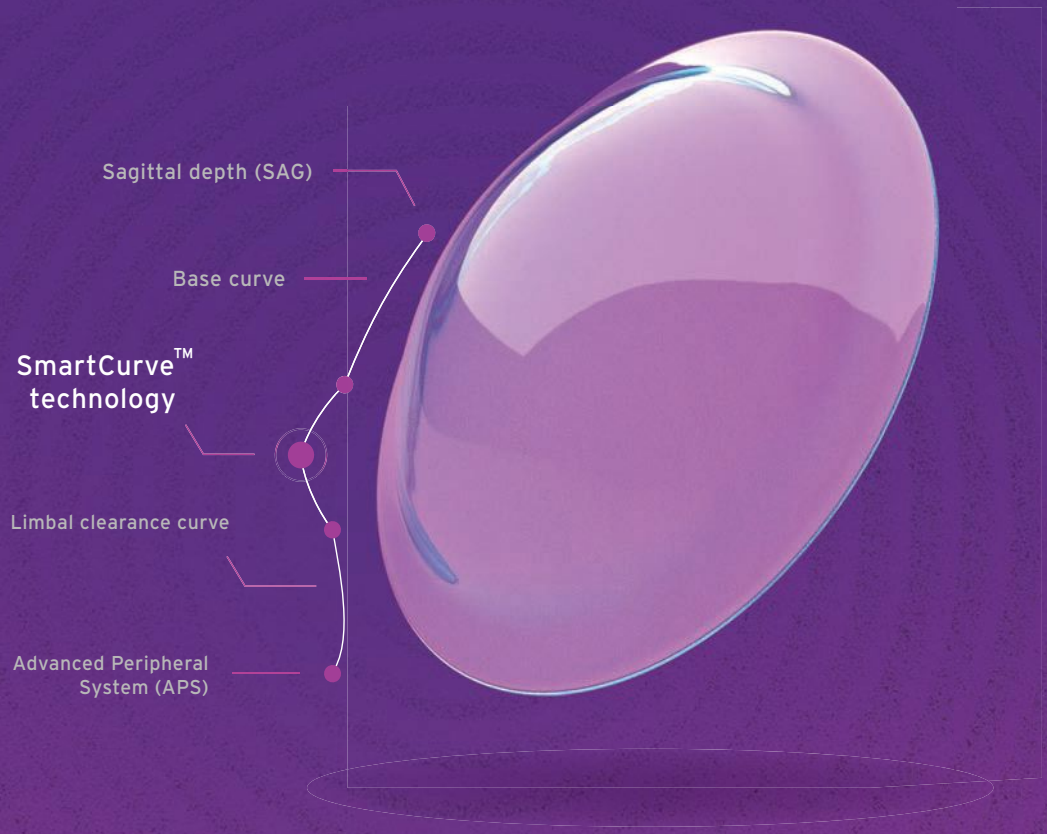

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Pandemic Disrupts Optometric Workforce Status Quo

Since the 2017 workforce study was based on doctor feedback three years prior to the pandemic, a question remains on how the new safety and social distancing requirements imposed since the onset of COVID-19 have curtailed patient visits and, in turn, impacted physician productivity.

Dr. Heath believes the most immediate impact on the workforce will be on the rate of retirement, sine he says it's likely that a number of optometrists who were considering ending their active time in the profession over the next few years before the pandemic hit may have accelerated their decision.

Those trends will be part of the AOA's master data file and are worth examining, Dr. Heath says. "If there is a significant increase in retirements, it would tighten the labor market for optometrists, much as it has in other health professions."

He adds, "It's just conjecture on my part, but until safety measures, particularly social distancing, are relaxed by various state departments of health, I'm not sure practices will see a full return in the number of patients to pre-pandemic levels. Practices are limited physically, and there is only so much you can do by changing patient flow."

Also, staff delegation, telemedicine and new technologies can all impact efficiency and patient capacity regardless of the pandemic, he suggests.

MINORITIES STILL UNDERREPRESENTED

Ethnicity	
Hispanic or Latino	4%
Not Hispanic or Latino	92%
Data missing	4%
Race	
American Indian	0%
Asian	12%
Black/African-American	0%
Hawaiian/Pacific Islander	1%
White	81%
Other	0%
Data missing	6%

Source: American Optometric Association

A Trend Away from Private Practice

The study also examined the current levels of employed vs. self-employed optometrists, broadly defining those who earn a salary as "employed" and not limiting the designation to those who work in a commercial practice. For example, employed optometrists could work in a university setting, hospital, community health center or in private optometry or ophthalmology practice, Dr. Heath says.

The most significant finding in this area was a substantial shift in the percentage of employed optometrists, which increased from 29% in 2012 to 44% in 2017. There was, of course, a commensurate decrease in the percentage of self-employed optometrists from 71% to 56% in 2017, Dr. Heath says.

Dr. Chou finds it concerning—but also not surprising—that there is a trend toward employed vs. self-employed work settings.

"With private equity activity ramping up significantly since 2017 when the latest survey was collected, we can expect an even greater number of ODs finding themselves in employed situations as time moves forward," Dr. Chou says. "While there are multiple interpretations of what underlies the trend toward employment, my take is that it is increasingly difficult to own a practice, which leads to more aspiring for employment."

Both studies generally found employed and self-employed optometrists worked the same number of hours and were equally productive and satisfied in their roles. However, the employed optometrists saw significantly more patients per week on average than those who were self-employed (58 vs. 54).

The trend toward an increasing number of employed optometrists is also mirroring what is occurring in other health care specialties, as large health care systems expand and private equity acquires more practices and employs their previous owners, Dr. Wroten suggests.

"It also seems like a higher percentage of recent optometry school graduates are content to seek an employment relationship vs. purchasing an existing practice or starting one from scratch, some of which may be driven by escalating student debt," he says.

Opportunities for ODs to work for industry have also increased, Dr. Wroten explains.

Much of the debate about whether there is a surplus or deficit in the capabilities of optometrists to meet the needs of the American public is purely speculative and can be influenced by many factors, Dr. Heath suggests.

"There is often an assumption that at any given moment, the demand for eye care is equal to the supply and that future projections, if not equal to population growth, will result in an over- or under-supply," Dr. Heath explains.

To accurately project, it would be necessary to know if there is currently an unmet need (demand), in addition to what is being provided.

"This study didn't examine the demand for optometric services, so we need to be cautious on this issue," Dr. Heath adds.

Next Steps

Dr. Heath says he believes it's critically important for the profession to continuously monitor the optometric workforce for not only overall supply and demographic shifts but also for trends that impact national, state and local health care policy. The lack of publications in peer-reviewed journals addressing these issues is startling and puts the profession at a significant disadvantage in its ability to advocate for and affect public health policy, he adds. ■

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THE DRIVE TO DIVERSIFY

Efforts are now underway to make optometry more inclusive, and Ruth Shoge, OD, MPH, wants to be sure the profession doesn't take its foot off the gas.

Although she's encouraged by the recent momentum in this area over the past year—including the expanded number of minority students applying to optometry schools and cultural competency courses added to some curricula—Dr. Shoge, who will join Berkeley Optometry School as its inaugural Director of Diversity, Equity, Inclusion and Belonging later this month, says there's still plenty of work that needs to be done and worries that people will lose interest in efforts to invest in a diverse and culturally competent optometric workforce.

"We have to keep going, and accountability will help with that."

Ten years into her optometric career, Dr. Shoge pursued her Master in Public Health degree with a concentration in Social and Behavioral Science at Temple University in order to better understand the ways in which she could best serve her community, a decision that reaffirmed her dedication to the pursuit of expanding diversity and cultural competence in the profession and addressing health disparities among underserved populations. In fact, her master's thesis at Temple was "Social Experiences of Underrepresented Minority Optometry Students."

One barrier to minority enrollment is that these individuals often don't see themselves reflected in the profession, she explains. As part of her master's project, Dr. Shoge surveyed students and found most had previous exposure to optometry in their youth, including eye exams.

"It makes a big difference if you actually see someone in that role, and it's even more powerful if you see someone in that role who looks like you," she says.

Not seeing other minority students enrolled in optometry schools also poses a barrier, in addition to the financial burden of higher education, she adds.

Ruth Shoge, OD

Minority Applications Rising

Dr. Shoge, who will be leaving her current role as associate professor at Salus University, points to some promising news she just learned as chair of ASCO's Diversity and Cultural Competency Committee: minority applications to optometry schools are on the rise.

While there has been a 4.7% overall increase in applicants this year, almost 6%

of the applicant pool is Black or African-American, a number that has traditionally hovered between 4% and 5%. Additionally, there has been an uptick in the number of Hispanic and Latino applicants: 12.6% compared to the usual 9% to 11%.

"It may seem like a small number, but it's a big difference, and it means our efforts are working," she says.

The Profession Reacts

Some of these successful efforts include ASCO's "Optometry Gives Me Life" initiative that aims to bolster interest in optometry schools, in addition to the continued minority recruitment and health care disparity initiatives of the National Optometric Association. Dr. Shoge cites another leader in this area: Black Eyecare Perspective, an organization created to foster lifelong relationships between those who identify as Black and the eyecare industry, and to create more black representation in all levels of the profession.

"Black Eyecare Perspective is doing a tremendous job at creating buzz and mentorships with Black students looking to optometry as a career," Dr. Shoge says.

Corporations have also recently stepped up with donations to help support some of these initiatives, including one she oversees: Salus's Summer Enrichment Program, which aims to improve the matriculation, attrition and graduation rates of underrepresented minority applicants through a free, five-week program. It had been on hiatus for several years due to lack of funding but has been reinstated in response to growing demand for attention to the diversification effort.

Still other institutions have added cultural competency and anti-racism courses to their curricula.

"One branch of our purpose is to increase the number of underrepresented minorities in optometry school, but the other part is to ensure the overall cultural competency for the entire student body. Having these courses as resources helps everyone understand that it's an 'all of us' issue. All of us need to be involved," Dr. Shoge says.

Dr. Shoge was recently on a panel hosted by SUNY College of Optometry, "Race in Optometry: Accountability—One Year Later," which was held on the same night that Juneteenth was declared a Federal holiday by President Biden and was a follow-up to three previous seminars on race in the profession. The series put a spotlight on the experiences of Black optometry students, residents, faculty and practitioners. The intent of the series was to foster a dialog that would lead to needed changes in optometric education and in the eyecare industry.

Expanding Care to the Underserved

The broadest goal in diversity and inclusion is to make sure individuals who live in underserved communities receive care, Dr. Shoge says.

"I like to think about the purposes of our initiatives as short-term, intermediate-term and long-term."

The short-term goal is to increase the matriculation and graduation of underrepresented minority students, and particularly Black students. Intermediate goals include nurturing and encouraging these students to pursue leadership roles in their profession, whether in an academic, corporate or political setting.

"Our long-term goals should include doing everything we can to help reduce health disparities and improve health outcomes in the communities we serve, particularly in places where we know there are poorer outcomes and more exaggerated health disparities."

She adds, "One thing we know that helps combat this is concordance, which simply means the physician and patient share some sort of cultural, social or linguistic identity."

Research has shown concordance helps improve the likelihood that a patient will seek care in the first place, trust their doctor, adhere to treatment protocols and follow up—and maybe even bring along their family, friends or neighbors to seek care," Dr. Shoge adds.

Through collective, ongoing efforts, Dr. Shoge hopes the profession will continue to make strides in diversifying its ranks—in classrooms today and exam rooms tomorrow.

CLEAR UP CATARACT CONCERNS

Properly understanding and addressing patient questions could make a world of difference in their visual outcomes.

BY JULIE TYLER, OD
FULLERTON, CA

Cataracts are the most common cause of visual acuity (VA) loss in the world.[1] Most optometrists encounter this condition on a regular basis. Even though patients generally have heard the term "cataract"—derived from the Latin word *cataractes*, which means "waterfall" and relates to the foamy, hazy appearance of visually significant lens opacities—patients will still have questions about what's physically happening in their eyes to alter their vision, why these changes are occurring and what to do next.

This article covers five commonly encountered questions cataract patients ask and some appropriate corresponding responses. The way in which we answer these questions and provide guidance can help reduce anxiety and go a long way toward helping patients achieve more successful surgical results.

1. "Aren't I too young for cataracts?"

In one study that reviewed geographical variation in the rate and timing of cataract surgery between US communities, the overall median age for first cataract surgery was 67.7 years, with almost a 20-year variation between regions; median age ranged from 60 to 80.[2] While cataracts are most commonly the result of expected age-related changes within the crystalline lens, a variety of conditions may lead to cataract development at an earlier age than anticipated. The study recognized a large variety of contributing factors, some of which are well known, such as comorbidity of diabetes, chronic exposure to UV light and use of corticosteroids. Other associations are less familiar and vary by region, such as exposure to ambient air pollution.[2]

A different study, conducted in Korea, revealed that individuals, especially females 65 years or older, who were exposed to greater amounts of air pollutants, specifically airborne particulate matter with a diameter of 10μm or less and nitrous dioxide, had a significantly increased hazard ratio and incidence for cataract development.[3] Another example of potential environmental effects of toxins/pollutants on cataract development was illustrated by research that found Lake Charles, LA—a major center for petrochemical refining that potentially allows for chronic exposure to naphthalene and other pollutants—had the country's highest cataract surgery rate.[3,4]

When a patient believes they are not the "right" age for cataracts but falls into our expected stage for development, reassurance and patient education are recommended. However, when a patient is actually younger than the expected age for lens opacities, it is important to identify potential risk factors or comorbidities by carefully reviewing their history. In addition to known systemic associations, unknown or undertreated conditions may also result in opacification. Reviewing medications closely is valuable, as there are several classes, including but not limited to corticosteroids, phenothiazine and amiodarone, that may disrupt or deposit within the lens. When reviewing family history, a strong presence of cataracts that appears at a younger than anticipated age may also suggest a possible genetic or systemic comorbidity.

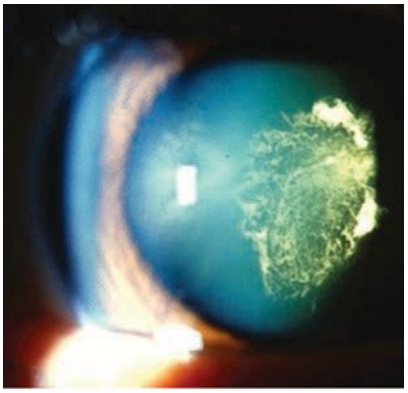

Fig. 1. PSC is more common with diabetes.

Diabetes mellitus (DM) is a common systemic condition that may present with a variety of crystalline lens changes. Patients with DM, especially those who have poorly controlled blood sugar, are more likely to develop cataracts at an earlier age due to accelerated stress on the lens induced by

About the author **Dr. Tyler** is an associate professor at Southern California College of Optometry at Marshall B. Ketchum University. She has no financial interests to disclose.

fluctuations in the sorbitol pathway and chronic lens adjustments in response to variable blood sugar levels. All diabetic patients may present with traditional age-related varieties of cataracts but are more likely to have posterior subcapsular cataracts (PSC) and "sugar cataracts" that are cortical in nature (*Figure 1*). In addition to earlier development, cataracts in patients with DM tend to progress more rapidly.

Wilson's disease is another metabolic condition that results in lens changes. Individuals with this uncommon autosomal recessive condition may present in the second to fourth decade of life with a rare "sunflower cataract" during active or uncontrolled disease states.[5,6] In these patients, the lens may be deposited with copper centrally, which presents as a green-to-brown lens defect surrounded by short stellate processes (small "petals"). However, one study noted that sunflower cataracts are rare in Wilson's and because there is not a true persistent disruption of the lens and VA is generally not permanently or significantly affected, the term "cataract" might not be the most accurate description for the finding.[5,6] While the lens isn't permanently changed, recognizing this finding in a young patient could save them from permanent complications.

Myotonic dystrophy, a relatively rare systemic condition, is also associated with lens changes. There are several varieties, based in part on chromosomal anomalies that result in type 1 (chromosome 19, CTG repeat) and type 2 (chromosome 3, CCTG repeat), nearly all of which present with early-onset (*i.e.*, younger than 40) characteristic cataracts described as iridescent, cortical "crystals" that are central and polychromatic.[7] These pathognomonic lens opacities are known as "Christmas tree cataracts." Type 2 myotonic dystrophy has also been reported to present with PSC at a relative early age of onset.[8]

Additional systemic diseases associated with lens changes include but are not limited to Fabry's disease, atopic dermatitis and neurofibromatosis type 2. Identifying lens anomalies, as well as recognizing associated systemic conditions, may give patients a better understanding of what is happening in their eye as well as their body.

2. "Is there something I did that made me develop cataracts?"

Ocular history may also be crucial in determining why a patient has visually significant lens changes. The term "complicated cataract" is used to describe opacities that form secondary to a concurrent or chronic ocular comorbidity. As the crystalline lens is avascular and relies on the surrounding ocular fluids to provide support and nutrients for continued, normal lens development, when situations occur where the surrounding material is concurrently exposed to inflammatory cell mediators, lens quality may be compromised. Examples of conditions that are associated with complicated cataracts are most commonly pathologies that cause chronic eye inflammation, such as uveitis associated with juvenile idiopathic arthritis (JIA) or sarcoid disease, retinitis pigmentosa or ocular tumors.

In addition to chronic inflammatory mediators in the eye, acute traumatic events from a variety of sources (*e.g.*, heat, electric shock, irradiation, perforating eye injuries, concussive forces) may appear with a range of cataract presentations. In mechanical trauma, examination may reveal a "rosette" type of cataract with petaloid opacifications radiating from the center of the lens (*Figure 2*). Rosette lens opacities may be seen in patients who have encountered a blunt trauma to the eye with coup (potentially resulting in a concurrent Vossius ring)-contrecoup injuries.[9] The contrecoup injury creates shock waves along a line of traumatic impact, which may generate posterior cortical opacification in the hallmark flower/star pattern.[10,11] Patients with visible lens findings after trauma need to be further examined diligently for evidence of other traumatic complications such as disruption of lens zonules, which may occur from axial elongation associated with an injury and significantly complicate future cataract surgery.

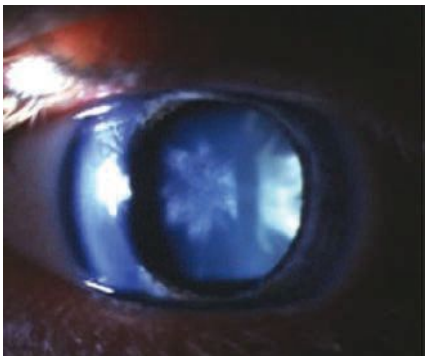

Fig. 2. Rosette lens opacity post-trauma may permanently affect best-corrected VA and is an indication for assessment of other traumatic complications in the eye.

3. "How did I develop cataracts without any warning signs?"

Prior to decreased VA or symptoms of glare that may result from cataracts, patients might present with early, "soft" signs of changes occurring within the lens. When clinical findings become evident, even prior to changes in best-corrected VA, we should consider using these initial findings as an opportunity to educate the patient on the normal process of lens changes with age and discuss ways to slow progression of visually significant cataract development.

Throughout life, the lens increases in size as both the nucleus and cortex continuously grow. Additionally, the lens may develop an accumulation of urochrome pigment that results in notable color variations on exam, ranging from yellow to brownish red (*Figure 3*). The increased density of the crystalline lens fibers within the capsule contributes to a change in the index of refraction of the lens and a common myopic shift, often accompanied by a change in astigmatism.

In 2015, a study helped clarify the types of refractive differences that occur by reporting quantified results of 276 subjects after assessing the levels of refractive changes compared with cataract type and severity when classified using the Lens Opacities Classification System III as a grading tool.[12,13] The study confirmed that cataracts produced changes in refractive error that were dependent on both the severity and type of opacity, revealing that with

Fig. 3. Crystalline lens yellowing is due to an accumulation of urochrome pigment in the lens. Anterior cortical cataract and nuclear sclerosis are pictured here.

higher severity of nuclear sclerosis and PSC, greater myopic shifts and axis rotational changes occurred, and that while cortical cataracts did not produce a myopic shift, there were astigmatic power changes.[12]

When lens-related refractive changes are identified, the practitioner can educate patients that the shift in prescription might be a "pre-cataract" change. Also, when patients report a need for increased illumination to complete visual tasks (especially at near), this complaint may also serve as an opportunity to make patients aware of early lens alterations. Educating patients about the association between lens changes and more subtle visual refractive symptoms when they occur may prepare them for future vision issues and motivate them to use preventative measures to delay cataract development and progression.

4. "How do I prevent my cataracts from worsening?"

There are a large variety of agents we are exposed to throughout our life—internally and externally—that may contribute to accelerated opacification. Therefore, if we limit exposure to known causative agents, we may be able to decrease the rate at which cataracts become visually significant. Perhaps the most common recommendation to assist with deterrence of lens progression is the use of UV protection in glasses and contact lenses. UV radiation reaches the earth's surface in the forms of UVB (280nm to 315nm, 1%) and UVA (315nm to 400nm, 99%), penetrating and absorbing into our skin and eyes to result in degenerative changes.

A study analyzing Medicare data for cataract surgery found that after controlling for age, sex, race, income, access to eye care, price of surgery and local practice costs, the strongest predictor of surgery was the person's latitude of residence.[14] Latitude has a direct correlation with the UVB content of sunlight, and data suggests the probability of cataract surgery in the United States increases by 3% for every 1° decrease (*i.e.*, more southerly) in latitude.[14] Another study concluded, "Chronic UV effects on the […] lens are cumulative, so effective UV protection of the eyes is important for all age groups and should be used systematically."[15]

Encouraging patients to use UV coating on their spectacle lenses, whether tinted or clear, is something ODs can do in the exam room, as well as in the optical. When educating patients on UV, there is value in explaining that UV protection is not necessarily a tint, but rather something that can be added to both clear and colored lenses to be worn whether a day is foggy or clear. E-SPF (Eye Sun Protection Factor) is a relatively new system with a goal of standardizing and quantifying UV protection for lenses that takes into account UV transmission, back reflectance and protection of structures of the eye and adnexal skin tissues.[15]

Besides encouraging patients to be proactive by using wide-brimmed hats as physical barriers to UV radiation and spectacles or contact lenses that include UV filters, educating those who take steroids on the appropriate use of their medication may further assist in slowing cataract progression. Any chronic use of this drug class may contribute to earlier cataract formation. The well-established association between steroid use and cataracts was further reinforced by a retrospective review of children with JIA who saw their risk of cataracts increase as the number of drops of topical steroids used daily increased.[16] The rate dramatically increased from dosing three to four times daily, and the association between steroid use and cataract formation was independent of uveitis activity, severity, duration and relapse.[16] Prescribing steroids judiciously and educating patients on associated risks of cataract formation (and intraocular pressure increase) with their steroid-containing medications may assist in slowing visually significant changes.

Smoking is also known to cause morphological and functional changes to the lens. Tobacco modifies ocular capillary perfusion and generates free radicals, thereby decreasing the levels of antioxidants available in the blood, aqueous humor and ocular tissue.[17] A 2015 report evaluated the relationship between smoking and cataracts by reviewing 27 studies using causality criteria.[18] The review documented a strong association between the two, particularly supporting a causality with nuclear sclerosis.[18] In more recent years, traditional cigarette smoking has been accompanied and/or replaced by alternatives such as electronic cigarettes. While there is significantly less literature in regard to electronic cigarettes and cataract development, the association between cataract formation and tobacco use with electronic cigarettes continues to suggest an increased risk of associated lens changes.[19]

5. "What's the chance of complications during surgery, and what increases the risk?"

In most cases, optometrists are able to reassure patients that cataract extraction is a safe procedure associated with improvements in daily living, including decreased motor vehicle accidents and falls.[20,21] In one study comparing traditional microscope cataract extraction with heads-up cataract surgery (3D), overall complication rates were less than 1% and nearly identical between the traditional group (0.77%) and the 3D group (0.72%).[22]

Initial steps to assess, manage and reduce potential risks associated with cataract surgery start with the patient's history. A patient with a history of significant ocular trauma has an increased risk of weakened lens zonules, as well as potential lens subluxation. Additionally, if a patient has a known ocular history of pseudoexfoliation (PXE)—or

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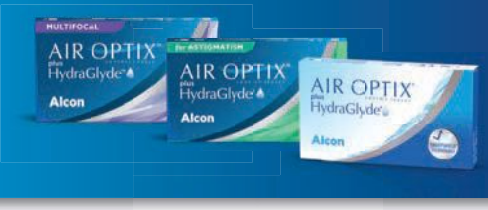

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a history that reveals ocular hypertension and/or glaucoma—perform a careful anterior segment exam to assess for masquerading PXE (*Figure 4*).

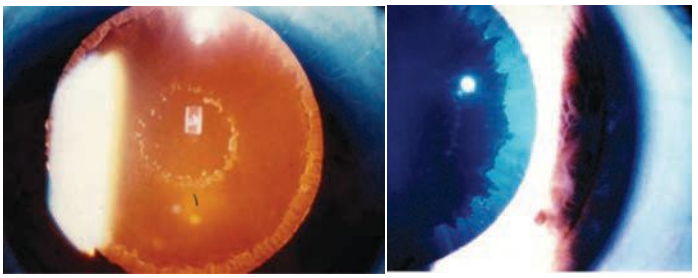

Fig. 4. PXE material on the lens surface can be seen on retroillumination (A) and with white light (B).

While the exact etiology for PXE is not well understood, multiple factors play a role in the pathogenesis of the disease and how it can complicate cataract surgical outcomes.[23] While PXE material may be seen on the anterior lens surface after being deposited from iris vessels with weakened basement membranes, it also deposits on the crystalline lens zonules and weakens them. The zonular compromise may lead to lens subluxation and complicate traditional cataract surgery. Additionally, there are more anterior segment implications associated with PXE material, including iris rigidity and poor mydriasis, which may further complicate cataract extraction and prevent a good overall outcome.

Another risk factor for complications during cataract extraction, which may be identified with a careful history, is the current or past use of tamsulosin and other alpha 1-adrenoreceptors. These medications in particular have been associated with intraoperative floppy iris syndrome (IFIS), a potentially challenging event for even the most prepared surgeons in which iris dilation is compromised and the iris tone is not maintained due to structural changes induced from use of the medication. Since the original association between tamsulosin and IFIS was made, a large number of additional medications and correlated risk factors have been revealed, including gender, age, hypertension and hypertension drugs, benzodiazepines, antipsychotics and finasteride, among others.[24]

Besides careful histories reviewing ocular status and medications, recognizing patients with posterior polar cataracts (PPC) is also important to reduce the chance of surgical complications. PPC increases the risk for posterior capsular rupture due to preexisting posterior capsular dehiscence. Careful slit lamp exam of the lens of patients with PPC will reveal the presence of a dense, white central opacity on the posterior capsule with multiple concentric layers (bull's eye-type appearance). These cataracts, which present early in life, may be stationary or progressive, and patients will generally complain of glare. If a patient who needs cataract surgery appears to have a PPC based on general exam findings, further evaluation with anterior segment OCT (AS-OCT) with an emphasis on the posterior lens capsule may provide invaluable insights. A 2018 study looking at 64 eyes of 62 patients that reviewed the effectiveness of AS-OCT for detecting posterior capsule dehiscence found sensitivity and specificity values of 100% and 95%, respectively.[25]

When PPC is identified prior to cataract extraction, surgeons can implement different techniques to enhance the safety of surgery and reduce the risk for posterior capsular rupture. While there is an inherent degree of risk with any surgical intervention, when potential complications are recognized and precautions are taken, the patient and surgeon can more appropriately set themselves up for an optimal outcome.

Takeaways

In our role as eye care practitioners, we can best serve our patients by thoroughly answering their questions and understanding their concerns. Interestingly, in both of the studies included that reviewed geographical variations in cataract surgery among US communities, when considering the number of ophthalmologists or optometrists in an area, cataract surgery rates were not associated with the number of ophthalmologists but rather with the number of optometrists per 100,000 residents.[2,4] Thus, it appears that by better addressing patients' questions and concerns as part of their cataract care team, we may be able to better assist them in solving their visual shortcomings. ■

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A RESULTS-ORIENTED NEURO WORK-UP FOR GENERALISTS

Be thorough to identify any underlying causes of optic disc edema and ensure those issues are managed appropriately at the ED.

BY MICHAEL TROTTINI, OD,[1] AND CANDICE TOLUD, OD[2]
[1]MONROE, NJ; [2]COLUMBUS, NJ

Seeing optic disc edema can be quite alarming. The causes are more serious in nature, and vision loss associated with optic disc edema can sometimes be permanent—not to mention that some causes can be life-threatening. If possible, the immediate reaction most times will be attempting a referral to neuro-ophthalmology; however, it can be very difficult to find a specialist who accepts same-day referrals. Some are booked for weeks or months ahead of time. Many optometrists don't feel comfortable working up these types of cases, but some are true emergencies and need to be sent to the hospital anyway.

This article will focus on identifying various presentations of unilateral and bilateral disc edema, knowing the most common causes and making hospital ED referrals with good doctor-to-doctor communication to ensure the appropriate patient work-up.

Common Causes

In general, the more common causes of disc edema that present to our offices are either non-arteritic ischemic optic neuropathy (NAION), arteritic ischemic optic neuropathy (AION) or papilledema.

NAION. The typical presentation is sudden, painless vision loss in one eye and usually noticed upon awakening. Patients are typically 50 years of age or older. NAION affects males more than females and those with preexisting microvascular disease. However, it can still occur in those younger than 50 and even in patients without vasculopathic risk factors. Visual acuity can be variable but is not significantly affected, with 49% of NAION cases presenting with 20/30 vision.[1] Visual field defects are almost always either altitudinal or arcuate type deficits.

The disc edema associated with NAION can be either sectoral or diffuse and generally has a hyperemic appearance to it.[1] The fellow optic nerve will almost always have a "disc at risk" appearance with a cup-to-disc ratio of 0.3 or less.[1]

AION. This by far is much more concerning than NAION and will oftentimes have a much different presentation, although there can be some overlap with signs and symptoms. It is critical, however, to try and differentiate these two disorders clinically. Patients will typically be 50 years of age or older; in our experience, we typically see this in those between their 60s and 80s. Females are affected more than males.

Systemic symptoms will generally be present but not 100% of the time, with potential patient complaints of headache, scalp tenderness, jaw claudication, fatigue, weakness and/or myalgias. Patients may have had episodes of amaurosis fugax prior to vision loss. The loss will generally be much more severe when compared with NAION, usually presenting with worse than 20/400 vision.

The disc edema seen in AION will generally have more of a pallid swelling appearance vs. the hyperemic appearance seen in NAION.[2] Additionally, cotton wool spots and or retinal artery occlusion can also be seen.[2]

About the authors

Dr. Trottini practices at Outlook Eyecare in Monroe and Princeton, NJ, as well as Robert Wood Johnson Hospital in New Brunswick NJ. **Dr. Tolud** practices at South Jersey Eye Physicians in Columbus, NJ. She also serves as vice president of the New Jersey chapter of the American Academy of Optometry. They have no financial interests to disclose.

Papilledema. By definition, papilledema is bilateral optic disc edema secondary to increased intracranial pressure (ICP).[3] Because of the various causes for the condition, it can be seen across all age groups.

If the elevated intracranial pressure exceeds the intraocular pressure, there will be an absence of the spontaneous venous pulse.[4] This discs will usually be hyperemic, have hemorrhages and Paton's lines can sometimes be seen (retinal folds within the peripapillary region).[4] Acuity can range anywhere from 20/20 to almost total vision loss. Sometimes, a patient won't present initially or will wait until very late into a disease process to the point where their vision is significantly impacted on first presentation. Visual field defects can have great variability as well.

Additionally, a variety of patient symptoms can be present depending on the cause of the papilledema. Visually, patients can describe reduced acuity, transient vision loss or transient visual obscurations. Diplopia may be present as cranial nerve VI palsies can be seen in association with papilledema but, depending on the etiology, other cranial nerves can be affected as well.

Generalized neurologic symptoms can also vary greatly and can include headache, dizziness, lightheadedness, nausea, vomiting, altered consciousness and fever.[5]

Diagnosis and Management

After describing the more common causes of disc edema and what distinguishes each one, let's discuss what to do when presented with someone who has disc edema. In addition to doing a complete dilated examination, visual fields and optic nerve photos are a must for baseline and future comparison.

The visual field can also be exceptionally helpful for diagnostic or possibly localization purposes. For example, patients with NAION will often have an altitudinal field defect. The next step then depends on whether the disc edema is unilateral vs. bilateral.

Bilateral. With bilateral disc edema, there is a good chance that it is secondary to increased intracranial pressure.

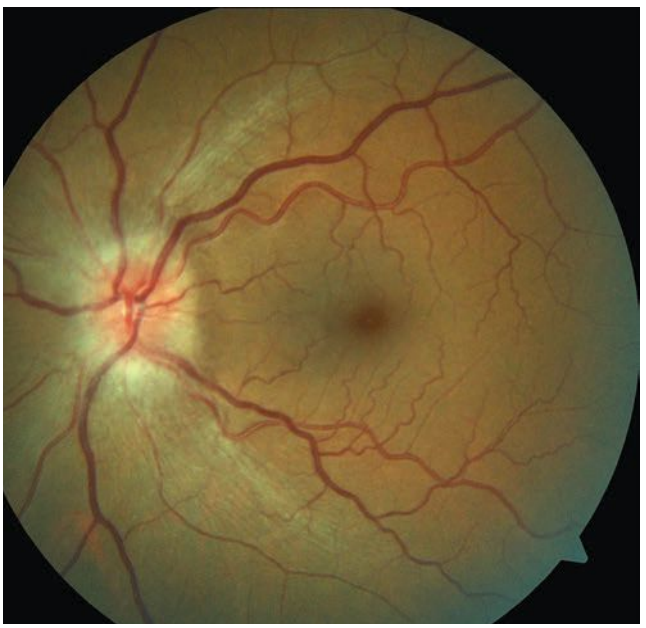
> **PTC itself generally isn't an emergency, but the diagnosis cannot be confirmed until imaging has been performed and deemed normal and lumbar puncture shows an opening pressure of >25cm H_2O.**

First, check the patient's blood pressure. Malignant hypertension can cause papilledema and it's quick and simple to measure. There will be other retinal findings of hypertensive retinopathy, and if the blood pressure is significantly elevated (>180/>110), then it's likely the answer to the disc edema. Malignant hypertension is a medical emergency, and these patients need to be sent to the emergency department (ED) for immediate work-up and treatment of their blood pressure.

Neuroimaging will sometimes be done in the general work-up of malignant hypertension, and we suggest recommending brain imaging be done in this scenario just to completely rule out any other cause of the disc edema. Once this diagnosis has been established, it's a matter of stabilizing the patient's blood pressure and serial monitoring of the disc edema, retinal findings and visual fields for improvement.

If the blood pressure is normal or at least not high enough to consider malignant hypertension as a cause, the second question to ask yourself is whether the patient and presentation seem consistent with pseudotumor cerebri (PTC). The characteristic presentation will be a younger female between the ages of 20 and 40 who is either overweight or taking medications associated with PTC. Such meds include: lithium,vitamin A and derivatives such as Accutane (isotretinoin, Roche) and tetracyclines.[6] The classic symptoms include headache, transient visual obscurations, tinnitus and nausea.[6]

In a situation where the patient's demographics, symptoms and clinical findings all point towards PTC, we recommend first performing a visual field in office. Although PTC in itself generally isn't an emergency, the diagnosis cannot be confirmed until imaging has been performed and deemed normal and lumbar puncture shows an opening pressure of greater than 25cm H_2O. Until then, it's still a patient with papilledema of unknown cause and should be treated as an emergency. Because of that uncertainty, we suggest sending these patients to the ED, recommending neuroimaging, such as a computerized tomography (CT) scan, magnetic resonance imaging (MRI) and magnetic resonance venography (MRV), to rule out any mass or



Note the hyperemic disc edema in this patient presenting with NAION.

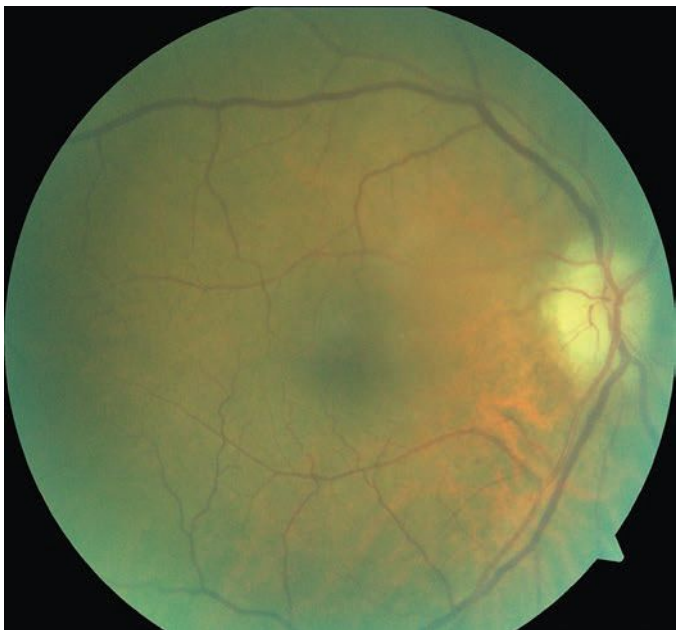
intracranial process and, if they come back normal, recommending a spinal tap with neurology consultation for PTC.

Once the diagnosis has been confirmed, neurology will generally manage reducing the intracranial pressure. Monitor the patient's fields, usually on a monthly basis. Assuming no significant or progressive field deficits are present, management will consist of weight loss, starting a diuretic such as acetazolamide and/or discontinuing any predisposing medications mentioned previously. However, in more severe cases with significant vision or field loss, treatment will require a more aggressive approach such as ventriculoperitoneal shunting or optic nerve decompression. These decisions are usually made by the neurologist after interpreting the extent of the vision and field loss and, if necessary, at that point they would request neuro-ophthalmology evaluation.

Lastly, if we see papilledema in this patient, there is no malignant hypertension and the patient doesn't fit the profile for PTC, then there is likely a very serious pathology causing the disc edema. These patients will always be sent to the ED and with much more concern for some intracranial process. Emergent neuroimaging and depending on the findings neurology and or neurosurgery consultations will generally be recommended.

One clinical pearl to keep in mind is that bilateral disc edema won't always be secondary to elevated intracranial pressure/papilledema. For example, a patient with bilateral AION from giant cell artheritis (GCA) may present with bilateral disc edema. Take into account all pertinent historical elements, findings and patient demographics to help determine if the likely cause of the bilateral disc edema is from elevated ICP or some other process. In general, brain and orbit imaging along with GCA testing will almost always identify the abnormality and if normal, other serologic testing or lumbar puncture is usually the next step.

Unilateral. In addition to NAION and AION, unilateral disc edema can be seen from tumors or anything that would cause optic nerve compression, optic nerve infiltration (*i.e.*, sarcoid, lymphoma) and optic neuritis (more common to be retrobulbar but can present with disc edema). Vision loss from a compressive or infiltrative process tends to be a bit slower in onset—usually days to weeks to months vs. NAION and AION, where the vision loss is sudden.



Note the pallid disc edema in this patient presenting with AION.

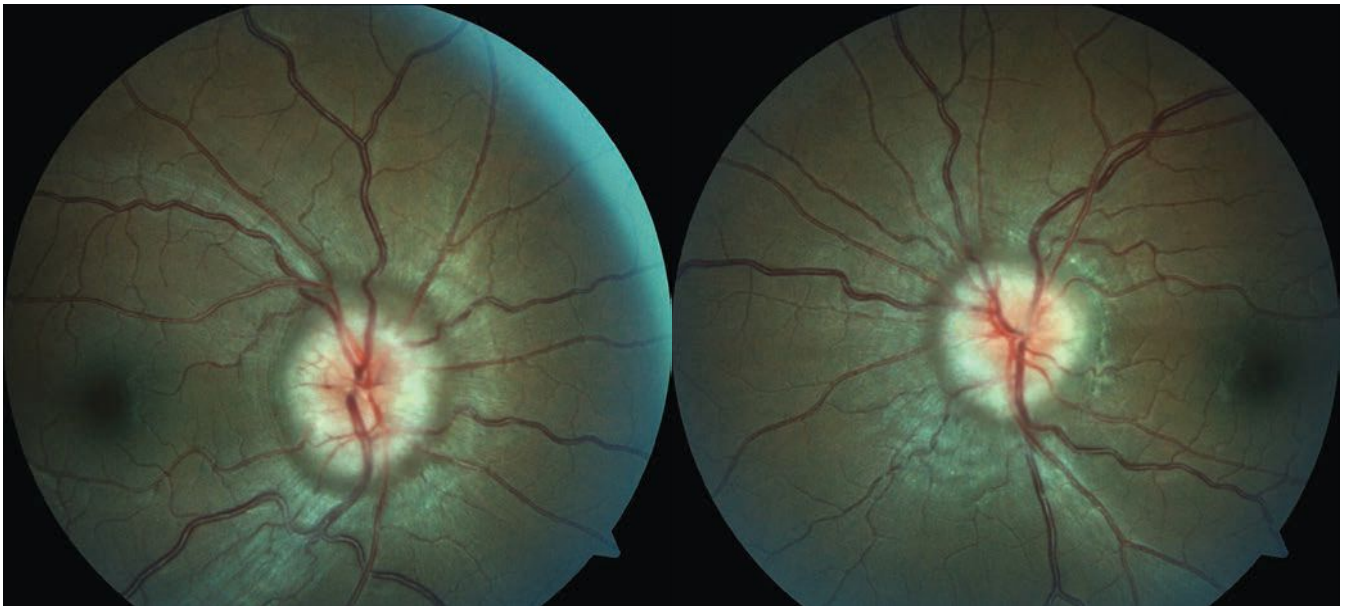
Optic neuritis can present with acute vision loss, but it's typically associated with pain, especially during eye movement. A major distinguishing feature between painful vision loss from optic neuritis vs. painful vision loss from AION is age. Optic neuritis is more typical for younger patients while AION is typical for older patients. Additionally, vein occlusions will commonly cause disc edema, but these are very easy to identify because of the retinal hemorrhages associated with the disc edema. Lastly, neuroretinitis will also cause disc edema, but usually in association with other findings such as macular exudates, uveitis and vascular sheathing.

Disc edema from a vein occlusion can almost always be easily spotted and these patients do not requite any neurological work-up. And the remaining causes can be frequently identified with neuroimaging, basic serology testing and, when indicated, lumbar puncture. So, with this in mind, if you are presented now with a case of unilateral disc edema and can't find an immediate referral source or if you're not comfortable with working up these patients yourself, ED referral is still a great option.

If an AION is suspected, evaluate the patient for GCA. Checking ESR, CRP and also looking at the patient's platelets would be the first step. Rheumatology can also be consulted to further evaluate for GCA.

In regard to patients with suspected NAION, there is debate on whether or not these patients need to be imaged initially. Some doctors feel that if the presentation is consistent with NAION, they can be followed initially without imaging and only recommend testing for GCA if the patients are older with any suspicion. There is no standard of care rule on this and we've seen optometrists, ophthalmologists and neuro-ophthalmologists on both sides of the fence. We personally feel it's in the patient's best interest, even when very confident that the diagnosis is NAION, to obtain at least an ESR and CRP as well as neuroimaging.

Even if you are presented with a case that points towards NAION but are not comfortable with following initially or managing yourself and can't find a timely outpatient referral, it is still acceptable to send even these patients to the ED. Emphasize with good communication that the patient has a likely NAION, but you would



In this patient with papilledema secondary to PTC, the diagnosis cannot be confirmed without normal MRI/MRV and after lumbar puncture has been performed.

suggest at least testing ESR, CRP and neuroimaging to rule out any other causes. Additionally, patients with NAION need to ensure their microvascular disease is under appropriate control. In cases of NAION where no obvious microvascular risk factors exist, such as diabetes, hypertension and sleep apnea, patients will often need hypercoaguable and autoimmune work-ups.

If the suspicion of disc edema points towards optic nerve compression, infiltration, inflammation or demylenation, the focus will be largely on neuroimaging and possibly lumbar puncture. These specific decisions don't need to be made by the optometrist, but communicating these suspicions and any guidance is important when sending these patients to the ED. For example, if optic neuritis is suspected, recommended that the patient eventually have a brain and orbit MRI and not just a CT scan, as the CT is usually not sensitive enough to detect those findings.

Also, when referring patients like this, a recommendation for neurology consultation should almost always be made. The on-staff neurologist will also help to guide the appropriate work-up if necessary.

Takeaways

Being on staff and working at a hospital, we see referrals such as these commonly. We've never seen an ED attending get upset over this type of referral as long as the exam findings and recommendations are properly communicated. A doctor-to-doctor phone call describing the pertinent findings and recommendations, as well as a detailed letter handed to the patient, seems to be the best approach.

This is especially true if you are sending the patient to a hospital that doesn't have an optometrist or ophthalmologist on staff. In general, if you are going to make this type of referral, it's best to find out which hospital closest to you has these providers on staff. If they do not, it just makes your communication of the findings and recommendations all the more critical.

Additionally, optic nerve photos and visual field results are equally as important as communicating your findings. Almost every optometry office these days is equipped with both and it's just another layer of data for the ED staff to have. Otherwise, these work-ups are usually done on blind faith of the findings communicated to them. Although most ED rooms are equipped with an ophthalmoscope, it's rare to find a hospital staff proficient in using them. Even if your office is mostly electronic and it's difficult to print out field or fundus photos, a simple picture taken and texted to the patient is extremely helpful.

While neuro-ophthalmology is always the first option when presented with cases like this, when that's not available the hospital setting can be an equally effective and, many times, a necessary alternative. As long as proper communication exists between the optometrist and hospital staff, work-ups can certainly be guided in the right direction. Testing and specialty consultations can all be done within the same facility aiding in good doctor-to-doctor communication and also will be performed in the fastest and most efficient manner. ■

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KEEP GLAUCOMA CARE CLOSE TO HOME

While comanagement is a key component of long-term success, ODs must take on the lion's share of day-to-day responsibilities for these patients.

BY CATLIN NALLEY
CONTRIBUTING WRITER

Referrals are a critical aspect of optometric practice, even among ODs who practice to the full extent of their license. Therefore, they must be prepared to collaborate with specialists while maintaining their position as the primary eye care provider. This involves offering detailed recommendations to any specialist to whom they are entrusting their patient's care.

"The role of the OD as the manager of their patients' vision and medical eye wellness is a critical component of optometry that has been taken on by our profession very appropriately over the last decade or so," notes James Thimons, OD, of Ophthalmic Consultants of Connecticut.

"Your patients have chosen you as their primary eye care provider, the individual who will serve their best interests and needs," he emphasizes. "One way we do this is by knowing when to refer to a specialist. The concept of shared patient care has become the standard that we live by."

Why is a collaborative approach important? First and foremost, the quality of care provided to the patient is—in most cases—markedly better, according to Dr. Thimons. It offers patients convenience as well as the ongoing support of the primary eye care provider they know and trust.

For optometrists, it keeps them connected to the patient's medical care while providing the additional benefit of an extra set of eyes. It also offers an avenue for professional development. ODs can use this relationship to grow their skills as well as their practice.

"Glaucoma is a disease that involves a lifetime of continued management," says Dr. Thimons. "And so, the comanagement of these patients is very different than the episodic relationship we see with other diseases. It is much more intimate and evolved. It is also the one—in my opinion—with the highest level of responsibility but also holds the greatest rewards."

Establishing Relationships and Effective Collaboration

Successful collaboration depends on the skills of both the optometrist and specialist as well as a strong relationship founded in mutual trust and respect. This is especially true when it comes to glaucoma—a chronic condition that requires ongoing care and close comanagement.

Collaborative care begins by fostering connections in your community. "Connecting or meeting with surgeons in the area is a great first start to build a relationship," suggests Abagail Kirk, OD, of Memphis. "It's important for an optometrist to introduce him or herself to the new ophthalmic community they are joining," and that includes both other optometrists and ophthalmologists, she notes.

"At my practice, we have a dedicated community outreach team that meets and introduces new optometrists in the area to our practice and mission," she continues. "This way, the optometrist feels welcomed and already has the names and numbers of specialists that can participate in patient care."

When choosing the right glaucoma surgeon—or any comanagement partner—there are a number of factors to take into consideration, including training and expertise. Another critical component is making sure you are working with someone who aligns with your practice.

"Connecting with someone who shares your philosophies when it comes to glaucoma management is key," notes Dr. Thimons. "What you don't want is to join forces with someone whose approach conflicts with your own; this can only be derived through discussion."

Dr. Thimons also recommends that you spend time with them, particularly in the OR, if possible. "That's the best place to observe their skills while also getting a sense of how they interface with patients. Do they match or complement your style? Will your patients feel comfortable with this person?"

Once you have connected with a glaucoma surgeon, building a strong relationship that includes effective communication is critical. You have to feel comfortable with how you work together and exchange information. "Comanagement is successful when it involves partners who value one another," Dr. Thimons emphasizes. "Open lines of communication ensure you are both on the same page and that the patient receives seamless, high-quality care."

Optimizing Your Approach to Glaucoma

Unlike some other ocular conditions for which there's a clear need to comanage—such as cataracts—glaucoma is a lifelong disease that will most likely require ongoing collaboration. Therefore, ODs must have the knowledge and skills necessary to optimize not only the care they provide as an individual, but also their work as a comanagement team.

While the breadth of an optometrist's glaucoma management depends on the state in which they practice, all ODs have the authority and expertise to medically manage these patients.

How a glaucoma case is handled really depends on the stage of the disease at diagnosis. If the patient has mild glaucoma and is older at the time of diagnosis, they may never need surgery, notes Houston glaucoma expert Danica Marrelli, OD. In those cases, she may not need to comanage with a glaucoma surgeon at all.

"If I have a patient who can be managed medically, there's no reason to involve an ophthalmologist," she emphasizes. "On the other hand, if I have a younger patient or the diagnosis is more severe, I will begin my comanagement relationship earlier. I'll discuss the need for potential laser or surgical interventions with my patient and I may get a consultation with the surgeon sooner."

Managing medications includes understanding when adjustments need to be made to a patient's treatment regimen and which medications work well together. "There's a tendency for optometrists to prescribe one medication and then, if the patient's not well-controlled, to send them to an ophthalmologist," notes Dr. Marrelli, while suggesting that this may be premature. "If needed, I am more than capable of prescribing multiple medications to a patient and monitoring them appropriately."

However, that does not mean you withhold interventions that could benefit your patients, she remarks. Medication does not work for every patient and there are a variety of reasons they may not adhere to medical therapy. In those cases, Dr Marrelli stresses the need for additional interventions. "You don't want to hold on to your patient just to hold on to them," she notes. "But I also don't think there is a significant reason to involve ophthalmology if a patient can be successfully managed with medicine."

Another important aspect of glaucoma management is minimally invasive glaucoma surgery (MIGS), which has dramatically lowered the threshold for surgical consideration. The comanagement for these procedures is typically very straightforward, notes Michael Chaglasian, OD, of Illinois College of Optometry. The challenge can be building your knowledge and understanding of the wide variety of MIGS procedures available.

"This can be confusing and sometimes overwhelming," Dr. Chaglasian acknowledges. "Educating yourself takes time and experience, but it is an important aspect of care that ODs need to feel comfortable discussing with their patients and managing with specialists."

It is also important to recognize that not every glaucoma and cataract surgeon performs every MIGS procedure. Therefore, ODs must communicate with the specialists in their area and determine if that individual is trained to perform the procedure that is the best fit for your patient, notes Dr. Chaglasian.

The increasing reliance on selective laser trabeculoplasty (SLT) earlier in the course of glaucoma adds another

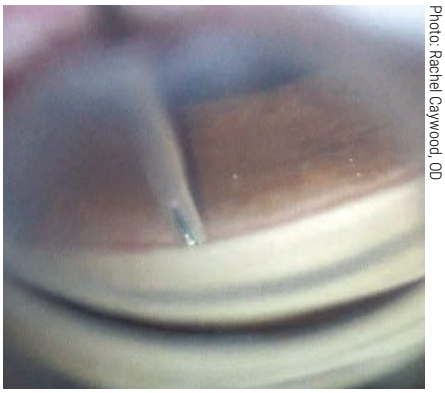
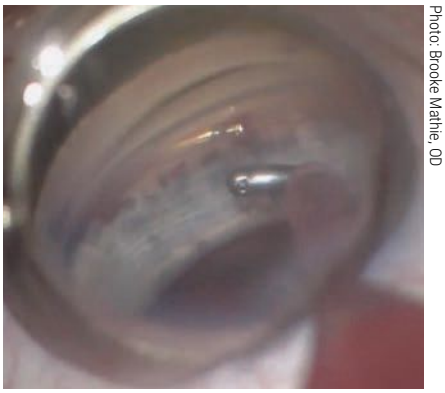


Photo: Rachel Caywood, OD



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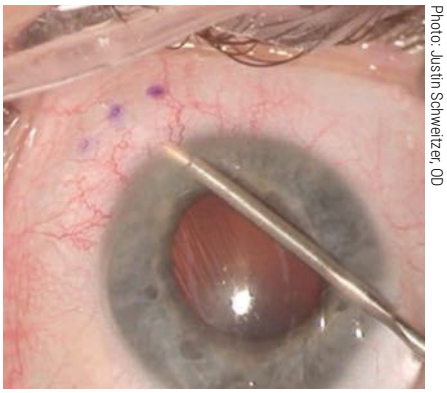
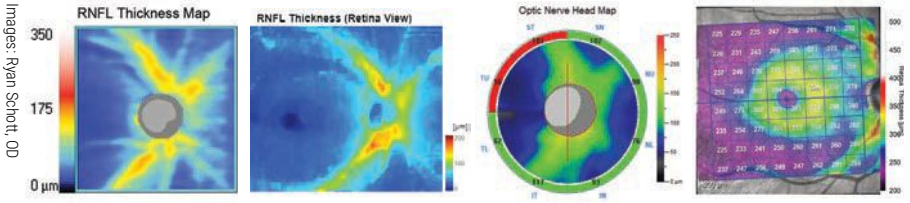


Photo: Justin Schweitzer, OD

A stent for all occasions. *Left to right:* iStent Inject (Glaukos), Hydrus Microstent (Ivantis), Xen gel stent (Allergan). ODs should be well-versed in the uses of these and other options to play a part—perhaps a decisive one—in the timing and selection of a MIGS procedure.

Images: Ryan Schott, OD



Understanding the fundamentals of OCT use in glaucoma is now an essential part of care and greatly extends the OD's ability to manage cases themselves prior to referral. Shown here are four RNFL thickness maps of the same patient on different platforms, from an analysis *Review of Optometry* published in February 2020; it can be found on the website.

***From left to right:* Zeiss Cirrus 6000, Topcon Maestro2, Optovue Avanti, Heidelberg Spectralis.**

opportunity for OD-MD comanagement before the disease advances. Increasingly, it's the primary care optometrist who initiates discussions about SLT as an alternative to medications at the outset of care, and weighs in on the decision in conjunction with the patient and surgeon.

As with any referral, making sure all members of the care team are on the same page is vital to success. Just as the OD must share detailed information and recommendations, they must also be aware of the glaucoma surgeon's process. Optometrists should ask for the surgeon's pre-op protocols for all of the many different procedures they perform so they know which medications the patient will be on before or after surgery, suggests Dr. Kirk.

"For example, we start patients on acetazolamide (if there are no contraindications) and prednisolone acetate 1% prior to having Xen Gel Stent surgery, to reduce IOP spikes during or after the procedure and to cut down on post-op inflammation leading to potential scarring of the bleb later," she explains. "If cleared by cardiology, we may stop blood thinners to lower the chance of developing a hyphema. At day one post-op intraocular pressure, we adjust the medications accordingly to avoid hypotony."

Postoperatively, ODs can and should manage their patients. "Optometrists are more than capable of treating and managing glaucoma post-surgical interference; the data we collect and assess is the same," notes Dr. Kirk. "If the intraocular pressure is not reaching the expected outcome following glaucoma surgery, then communicate that to the surgeon."

Effective comanagement following surgery starts on day one post-op. "Assess the incisions, levels of inflammation and edema, the appearance of the bleb (if applicable) and the intraocular pressure," she outlines. "Becoming familiar with what is normal makes it much easier to catch those rare occasions that are abnormal."

Seeing the patient post-op in the morning is also important, Dr. Kirk advises. Some patients may be in pain while others may not have picked up their post-op drops like they were supposed to and will need to go to the pharmacy. "Sending an emergency back to the surgeon is much easier to do if you catch it in the morning vs. late afternoon," she notes.

"Having a basic understanding of the procedures available via research, attending a CE or asking the surgeon is not just important but imperative to your confidence and ability to accurately continue certain treatments," Dr. Kirk emphasizes.

Effective patient communication is also vital to glaucoma comanagement. This begins by helping patients understand the process. Setting expectations when it comes to potential interventions prior to sending them to an ophthalmologist is extremely helpful, according to Dr. Kirk. "A patient that already comes in with an understanding of what to expect shows the surgeon they can trust your assessment of your patients, which streamlines the consultative process and sets the patient up for success."

Addressing Challenges & Gaps

A critical aspect of glaucoma management is knowing when to escalate from medical to surgical intervention. Navigating this requires that ODs have confidence in their skills and expertise as well as a strong relationship with the glaucoma surgeon.

To avoid under- or over-referral, the optometrist must identify the appropriate target pressure for their patient from the onset of diagnosis, suggests Dr. Chaglasian. "Keeping track of that target pressure and whether or not you have achieved it is key," he says.

"Simultaneously, you should be tracking your patient for progressive changes to the visual field and OCT," he continues. "Any evidence of progression is an indicator that you are not at target pressure and further treatment is necessary, which could entail a number of different approaches such as SLT or MIGS."

Successfully determining the timing of a referral comes down to tracking a patient appropriately during the medical treatment phase, emphasizes Dr. Chaglasian. While identifying progression in glaucoma is more challenging than other conditions due to the diagnostics required, it is well within the skills of an OD, he notes.

REFERRAL LETTER TIPS FOR THE OD

- Be clear and concise in your communication.
- Tell them why you are referring and what you have discussed with the patient.
- Include all relevant investigations such as a progression analysis on the visual field or OCT.
- Share pertinent patient information (*i.e.*, history of non-adherence; well-controlled on meds; narrow angle).
- Make recommendations based on your knowledge of the patient.
- Outline the comanagement relationship you expect to have with the specialist.

"Many glaucoma specialists are not interested in managing straightforward medical glaucoma, Dr. Chaglasian explains. "They want to work with optometrists who are willing to take on this aspect of care; when done well, this represents a true comanagement partnership."

In Dr. Kirk's practice, few of the glaucoma evaluations she sees are unwarranted. "Unfortunately, my experience is usually the opposite," she says. "I see a handful of patients present with severe or end-stage glaucoma only using latanoprost 0.005% when they can remember to take it.

"These cases are the toughest," she adds. "Seeing an established patient referred in for glaucoma surgery when drops had been failing for decades and now the only option is a tube is disheartening."

It is important that ODs think long-term when strategizing for these patients, Dr. Kirk notes. The question to ask themselves is, "How can I keep this patient seeing as much field as possible for the rest of his or her life?"

There are many new devices and surgical procedures that are low-risk, low-anxiety experiences for the patient, and ODs need to be well-versed in their indications and outcomes, she explains. "SLT is a safe and easy treatment, implantation of iStent inject or Hydrus Microstent during cataract surgery are very easily implantable and can lower intraocular pressures up to 30% which adds little to no extra time in the operating room."

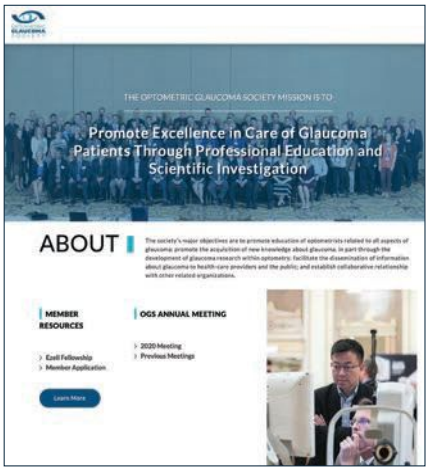
> **"Many glaucoma specialists are not interested in managing straightforward medical glaucoma. They want to work with ODs who are willing to take on this aspect of care."**

Another area that requires attention is gonioscopy, which, according to Dr. Thimons, is not used as frequently as it should be among both optometrists and ophthalmologists. "Gonioscopy should be used regularly for patients with open-angle glaucoma." Over time, the angle may begin to narrow and that can go unrecognized without the proper assessment, he says.

"Long-term progression analysis is at the heart of glaucoma therapy," Dr. Thimons says. "And it's quite critical that people incorporate technologies at the highest level possible, which is not always the case. It is also important that we don't default to newer technologies in replacement of previous ones—like gonioscopy—that remain essential to glaucoma care."

Technologies for Success

In general, the tools an optometrist uses are a main element of the level of care they are able to provide. It is also a key aspect of effective comanagement. Just as you need to have open lines of communication with the specialist, it is important to make sure the tools you use are in alignment.



Skill-building is critical, especially in glaucoma. The Optometric Glaucoma Society's website offers many educational resources teaching basic and advanced techniques, plus information about upcoming meetings and networking opportunuties.

"The tools don't have to be exactly the same as those the doctor you are comanaging with," notes Dr. Thimons. "However, you have to have the ability to communicate technically with equipment, so that you're not having to constantly translate information between offices into another language."

The technologies necessary for effective glaucoma management, according to Dr. Kirk, include OCT, visual field, pachymetry, gonio lens, direct ophthalmoscopy lens (a 60D is helpful as it provides the most accurate size of the nerve and a high-quality, detailed view) and a tonometer. She prefers a Goldmann.

Ideally, Dr. Kirk notes that ODs would also have access to a device to evaluate corneal hysteresis. While she acknowledged she currently does not have an Ocular Response Analyzer (Reichert) or similar product to assess corneal hysteresis, she is vying for one.

"I am also using our Heidelberg Spectralis OCT more in tandem with our Zeiss Cirrus OCT," she explains. "The tomograph of the Heidelberg is extremely clean and detailed, but the normative patient database is less diverse than the Cirrus and their demographic does not relatively reflect that of Memphis, where I practice."

Certain instruments also offer excellent reports that are highly valuable, suggests Dr. Kirk, such as at the guided progression analysis on the Zeiss Humphrey Visual Field Analyzer, or the Hood Report from the Heidelberg Spectralis and Topcon Maestro2. "This program offers a highly insightful ways of looking at the data and can easily identify structure-function relationships when present," she notes.

Continued Growth and Development

Optimizing your glaucoma management depends on your ongoing growth as a provider of optometric care. There are a number of ways to do this, and comanagement in and of itself is a means for professional development. It is an opportunity for ODs to enhance their glaucoma management skills and take a more active role in the management of these patients.

"Managing cases with the help of a specialist allows optometrists to expand their knowledge and skill," explains Dr. Thimons. "Next time, instead of referring that type of case, you may now have the confidence to manage it on your own. True comanagement is a teaching opportunity between professionals, which is what medicine should be when it's at its best."

When looking to improve and grow your glaucoma management skills, it is important to familiarize yourself with current procedures and devices, notes Dr. Kirk. "For instance, there is a wealth of free knowledge on the very hot topic of MIGS right now," she says. "Watch videos of the techniques and see how they are performed. It would behoove your clinical decision making and your patients' well-being to stay up to date."

Professional organizations are another excellent resource for education and support. "Whether through mentorship or continuing education, there are a number of ways for optometrists to enhance their skills," notes Dr. Marrelli. "For instance, the Optometric Glaucoma Society offers a non-CE credit series where small groups of glaucoma experts and doctors meet to discuss cases."

Glaucoma management is a crucial component of optometric practice, and it is well within the ODs scope of practice to medically manage these cases. To do so successfully, optometrists must dedicate their time to not only enhancing their skills, but also building a strong comanagement relationship where they take the lead as the primary eye care provider.

Building the foundation for effective glaucoma management—and comanagement—takes an ongoing effort. This includes embracing additional responsibilities within your scope of practice; however, this can and should be done at your own pace.

"I am so excited for Massachusetts and the recent Texas glaucoma law," notes Dr. Marrelli. "But the truth is, just because a law goes into effect doesn't mean that the optometrist is ready to start this process. However, they also shouldn't be intimidated either. Comfort and confidence come with time.

"I would encourage ODs to reach out to fellow optometrists who are actively managing glaucoma," she notes. "They are very willing to walk alongside those who may want to start, but just don't quite know how. Taking that first step can be overwhelming, but there are a variety of resources available, and optometrists are well-equipped to take charge when it comes to caring for these patients." ■

KEY TAKEAWAYS

- Actively share your recommendations with the glaucoma surgeon when referring patients.
- Open lines of communication between practices is essential when comanaging patients.
- Educate your patients and set expectations prior to sending them to a specialist.
- Comanagement is an opportunity to learn. Take advantage of this to enhance your skills and build your confidence.

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OCT INTERPRETATION FOR GLAUCOMA: DON'T GET FOOLED

Learn how to obtain and interpret accurate, reliable ophthalmic images.

BY JULIA REIMOLD, OD, AND CHRIS WROTEN, OD
DENHAM SPRINGS, LA

Optical coherence tomography (OCT) is truly an engineering marvel, with scan resolutions ever-increasing, wider areas of tissue imageable than ever before and more layers of the eye included in every new instrument iteration. It's been cleverly described as an "optical biopsy."[1] That box sitting in the special testing room at our clinic is able to biopsy *in vivo* tissue using light. Not only is that cool, but just think of how few other medical specialties have access to a quick, accurate, painless and noninvasive "biopsy" of the organ they are caring for?

Let's briefly review where this amazing technology came from, where it is now and where it's headed, then review best practices on how to use OCT to diagnose and manage glaucoma.

Development of a Marvel

The concept of OCT was first proposed in 1971, but it took another 20 years for the first optical coherence tomogram to be obtained—an *in vitro* scan of the retina. By 1993, *in vivo* OCT images of the optic nerve and macula were obtained, further accelerating commercial development. Early OCT instruments used time-domain imaging with speeds of 400 A-scans per second to analyze ocular tissue with resolutions of 10 to 20µm to produce the now familiar temporal, superior, nasal, inferior, temporal (TSNIT) curves and RNFL pie charts (*Table 1*).[2]

The early 2000s ushered in spectral-domain OCT (SD-OCT), which is the current industry standard, with speeds of 50,000 to 100,000 A-scans per second and resolutions of 5µm to 7µm. However, in 2012, swept-source OCT (SS-OCT) was successfully commercialized, further enhancing OCT capability, followed in 2014 by OCT-angiography (OCT-A), a new add-on module for SD-OCT instruments that tracks movement in vasculature to indirectly evaluate blood flow.

In the near-term, SS-OCT and OCT-A both have approximately 5µm resolutions with scan speeds of up to 400,000+ A-scans per second and up to 70,000 A-scans per second, respectively. Both are poised to further enhance our diagnostic and management capabilities with their ultra-resolution and novel technology.

Image Acquisition and Interpretation

While anterior segment OCT continues to develop, in the posterior segment we can now image from the vitreal cortex to the sclera depth-wise, while capturing single images that span from nasal to the optic disc to temporal to the fovea. Scan protocols vary among manufacturers but share many similarities. Using both A-scan and B-scan technology, OCT instruments measure the amount of backscatter from the coherent light introduced into the eye. The more reflective the material encountered, the brighter its appearance on the OCT image. This allows analysis of each layer of the retina individually, as well as the optic nerve.

Within the retina, abnormalities such as lipid exudates, pigment, epiretinal membranes (ERM) and chorioretinal scars are brighter white; while subretinal fluid, edema and areas of detachment are optically dark (*Figure 1*).

About the authors

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Glaucoma Management Using OCT

Glaucoma is a group of progressive optic neuropathies characterized by degeneration of retinal ganglion cells (RGCs) and their axons, resulting in retinal nerve fiber layer (RNFL) loss, neuroretinal rim thinning and corresponding visual field (VF) deterioration.[3,4] Since it's one of the leading causes of preventable blindness, early detection and intervention are vital to prevent vision loss for patients with glaucoma. OCT allows clinicians to monitor for glaucomatous structural damage involving the optic nerve, peripapillary RNFL and macula, as well as to correlate that structural damage to functional vision loss.[3]

Circumpapillary RNFL measurements have become a commonly used parameter in glaucoma management for assessing RGC structural loss. However, this method of analysis only looks at the axonal portion of RGCs without considering the cell bodies or dendrites, potentially missing early glaucomatous damage.[3,5] For these reasons, examination of the macula and, more specifically its ganglion cell complex (GCC), has arguably become the standard of care in detecting early glaucomatous changes.[6]

Approximately 30% of RGCs are located within the macula (despite the fact that the macula only represents 2% of the retina) resulting in a much thicker retinal ganglion cell layer (GCL) than in the circumpapillary area.[7] Consequently, macular GCC thinning has been identified as an early indicator of glaucomatous structural damage.[3-8]

The GCC is comprised of the three retinal layers that are damaged in glaucoma: the RNFL, the GCL and the inner plexiform layer (IPL). It is hypothesized that the earliest changes are seen as a loss of dendrites (IPL), resulting in subsequent apoptosis of RGC bodies (GCL) and axons (RNFL).[5] Previous studies have attempted indirect estimation of RGCs through macular thickness analysis; however, results were found inferior to GCC measurements and inaccurate due to the significant influence from outer retinal layers.[3,9,10] Newer OCT instrumentation allows for automated segmentation of macular layers, completely excluding the effect of outer retinal layers.

However, GCC measurements are not always reliable indicators of progression, especially in patients who present with coincident macular disease (*e.g.*, macular degeneration, vitreomacular traction, epiretinal membrane), which can artificially increase or decrease foveal thickness measurements. In these instances, RNFL analysis may prove a more accurate indicator of glaucomatous progression.

Table 2 shows three commonly used OCT instruments and the parameters for which the GCC is calculated.[3,11-13] Although GCC analysis techniques vary among the different instruments, all accurately and effectively allow for quantification of the innermost retinal layers and detect pre-perimetric changes related to glaucoma. Further, to better identify damage, the nasal-superior-inferior-temporal-nasal (NSITN) curve and schematics include a map of the region just outside the macula, where most of the 24-2 VF test points fall.[14]

TABLE 1. THE EVOLUTION OF OCT IMAGING PLATFORMS

Imaging Platform	Scan Speed (A-scans per second)	Axial Image Resolution (µm)	Anatomy Imaged
TD-OCT	400	10	Vitreoretinal interface to RPE
SD-OCT	27,000 to 60,000	5 to 7	Posterior vitreal cortex to sclera*
SS-OCT	100,000 to 400,000	5 to 7	Posterior vitreal cortex to sclera
OCT-A	≥70,000	~5	Vascular structures

* To image the sclera, a special setting is used for SD-OCT that sacrifices posterior vitreal cortex and inner retinal layers to obtain the scan; SS-OCT does not suffer this compromise.

TABLE 2. MACULAR THICKNESS PARAMETERS FOR VARIOUS OCT INSTRUMENTS[3,11-13]

Instrument	Macular Thickness Measurements
RTVue-100 (OptoVue)	Ganglion Cell Complex (GCC) = RNFL + GCL + IPL layers
Cirrus HD-OCT (Carl Zeiss Meditec)	Ganglion Cell Analysis (GCA) = Macular RNFL, GCL + IPL, GCC
Spectralis (Heidelberg)	Posterior Pole Asymmetry Analysis (PPAA)
Maestro (Topcon)	GCC = ILM to IPL/INL

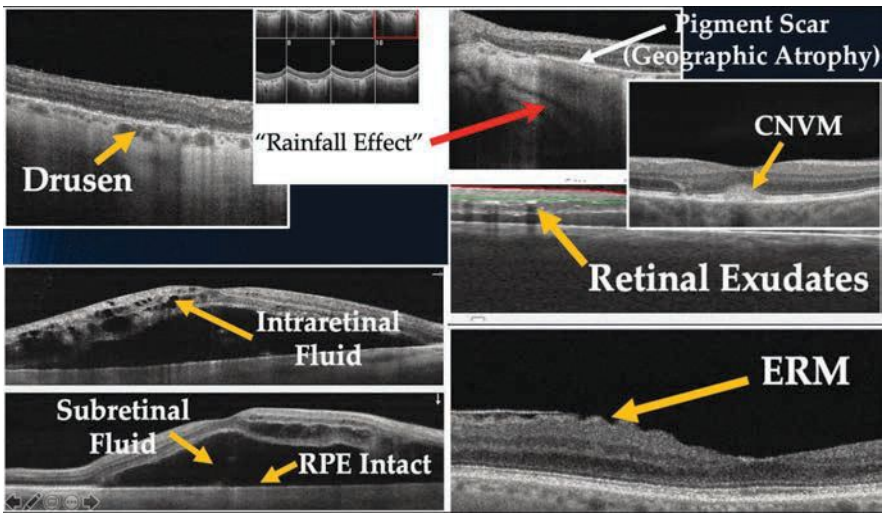

Fig. 1. Characteristics of ocular abnormalities on OCT images.

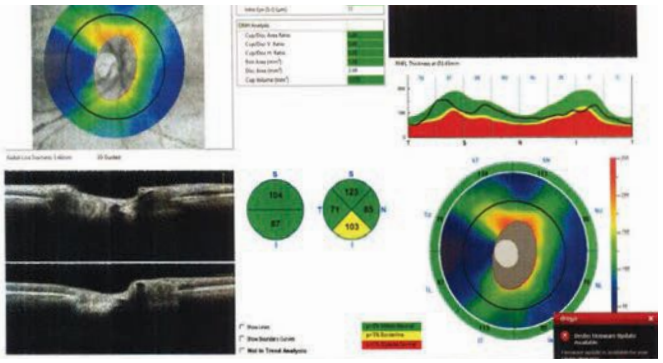

Fig. 2. A normal tension glaucoma suspect's RNFL scan of the ONH. The data is reliable and grossly normal, with the exception of the inferior RNFL pie chart thickness.

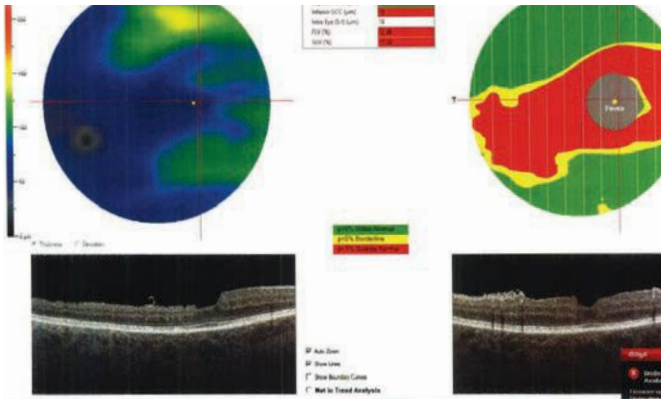

Fig 3. The GCC analysis of the macular region on the same patient as Fig. 2, taken on the same day. Results are reliable and indicated significant GCC thinning in the macular region.

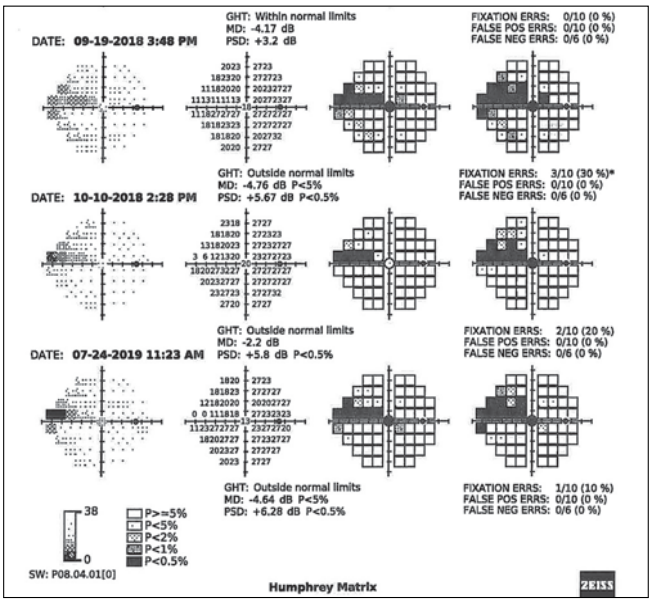

Fig. 4. Serial visual field results using Zeiss's frequency doubling technology on the right eye of the patient in Figs. 2 and 3, confirming functional VF defects in the form of a nasal step progressing to a superior arcuate scotoma.

Many studies have shown GCC parameters to be a superior tool in early detection of glaucoma compared to those of the circumpapillary RNFL in patients with normal macular anatomy.[3-5] More specifically, one study reports that the most accurate GCC parameters for detecting early glaucomatous changes are average and inferior GCC thicknesses.[3] Another found that one of the most sensitive predictors of glaucomatous progression is focal loss volume in the GCC and RNFL.[15]

Additionally, patients who are myopic present another clinical scenario where macular thickness analysis may prove superior to RNFL measurements. Glaucoma detection in highly myopic individuals can be challenging, given the deformation of the retina and optic nerve that accompanies axial elongation of the eye. Unlike RNFL, the diagnostic accuracy of the GCC is not affected by increasing axial length and, therefore, may prove one of the best parameters for detecting the presence and progression of glaucoma in cases where tilted nerves and peripapillary atrophy are present and the optic disc limits cannot be set.[4,6]

Few studies have demonstrated the superiority of RNFL over GCC analysis in detecting pre-perimetric glaucoma, early glaucoma and glaucoma progression.[5] However, it is important to note that GCC measurements are not always reliable indicators of progression, especially in patients who present with macular diseases, which can artificially thin or thicken foveal thickness. In these instances, RNFL analysis may prove a more accurate indicator of glaucomatous progression. As a result, practitioners should incorporate macular GCC measurements as an adjunct to RNFL parameters for all glaucoma patients and glaucoma suspects to aid early detection, diagnosis and disease management to prevent blindness.

Thankfully, many OCT manufacturers have begun incorporating macular scans as part of their glaucoma imaging protocols and analyses (*e.g.*, Carl Zeiss Meditec's PanoMap, the Hood report from both Topcon and Heidelberg, and Optovue's ONH/GCC Report).[7]

Case in point: *Figure 2* shows the RNFL OCT scan of a normal tension glaucoma suspect's right eye using the Optovue Avanti. The scan is reliable and all parameters appear relatively unremarkable with the exception of the inferior RNFL pie chart thickness, which is borderline but still 103µm thick. That same day, the patient's macular GCC was also analyzed (*Figure 3*), which provided additional, clinically useful information that presented a markedly different clinical picture. Even factoring in the coincident epiretinal membrane, the GCC scan is highly suggestive of structural glaucomatous damage that was further confirmed by frequency-doubling visual fields (*Figure 4*).

When only analyzing the RNFL in isolation, it may have been easy to miss the inferior thinning and just schedule the patient back in another year, rather than intervening as was clearly indicated by the VF and GCC scan.

"I was only seeing light flashes early on, but light FLASHES when you've not seen anything for so many years—it was wonderful"

—Keith H, retinal prosthesis recipient

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Common Confounders to OCT Interpretation

Knowing what to scan and how to interpret the OCT is vital to glaucoma care, but obtaining accurate, reliable ophthalmic images is also crucial for proper diagnosis and treatment. When image quality is unreliable or subpar, inaccurate data confounds image interpretation—potentially leading to improper clinical decision-making. SD-OCT has gained widespread use among clinicians for its ability to assist with diagnosis and management of ocular conditions; however, one must understand the nuances of the data obtained in order to use the technology effectively.

There are three main sources of artifact that can erroneously influence data during OCT image acquisition. These confounders can be classified as patient-dependent, operator-dependent and device-dependent.[16,17]

During OCT image acquisition, light from the device is emitted toward the structure being analyzed, then incident light is back-scattered and captured for processing.[2] Obstacles that block the pathway of light can contribute to poor scan quality and subsequently poor reliability metrics, yielding potentially providing misleading information.

Patient-dependent. Examples that can influence scan quality include coincident ocular pathology, as well as patient fixation and cooperation during image capture. Patients with glaucoma often have other co-existing conditions such as cataracts and ocular surface disease, given their increased prevalence with age and use of hypotensive agents.

Many studies have demonstrated the direct correlation between reduced scan quality and decreased tissue thickness.[18,19] *Figure 5* demonstrates this relationship on a patient with dense posterior subcapsular opacification (PCO) immediately pre- and post-YAG capsulotomy. Foveal thickness is falsely reduced prior to removing the opacity, resulting in a clinically significant increase in RNFL tissue thickness once removed.

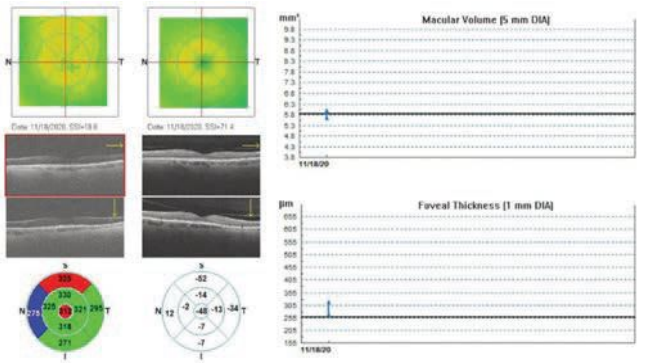

Fig. 5. Optovue retinal map change analysis of a patient with grade 3 PCO immediately pre- and post-YAG capsulotomy. Significant improvement in image resolution was obtained, demonstrating artifactual changes in macular volume and foveal thickness and decreased average RNFL thickness, compared with the same eye correctly imaged on the right.

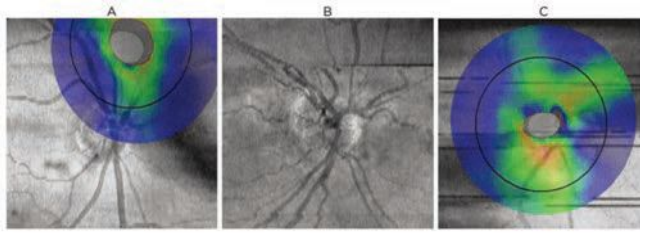

Fig. 6. Common scan artifacts and confounders to OCT interpretation on the Optovue RTVue-100. (A) Centration artifact: poor target alignment of analyzed structures results in a false increase/decrease of tissue thickness. (B) Motion artifact, *e.g.*, as results from head movement, saccades or breathing. Appears as misalignment of blood vessels and can lead to segmentation error. (C) Blink artifact: appears as horizontal gaps in data.

If we had diagnosed this patient with glaucoma at some point after cataract surgery when the posterior capsule was clear, obtained baseline OCT scans, initiated glaucoma treatment and they then developed PCO over the next several years, the trend-based RNFL analysis would've indicated progressive thinning suggestive of worsening disease. In actuality, it was just due to PCO formation. Take the entire clinical picture into account before making clinical decisions.

Instillation of artificial tears and having a patient blink before image capture are some possible solutions to improve scan quality in patients with and without dry eye. This can also help minimize blink artifacts, which are shown in *Figure 6*. The gaze-tracking feature on most instruments helps minimize the effects of blinking; however, the duration and position of a blink artifact during a scan can result in transient loss of data.

In addition to conditions that block the path of light of the device, it is equally important to determine how existing pathology influences OCT measurements. Vitreous traction and epiretinal membranes are some examples that cause false elevation of tissue thickness, while peripapillary atrophy and scarring results in loss of tissue that can mimic changes related to the glaucomatous process. By minimizing these variables and understanding their effects, optometrists can more accurately determine stability in diseased states.

Operator-dependent. These variables are another significant cause of OCT confounders, most of which can be avoided with proper training (*Figure 6*). Centration artifacts occur when the acquisition frame is not properly aligned over the structure being analyzed (*Figure 6a*). When monitoring for glaucoma, improper centration of the acquisition circle over the optic nerve can result in false thinning or elevation in various sectors or quadrants.

RNFL thickness is greatest at the optic nerve head and decreases as the distance away from the nerve increases.[20-22] While mild shifts in scan position do not significantly alter RNFL measurements, moderate to severe misalignment can cause a shift in the TSNIT curve, resulting in artificial thinning or elevation.[20] Truncation artifacts result when only a portion of the structure analyzed is in the acquisition frame (*Figure 7*). The area of missing

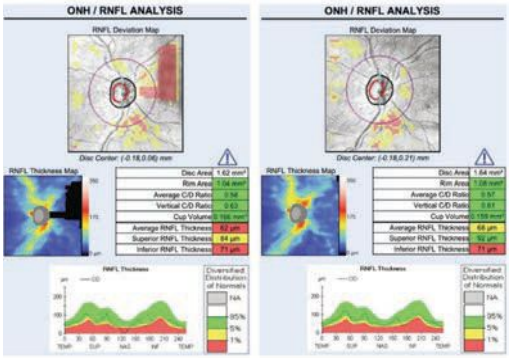

Fig. 7. Truncation artifact: structures being imaged (in this case the optic nerve/RNFL) are only partially included in the acquisition frame in the left image. Superior nasal loss of data results in incorrectly assumed thinning of the superior RNFL and a decrease in the average RNFL thickness compared with the same eye correctly imaged on the right.

data falsely underestimates the nasal tissue thickness. Appropriate alignment of data within the scan window ensures the most accurate measurements and ability to track progression.

Device-dependent. These errors occur when instrumentation improperly delineates retinal layers. These segmentation issues are commonly seen in patients who have high myopia, prominent posterior hyaloid membranes and media opacities as they affect image quality and make it difficult for the device to accurately determine retinal boundaries.[23] These types of artifacts are sometimes easily overlooked on the final clinical printouts, potentially leading to errors in clinical interpretation. Physicians can limit the effects of such confounders by having access to raw data in the exam lane before making any significant treatment decisions.

The influence of image quality on OCT metrics should be considered when analyzing data and making clinical decisions regarding a patient's care. Before considering the metrics, one must first assess quality by looking at the scan accuracy, signal strength, centration and segmentation and by taking into consideration any ocular pathology that could affect the data. By increasing the accuracy and reliability of OCT data measurements, clinicians can further enhance their management of ocular pathology, thereby improving healthcare outcomes.

Having a general understanding of any potential normative database biases unique to an individual manufacturer's OCT, relying on the manufacturer's sophisticated trend-based analyses instead of just "eyeballing" it to look at trends over time, and recalling the normal age-related changes in RNFL and GCC which occur (~0.50µm per year of RNFL loss and ~0.25µm per year of GCC loss) are also critical to accurate OCT interpretation.[24,25]

Takeaways

Remember to assess the macula when diagnosing and managing glaucoma. If your OCT instrument does not automatically include the macula in its optic nerve/glaucoma analysis protocol, then image the macula separately to enhance clinical decision-making.

For any OCT scan, prior to making any clinical decisions (and before looking at any colors on the printout), clinicians should always:

- Assess scan quality first.
- Look for any acquisition errors that may affect interpretation.
- Review the patient's clinical history for pathological confounders.
- Assess results relative to normative databases.
- Consider trend-based analysis results (if available) prior to making clinical decisions.
- Account for normal age-related changes in RNFL and GCC.

Using these ever-improving OCT instruments while maximizing the accuracy and reliability of acquired images, scanning both the macula and the optic nerve and correctly interpreting the data will yield earlier and more precise clinical decisions regarding glaucoma diagnosis and management. ■

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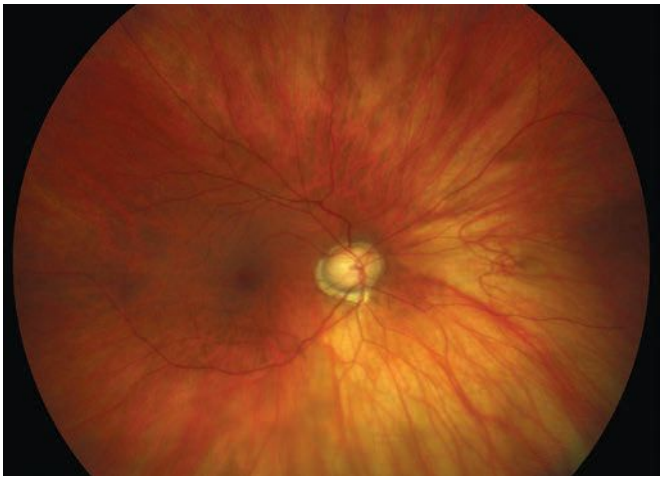
DIAGNOSING GLAUCOMA: HOW TO STEER CLEAR OF TROUBLE

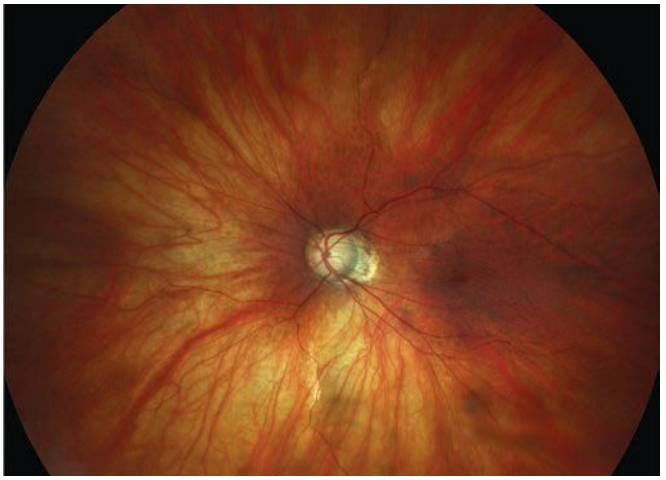
Here are several instances where conflicting or misleading information could lead you astray—and what to do about it.

BY SARAH B. KLEIN, OD,[1] AND MICHAEL CHAGLASIAN, OD[2]
[1]ROCHESTER, NY; [2]CHICAGO

Identifying glaucoma and assessing its severity in any given patient is like putting together a jigsaw puzzle without having the picture on the box in front of you. Strewn about are many different pieces of information—IOP, disc assessment, visual fields, RNFL analysis, corneal thickness, angle assessment, corneal biomechanics, age, race, family history and so on. You have to make all these pieces fit together into a coherent whole to decide whether the patient does indeed have glaucoma or ocular hypertension, is considered a suspect or gets a clean bill of health. But, lacking a completed picture to guide your efforts, you often find the pieces don't always fit nicely together. This often happens when our diagnostic tests, amazing as they are with today's technology, do not make sense or correlate with each other. There is also the possibility that tests produce false or inaccurate results due to other underlying factors.

When does this happen, and what can we do about it? Here are some examples of relatively common diagnostic pitfalls and how to use all the information at hand to make the best decision possible in the diagnosis and treatment of these tricky patients.





Figs. 1 and 2. Large discs and cups in a highly myopic patient.

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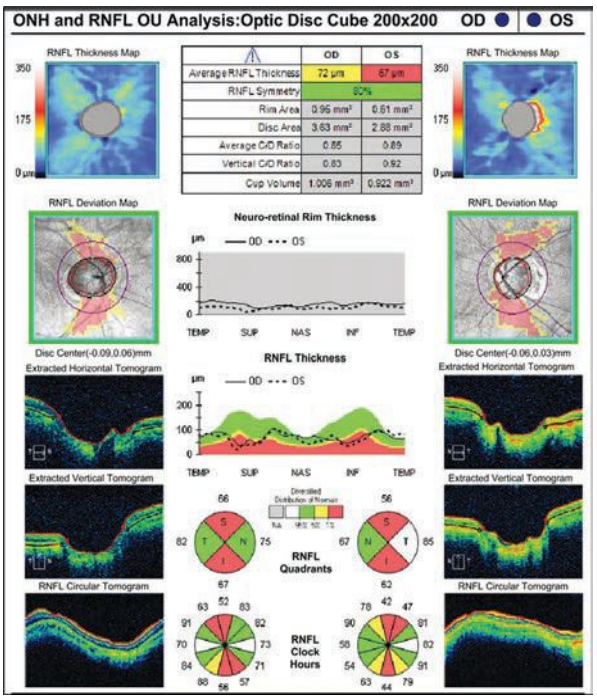


Fig. 3. RNFL analysis shows vertical thinning but confirms large disc diameter OD>OS.

High Myopia with Large Discs

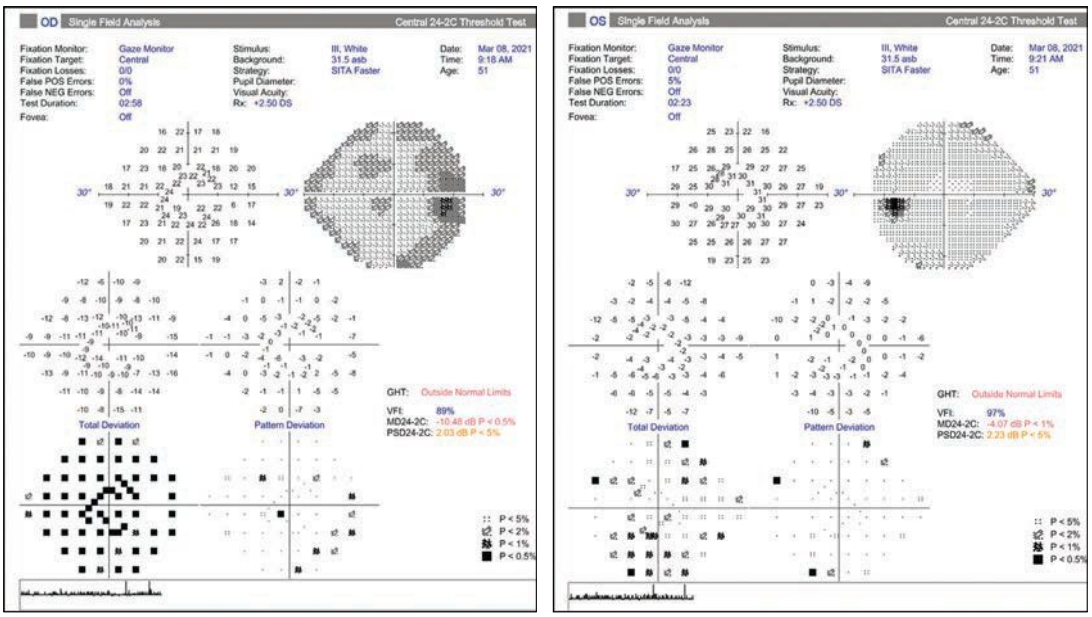
This type of patient has the distinction of being able to demonstrate two diagnostic pitfalls at one time: high myopia and large physiological disc diameters. As shown in the color disc photos, these optic nerves appear clearly suspicious for glaucoma (*Figures 1 and 2*). This patient is a -12D myope with mild cataract OD, an intraocular lens OS and a history of medical treatment for glaucoma (prior to cataract surgery). Currently her IOP is 16mm Hg OU without any therapy. Is treatment necessary? Is glaucoma the correct diagnosis or is this just a suspect?

When analyzed with retinal nerve fiber layer (RNFL) OCT, there appears to be relative thinning of the RNFL superiorly and inferiorly OU. Notably, the optic disc area is very large. When evaluating myopic patients with large disc size, practitioners need to consider two possibilities: (1) the instrument correctly identifies structural damage to the RNFL (or ganglion cell complex) due to glaucoma or (2) the software "misreads" the data and signals abnormality that is only anatomic or physiological in nature, and not related to disease (*Figure 3*).

Part of the explanation for this is related to the reference database (unique to each OCT), upon which the report analyses are based. Reference databases have limitations and do not include sufficient representation of large, normal physiological variations in optic nerve appearance. As related to this case, we know that high myopes (up to -12D) were eligible for inclusion in the database, but based upon disc area, only 11 of 282 subjects had a disc area of >2.5mm^2 for the Cirrus OCT by Carl Zeiss Meditec.[1]

On the RNFL report, we see that this patient has huge discs—areas of 3.63mm^2 and 2.88mm^2, respectively. So, although we see red flags here (literally) indicating very thin areas of RNFL, we need to be cautious due to possible presence of misinterpretation of the patient's scan, as it falls outside of the database. This potentially could be an example of "red disease," when non-glaucoma–related OCT findings are marked as abnormal on the color scale and mistaken as evidence for glaucoma by the clinician.

To help avoid this misinterpretation, it is critical to review the visual field analysis, which tells a different story (*Figures 4 and 5*). For the right eye, we see a generalized depression on the total deviation plot (due to cataract) that is reduced on the pattern deviation plot. Thus, there is no glaucomatous visual field loss, defined by three or more contiguous point defects that are 3db or more outside the normal range for the patient's age. The left eye demonstrates a relatively normal field, also without any definitive glaucomatous defects. Note again that scattered defects on



Figs. 4 and 5. Visual fields do not show loss that corresponds to RNFL loss indicated in Fig. 3.

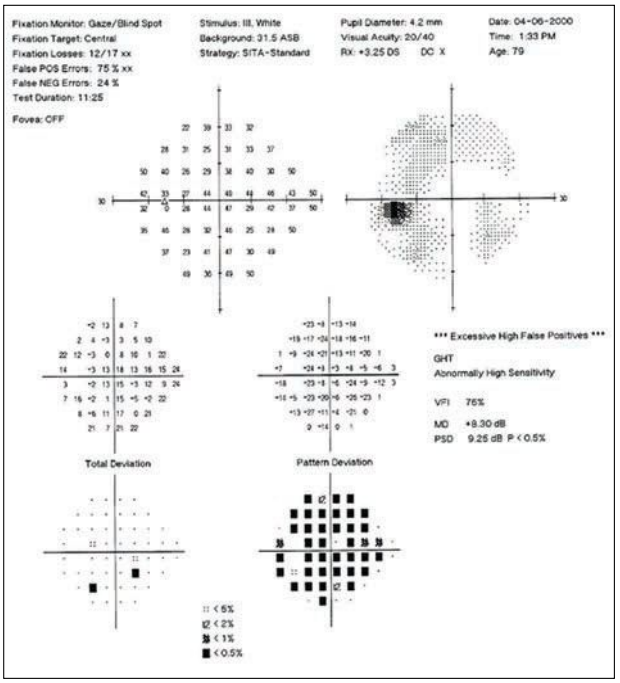


Fig. 6. Visual field of left eye with high false positives and a "reverse cataract" pattern.

the total deviation plot are mostly cleared on the pattern deviation plot.

It is crucial to compare the suspected zones of damage noted on the OCT with the corresponding points on the visual field analysis (here we would expect superior and inferior arcuate defects). This would confirm the typical structural and functional relationship that we observe in a true glaucomatous optic neuropathy.[2] Since the OCT and visual field diagnostic tests do not correlate exactly, we need to consider if the OCT is showing damage in advance of the visual field, which is certainly possible, or if the visual fields more accurately demonstrate lack of glaucomatous disease.

After several follow-up visits, careful review and repeating these diagnostic tests, we elected to monitor the patient closely, despite the suspicious OCT findings. Part of our consideration was that she may have had elevated IOP in the past, which is now in the normal range post-cataract extraction. She will be monitored through follow-up visits to detect signs of progression on visual field testing and in RNFL thickness on OCT.

Visual Field Pitfall: False Positives

Standard automated perimetry is one of the traditional gold standards in documenting the presence and progression of glaucomatous damage, and getting a good sense of test reliability is crucial. With the newer, faster test strategies, such as SITA-Faster (Carl Zeiss Meditec), we have the opportunity for more frequent testing due to shorter test time and less patient fatigue. However, the perks of faster testing come with a risk of increased false positive rates.[3]

False positives are the most important of the visual field reliability indices: the machine takes a pause and does not present a stimulus, checking to see if the patient clicks anyway. The calculated percentage is based on the number of times the patient clicks when no stimulus was presented. High false positives can result in field plots that have a clear or "white" looking greyscale map, do not show any defect on the total deviation but have significant defects on the pattern deviation plot. This "reverse cataract" pattern could fool one into thinking there is a depression or scotoma present, when actually it is an indicator of unreliable testing (*Figure 6*).

The general guideline is that test results with false positives of 10% to 15% or higher are unreliable and should not be used. Users should note that the new SITA-Faster strategy has eliminated testing for false negatives, as they are not helpful in glaucoma and add to the overall test time.

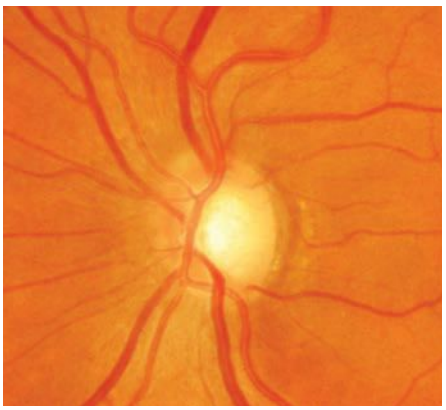


Fig. 7 (above). Suspicious optic nerve with above average c/d ratio.

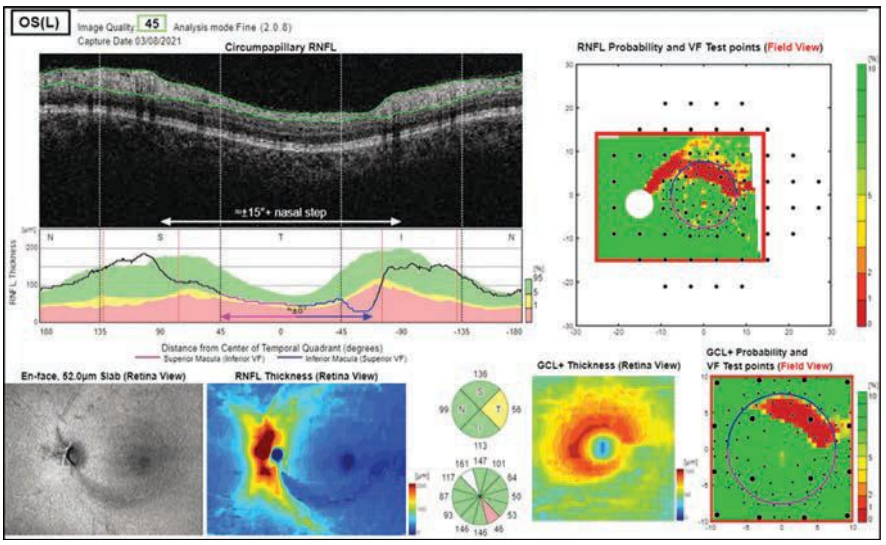


Fig. 8 (right). Borderline findings of thinner RNFL temporally and inferior arcuate GCC loss.

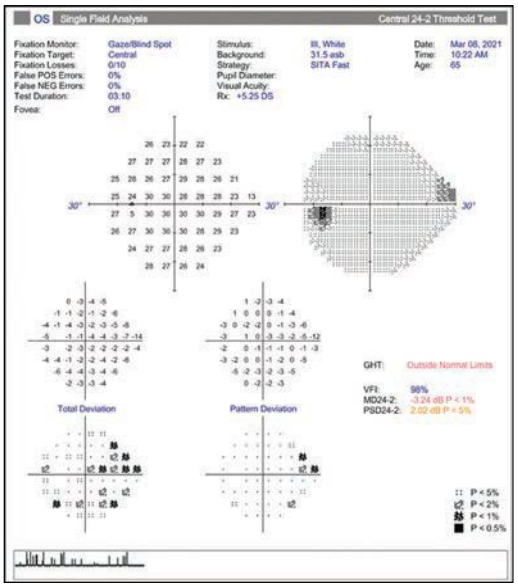


Fig. 9. Reliable HVF showing possible early superior nasal step in the left eye.

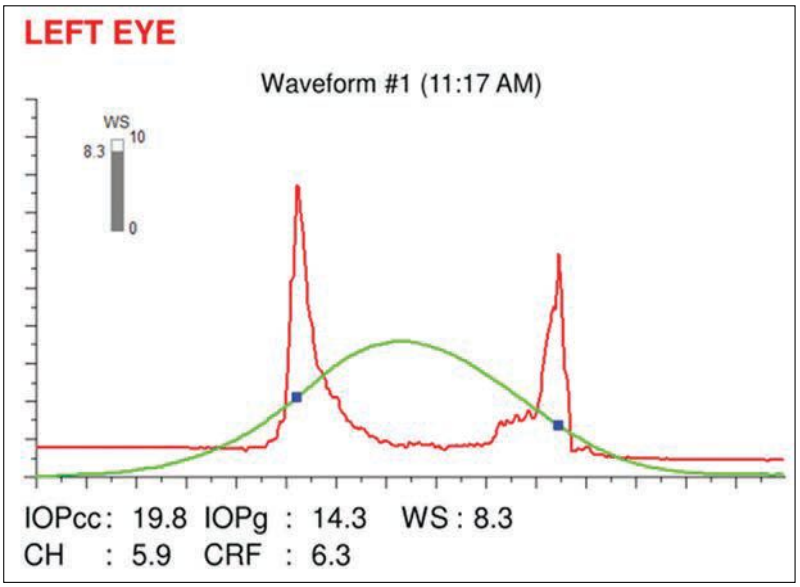


Fig. 10. Ocular Response Analyzer readout showing a corneal hysteresis measurement of 5.9, which is low.

Corneal Biomechanics Confound Assessment

Sometimes we have to make decisions about glaucoma diagnosis and treatment based on reliable test results that all end up in the "borderline" category. Just imagine a mixture of findings, some indicating disease while others support an assessment of no glaucoma at present. Another scenario is a patient who does have glaucoma and additional information directs us how to best treat and manage the patient.

Corneal hysteresis (CH), measured by the Ocular Response Analyzer (Reichert), fills in a helpful missing piece or adds another key risk factor to the confirmation of disease based on other findings.

Consider a case of a 65-year-old African-American female with a maximum IOP by Goldmann tonometry of 22mm Hg in the left eye, notching to the inferior temporal rim, significant inferior arcuate defects in the RNFL and GCL+ thickness color maps (*Figures 7 and 8*). Visual field is reliable, with three adjacent point defects in the superior nasal quadrant, and the glaucoma hemifield test (GHT) is outside normal limits (*Figure 9*). Here there is good structure-function correlation.

The very low corneal hysteresis measure (CH=5.9) is added confirmation to the presence of glaucoma and helps to indicate risk of future progression and thus the target IOP (*Figure 10*).[4] Low corneal hysteresis (<9.0) has been shown to be a reliable risk factor in determining the diagnosis and progression of glaucoma and tends to be low in glaucomatous eyes independent of IOP, central corneal thickness (CCT) and other factors.

In some studies, CH has been shown to be a stronger predictor of progression than low CCT.[4] In cases like this one, with the addition of the hysteresis measurement, we would set a lower IOP goal (~40% from baseline) and monitor the patient more closely than others who have a hysteresis measure in the normal range (10.5±1.5).

NTG and Diagnostic Testing Limitations

Patients with "normal" (or "low") tension glaucoma (NTG) can be particularly difficult to identify properly when traditional diagnostic testing fails us, as often it is all we have to rely on.

Here is an example of NTG in a 47-year-old Asian female with a history of NTG OD>OS. She had been treated with a topical prostaglandin analog for three years with excellent compliance, with an IOP range between 9mm Hg and 11mm Hg at all visits OU and a pre-treatment IOP in the mid-teens.

Looking at her diagnostic testing, all seems well. Visual field results appear to be essentially stable and reliable, with a dense superior altitudinal defect/paracentral scotoma with pattern standard deviation similar to initial testing (*Figure 11*). OCT of the RNFL is again very stable when looking at the nerve head compared to baseline (*Figures 12 and 13*). However, on dilated exam, with IOP of 9mm Hg at her last visit, her right optic nerve demonstrated a Drance hemorrhage at seven o'clock (*Figure 14*).

We know that although her IOP is excellent in-office and her testing appears very stable, the evidence of the disc hemorrhage tells us she may progress, with the possibility of showing additional RNFL loss in the area of the disc adjacent to the heme and then subsequent corresponding visual field loss in coming months/years.[5] Considering this, additional intervention and/or closer observation must be considered.

A look into her medical history tells us that she does tend to have low

blood pressure readings, with a diastolic that runs around 60, so ocular perfusion pressure may be an issue.[6] Obtaining some form of diurnal IOP may be warranted, to see if there is a time of day where her IOP rises. The iCare Home tonometer (iCare) is one method of obtaining this information.

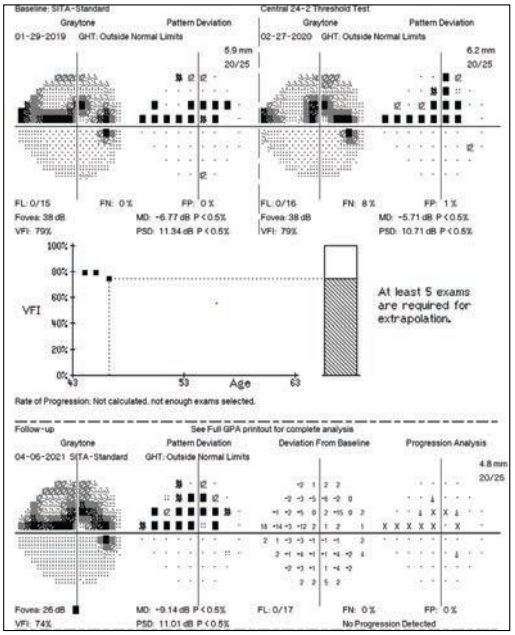
After discussing with the patient, we elected to add a second topical medication and selected the rho-kinase inhibitor netarsudil for its demonstrated efficacy in patients with intraocular pressures in the normal range.[7]

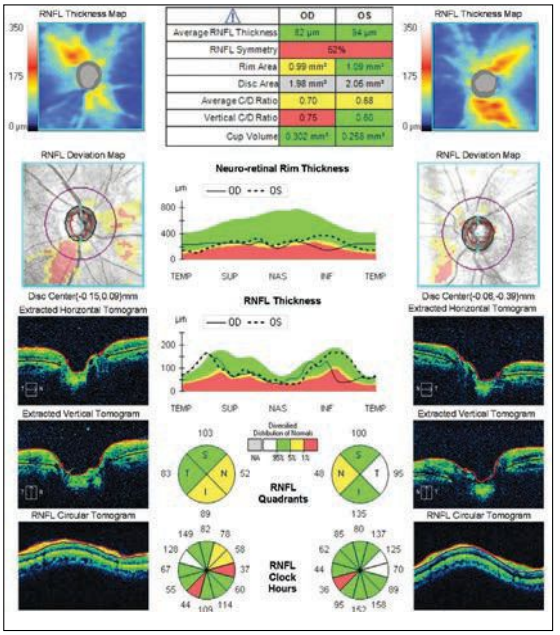
OCT and Fields: Anatomical Artifact or Glaucoma?

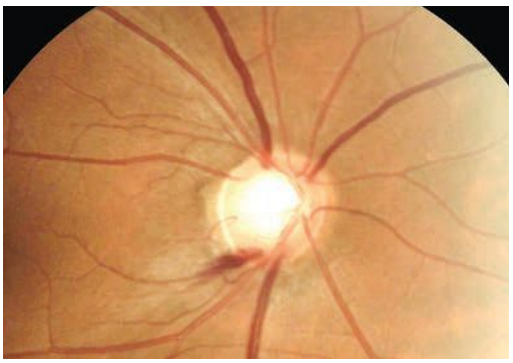
Sometimes, complicating factors such as concurrent retinal or ocular disease will interfere when we rely on testing for the diagnosis and treatment of glaucoma. This next patient, a 56-year-old African American male, presented as an outside referral for "glaucoma suspect vs. ocular hypertension." He had a history of elevated IOP in the high 20s, with Tmax values of 28mm Hg and 34mm Hg, respectively. He had open angles with no recession, average CCT, positive family history of glaucoma (mother) and diagnostic testing findings that were confounding. The left eye disc photo shows a moderately large cup-disc ratio, while the neuroretinal rim appears intact and healthy (*Figure 15*).

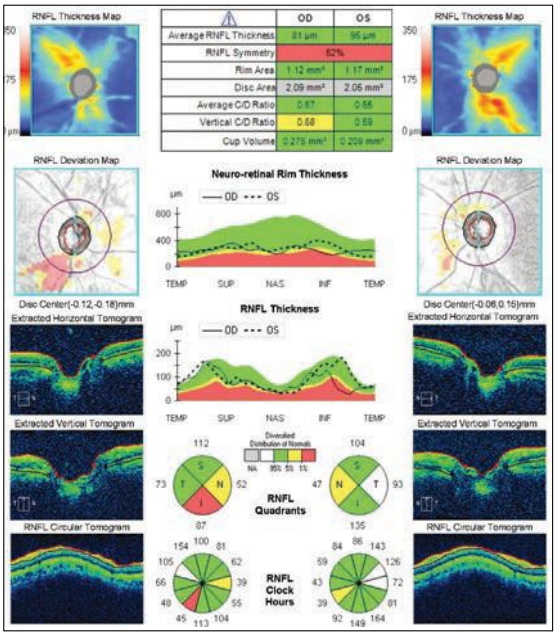
Examining the left eye RNFL thickness map for signs of glaucoma damage, some diffuse superior thinning to the left eye is evident, while the ganglion cell analysis shows a large, deep defect superior nasal to the macula, an odd location for glaucoma damage (*Figures 16 and 17*). The left eye visual field shows a dense inferior arcuate defect with temporal loss superiorly (*Figure 18*), also an atypical location for glaucoma. The right eye was normal (not shown).

Initially, this clinical history and information appears highly suspicious for glaucoma, but at closer examination of the left eye disc photo, there is diffuse chorioretinal atrophy involving the disc and macular regions. Further questioning revealed a history of a gunshot wound to the head that penetrated the globe in 2005 (funny how the patient did not report that at first!). Following surgical globe repair and a lensectomy, the patient did astoundingly well, but he was left with the dense field loss in the left eye. Now with elevated IOP, the diagnosis is somewhat complicated and we have to sort out the diagnostic testing data.









Clockwise from upper left:

Fig. 11. Repeatable superior arcuate defect OD with minimal progression from baseline (upper left).

Figs. 12 and 13. OCT shows inferior wedge defect and overall decreased average RNFL thickness that was stable from 2017 (upper right) to 2021 (lower right).

Fig. 14. Drance heme in a 47-year-old Asian female with NTG and IOP of 9mm Hg (lower left).

In this case, the OCT findings and visual fields don't correlate with glaucomatous loss but rather with the evidence of the chorioretinal atrophy following ocular trauma. Therefore, it is most likely that the patient is only at an ocular hypertensive stage (as can best be determined). Remember, just because there is visual field loss, don't assume that it's always from glaucoma. We treated this patient with a prostaglandin analog, lowering the IOP to around 20mm Hg, and will monitor his OCT and visual field closely for any glaucomatous loss.

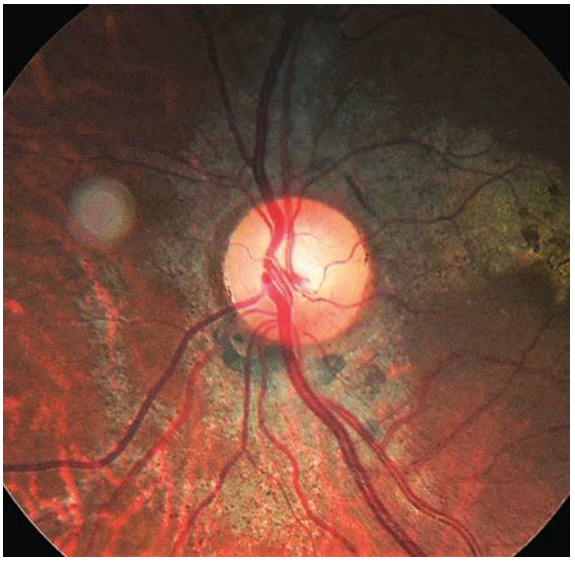


Fig. 15. Disc OS appears healthy, but notable peripapillary atrophy is seen on dilated exam.

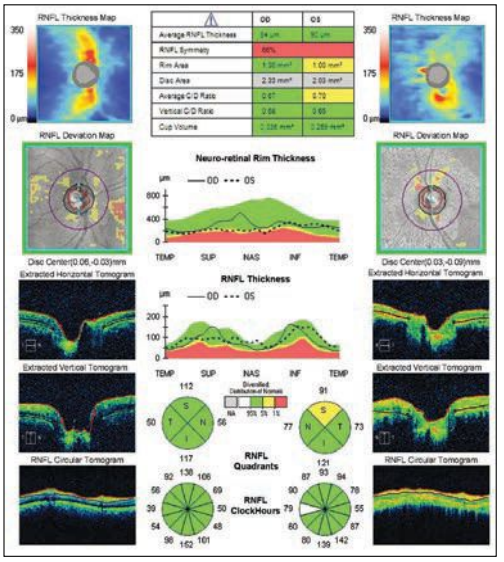


Fig. 16. OCT scan of the RNFL shows mild superior RNFL thinning OS.

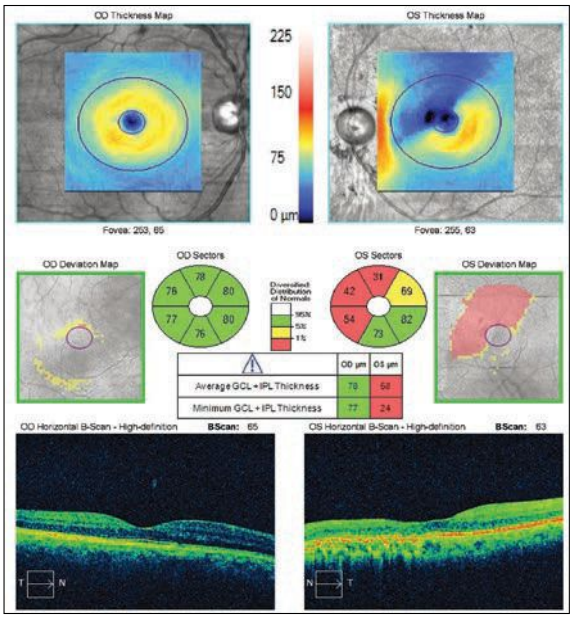


Fig. 17. Ganglion cell analysis shows marked superior thinning of ganglion cell complex at the left macula.

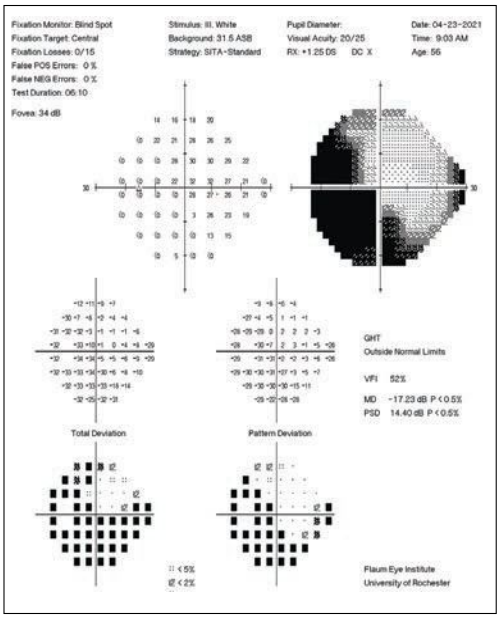


Fig. 18. Visual field OS shows dense temporal loss extending into inferior arcuate.

Red and Green Disease

OCT is often a crucial diagnostic tool when differentiating early glaucoma from ocular hypertension in suspects where nerves look somewhat suspicious on exam. In some cases, however, the RNFL and/or the ganglion cell analysis leads us astray. In this example, we have another high myope (-9D) with a family history of glaucoma and Goldmann tonometry readings of 18mm Hg OD and OS (*Figure 19*). Reviewing the RNFL and GCL+ probability analyses might suggest a glaucoma diagnosis due to the prominent red areas of nasal thinning of both discs (right eye shown in *Figure 20*).

This intuitively should raise a red flag in the examiner's mind, as the nasal nerve head appears normal and healthy in the photos and we know glaucomatous damage does not typically start in the nasal aspect of the optic nerve. The visual field provided also doesn't correlate with these findings; rather, it is clear and relatively reliable OU (although slightly high false positive rate of 16% OD) with the GHT within normal limits (*Figure 21*).

What happened here? The color coding on the OCT probability maps are off because of the tilted discs and fall outside of the reference database, potentially leading to an erroneous diagnosis of glaucoma. Of note, OCT reference databases can be improved upon, including for higher myopia.[8] Unfortunately, the time and cost of FDA-mandated clinical trials make it less likely for companies to perform them.

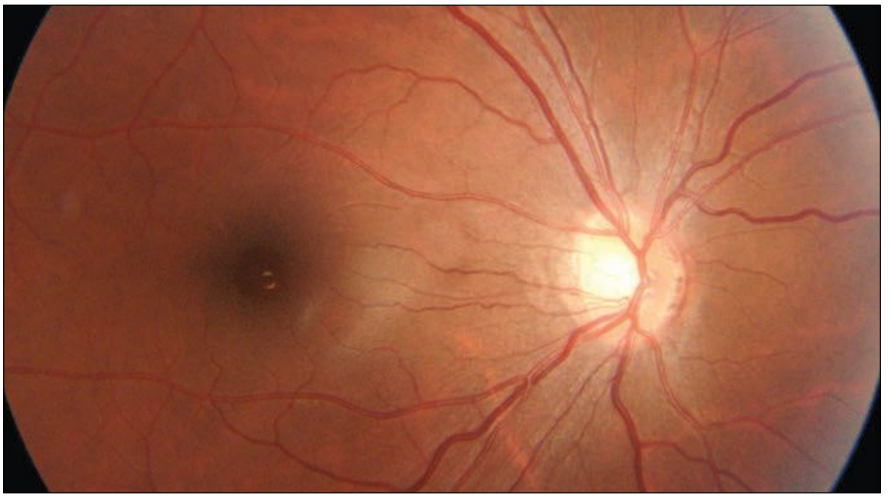


Fig. 19. Disc photo of high myope with large, indistinct cup.

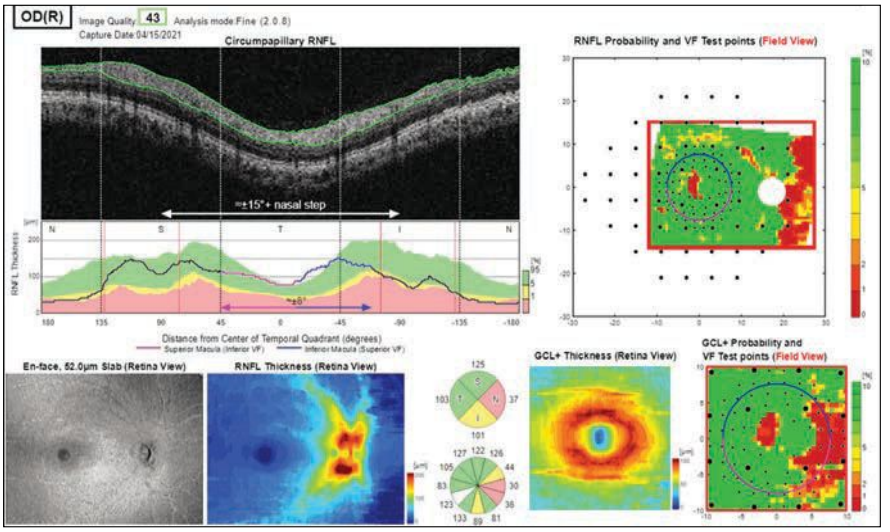


Fig. 20. RNFL analysis shows "red disease," non-glaucomatous abnormality to the nasal disc area.

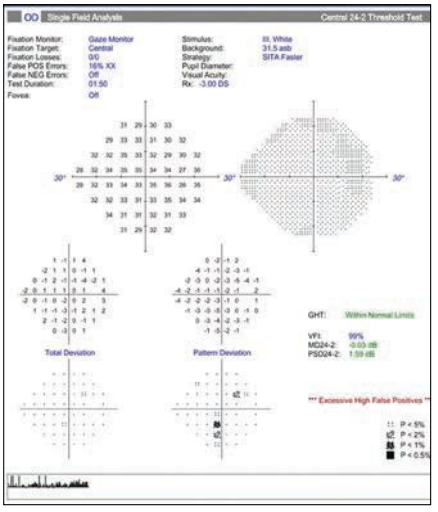
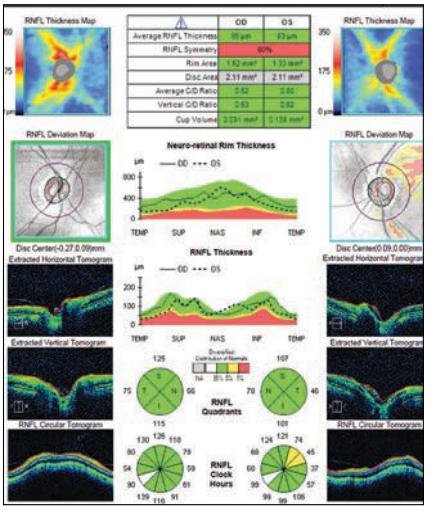
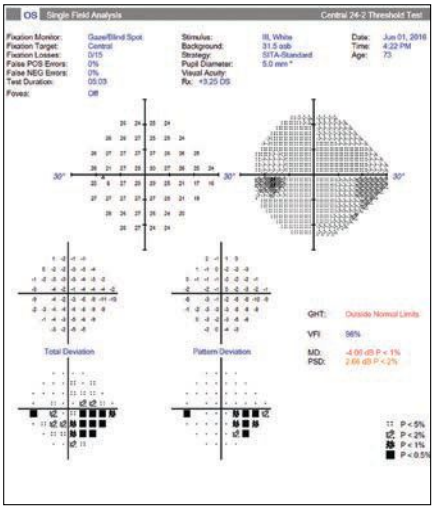


Fig. 21. Visual field of the same eye as Fig. 20 is clear and relatively reliable, and the GHT is within normal limits.





Figs. 22 and 23. So-called "green disease." Average RNFL thickness value is color-coded green and within normal range per the reference database. However, the OCT report shows a superior temporal focal defect and the visual field shows a correlating defect.

In a similar way, the presence of so-called "green disease" on an OCT report is another source of misdiagnosis.

Occasionally, the OCT data obtained on a patient shows no signs of abnormality at quick glace; the average RNFL thickness is "normal" and the four quadrants' sectors are all in the green zone as well (*Figures 22 and 23*). However, when more closely inspected, we see that there are focal sectors of RNFL superior temporal thinning in the left eye, which is often seen in early disease. In addition, there is notable asymmetry between the "average RNFL thickness" values, in the left eye much lower at 83µm as compared to the right eye at 96µm.

Statistical averages for diffuse vs. focal loss can "trick" the reference database coding system—be careful. As much as we can be fooled by "red disease" into treating, we can be falsely reassured by an average RNFL value in the green and overlook a clear case of glaucoma.

Advanced Glaucoma: The "Floor Effect"

OCT retinal nerve fiber layer imaging technology has had an amazing impact on our ability to diagnose glaucoma at earlier stages of the disease.

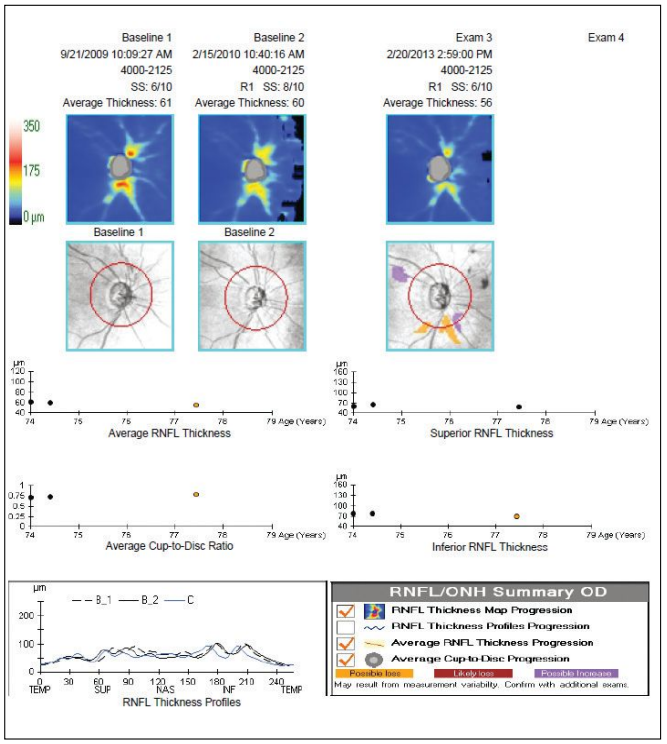


Fig. 24. RNFL OCT demonstrating the "floor effect."

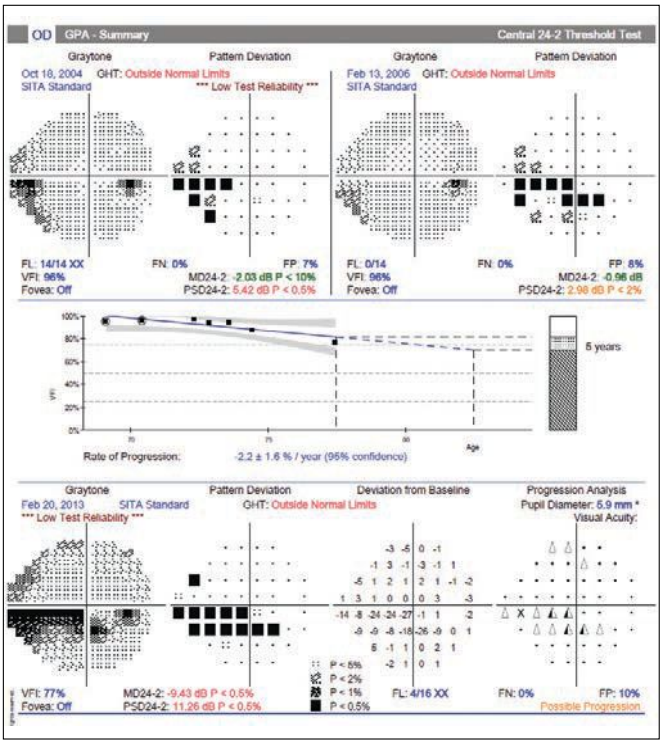


Fig. 25. Visual field progression analysis showing advancing loss.

On the flip side, there comes a time at the moderate-to-advanced stages of disease when the RNFL analysis at the optic nerve head becomes of limited to no value in monitoring a patient for progression.

When significant RNFL loss has occurred, a bottom "floor" is reached where the nerve fiber layer will not show further thinning and OCT progression analysis is unable to detect further loss. This happens around 50µm to 60µm of average thickness. Values don't go below 50µm (and never to zero) due to the residual retinal glial tissue and blood vessels.[9]

The important note here is that at this stage of disease the patient's visual fields can be quite useful in monitoring for progression. The case example shows right eye RNFL loss with approximately 61µm to 56µm of average thickness and minimal change over four years. Future OCT scans would not show much more thinning and thus cannot be used to track disease change. However, the corresponding visual field shows a much earlier relative stage of damage (mean deviation = -2dB) and continues to progress over a seven-year period (mean deviation = -11.2dB) (*Figures 24 and 25*).

The bottom line is to rely more on the OCT/RNFL for progression analysis at the early stages of disease and use the visual fields at the middle and later stages. Note, you'll need a series of regular (and reliable) field tests to do this, so don't put them off during the early stages.

A Matter of Perspective

In cases where a glaucoma workup—and the responsibility it entails to "make the call" correctly—may feel intimidating, it is important to keep things in proper perspective. The tools we use today in clinical practice are far more powerful and precise than they have ever been. OCT alone has been nothing short of revolutionary, but while many optometrists practicing today got good training on the devices initially, keeping up with all the new advances is challenging.

There will always be gray areas when the diagnosis isn't clear-cut, but these are getting smaller and smaller all the time. Your own clinical instincts will always be your best tool, no matter what set of findings are arrayed in front of you at the time. ■

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UNDERSTANDING ONH DYNAMICS IN GLAUCOMA AND BEYOND

Clinicians must know what a healthy optic nerve head looks like and how it functions before they can appreciate the dysfunction that happens with disease.

BY CATHERINE HOGAN, OD, AND ANDREW RIXON, OD
MEMPHIS

Glaucoma is the most common optic neuropathy found in adulthood, and the most prevalent form—primary open-angle glaucoma (POAG)—is projected to affect 7.32 million individuals in the United States by 2050.[1] This estimation is staggering and should encourage ODs to not become complacent or make assumptions about glaucoma diagnosis, especially as the condition is a diagnosis of exclusion.

A litany of non-glaucomatous entities can also cause optic nerve dysfunction, and although this fact confounds our job as clinicians, understanding fundamental facets of the optic nerve and employing a systematic diagnostic approach will help guide us to the truth, ultimately maximizing the patient experience.

This review will discuss the pathophysiology and clinical assessment of common optic nerve head (ONH) disorders with a practical emphasis on their differentiating features.

A Systematic Approach

Clinically, a detailed and systematic stereoscopic analysis of the ONH and its surrounding tissue using the slit lamp and various condensing lenses provides framework for the assessment. Although quantitative variables are important, attention to the qualitative aspects and morphology of the ONH should be the main point of emphasis, as there is considerable quantitative overlap between normal and unhealthy ONHs.[5]

Understanding how to evaluate the glaucomatous disc is key to differentiating the disease. One of the most repeatable systems you can follow for guidance is the 'five Rs' developed as part of FORGE (Focusing Ophthalmology on Reframing Glaucoma Evaluation): (1) observing the scleral *ring* to identify the boundaries of the optic disc and determine its size (larger vertical disc diameters will naturally have larger cups), (2) assessing the *retinal nerve fiber layer* (RNFL) for localized or diffuse loss, (3) examining the neuroretinal *rim* while specifically evaluating the width, shape and color in all sectors with the premise that on average the disc will follow the ISNT rule and will not have focal tissue loss, (4) checking for *retinal or disc hemorrhages*, which often straddle the rim but can be found at the level of the lamina or in the peripapillary retina and (5) evaluating for a *region of peripapillary atrophy* (PPA) of chorioretinal tissue particularly adjacent to areas where the neuroretinal rim is thinner.[5,6] An additional glaucoma feature to be on the lookout for is deep or laminar cupping created by tissue remodeling and subsequent excavation beneath the scleral canal.[7-9]

Employing a systematic method can help minimize diagnostic errors. Studies have shown that general ophthalmologists are twice as likely to underestimate glaucoma compared

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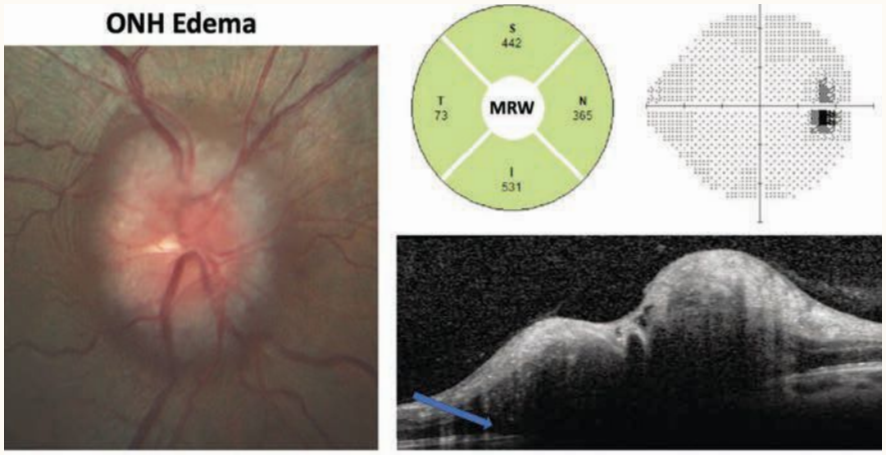

This IIH patient has an opening ICP of 42cm H_2O. Fundus photography shows Frisen grade 3 edema. Note the substantially increased MRW, enlarged blind spot on the grayscale plot and the OCT cross-section showing subretinal hyporeflective space (blue arrow) consistent with ONH edema.

with glaucoma subspecialists. ONHs most likely to be underestimated were horizontally ovoid in shape and those with moderate PPA. Factors associated with underestimation were failure to correctly grade vertical cup-to-disc (C/D) ratios, assess disc hemorrhages and capture RNFL and rim loss.

ONHs most likely to be identified as glaucomatous when they were not were large and had nerves with horizontal or vertical disc tilt, among other factors.[10] Researchers found that in a group of optometric observers, average-sized nerves were correctly identified 90% of the time. However, large ONHs tended to be misidentified as glaucomatous, and even more problematically, small ONHs associated with glaucoma were misidentified as normal, reinforcing the size bias during ONH evaluation that we all need to remain aware of.[11]

These findings may also be influenced by the lack of structured funduscopic evaluation. A study monitoring the gaze behavior of trainees and experts when assessing ONHs found that trainees, although they spent more time evaluating ONHs, exhibited no specific gaze pattern regardless of whether the ONHs had suspicious findings or not and mainly concentrated on the optic cup. Experts, however, took less time and were more systematic and consistent in their approach, concentrating on areas of interest (rim tissue, RNFL and superior and inferior poles) and suspicious structural findings as they presented.[12]

How does optic nerve damage occur in glaucoma? At present, we know that a number of factors influence the retinal ganglion cells and ONH. These include intraocular pressure (IOP), cerebrospinal fluid pressure (CSFP) and ocular perfusion pressure (OPP).[13] The complex interrelations among these factors result in glaucomatous optic neuropathy. Although there is not currently a universal consensus of the exact relationship between these factors, there is a characteristic pattern of glaucomatous ONH damage that is known, helping to more clearly delineate glaucoma from other ONH disorders.

In essence, glaucoma is a laminopathy, whereas other optic neuropathies are not. The aforementioned term "cupping" can create confusion as it can be conflated with C/D ratio. In fact, cupping has been proposed to involve two components: prelaminar and laminar.[8] Prelaminar cupping affects prelaminar neural tissue, and progressive loss of that tissue will increase the depth and the width of the cup, notably increasing the C/D ratio.

The following is where the distinction between non-glaucoma and glaucomatous cupping occurs.

Release Date: July 15, 2021

Expiration Date: July 15, 2024

Estimated Time to Complete Activity: 2 hours

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

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

- Discuss the pathophysiology of optic nerve head disorders.
- Explain how the optic nerve head functions.
- Identify potentially suspicious optic nerve pathology.
- Diagnose various optic nerve head disorders.

Target Audience: This activity is intended for optometrists engaged in eye care of the optic nerve head.

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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Multiple *in vivo* and cadaveric studies have shown that what separates glaucomatous ONH damage from other neuropathies is the involvement of both connective tissue and neural tissue. Glaucomatous optic nerves undergo progressive posterior bowing/displacement, disorganization and deformation of the lamina cribrosa and peripapillary scleral connective tissue, in addition to the loss of retinal ganglion cell axons.[7-9,14] This results in the laminar or deep cupping consistent with glaucoma.

Clinical Differentiation

Recognizing the morphologic changes found in glaucoma provides insight for differentiating the disease from other ONH disorders. These differential diagnoses can range from anatomical optic nerve disorders that clinically confound the observer to various neuropathies that cause optic nerve damage via other pathophysiological pathways. Using a combination of detailed clinical examination and ancillary testing will allow the optometric physician to make an informed and accurate diagnosis, treatment and management plan for the patient.[9] This all begins with differentiating glaucoma from its masqueraders, some of which we cover here:

Large optic disc. At first glance, a large optic disc with a correlating large cup commonly leads to glaucoma misdiagnosis. Looking for glaucomatous features instead of zeroing in on cup size alone is an important shift in the process.[15] First, consider the patient's demographics. Males and African Americans are more likely to have anatomically larger discs.[16] Measure the vertical height of the optic disc during funduscopy, and apply the correction factor for magnification per the dioptric power of the lens used. For instance, a Volk Super 66D lens or digital 1.0x offers a 1.0 correction factor compared with the Volk 90D lens' correction factor of 1.3.[17,18] Large disc height will suggest large disc area, a presentation which can be misdiagnosed as glaucoma.[5] Disc area values are populated and confirmed by most OCT modalities.[19]

Compare quadrants of rim tissue to better recognize how in a large disc, even with a large cup, the rim tissue can be intact. Assess the optic nerve and arcades using the red-free filter to better highlight the RNFL and confirm the lack of wedge defects, a progressive neuropathic disease finding wherein focal RNFL damage extends from the optic disc and into the papillomacular bundle.[20]

Look within the cup to assess the lamina cribrosa; normal cups not under IOP stressors may have visible laminar dots but should not have laminar deformation/reconfiguration. The clinical cupping associated with glaucoma involves permanent and progressive prelaminar thinning and laminar deformation as a result of IOP stress. This pathophysiologic damage over time is absent in anatomically large healthy discs.[9]

Gauging stability and confirming there is no progressive loss of rim tissue when dealing with anatomically large discs can be accomplished with multiple technologies. RNFL thickness, ganglion cell analysis (GCA) and visual field results will remain stable over time in patients with large healthy discs. However, RNFL thickness will be increased in superior, inferior and global quadrants in large discs.[21] Spectral-domain (SD) RNFL analysis will also confirm disc area and compare the measured area (in mm^2) to a database of normative values. One study used confocal scanning laser ophthalmoscopy (CSLO) and SD-OCT to define a large disc area as greater than $2.43mm^2$.[15]

Analysis of the rim area also provides contrast between anatomically large discs and glaucoma. Using CSLO, it has been found that progressive glaucomatous optic nerves experience global rim area loss 3.7 times faster than large healthy optic nerves.[22] OCT analysis of Bruch's membrane opening-minimum rim width (BMO-MRW) measures the minimum distance from the BMO to the internal limiting membrane, allowing for more accurate assessment of the neuroretinal rim regard-

A Closer Look at the ONH

Recognizing what an ONH looks like and how it functions allows us to better discriminate between normal and abnormal findings, thus unlocking the ability to more specifically diagnose diseases of the ONH. Functionally, the optic nerve transmits stimuli between the neurosensory retina and the lateral geniculate body and cerebral visual pathways. The ONH refers to the ophthalmoscopically visible anterior surface of the nerve.

The ONH consists primarily of four components: neural fibers, glial cells, extracellular matrix support and vascular tissue.[2-4] The neural tissue is formed by the retinal ganglion cell axons of the RNFL, originating primarily in the macula and converging radially on the ONH in arcuate bundles, forming a convex periphery and the first layer of the ONH, the surface nerve fiber layer. This neural tissue appears orange-pink on fundoscopy as a result of the reflectance of light off of the dense capillary network that supplies the disk surface. These fibers then bend abruptly, passing through the second layer—the prelaminar layer—which is made up of loose, capillary-containing glial tissue. The previously mentioned fiber bend creates the central and concave cup, which appears yellow as a result of the supporting collagenous fibers found in the third layer, the lamina cribrosa region.

The collagenous lamina cribrosa is made up of fenestrated connective tissue lamellae that contain capillaries and are covered in astrocytes. The nerve fiber layer passes through the scleral lamina cribrosa and the fourth layer, the retrolaminar region, prior to exiting the globe.[2,3] The blood supply to the optic nerve is mainly from the posterior ciliary artery, with the exception of the surface of the nerve which receives its supply from retinal circulation. Importantly, ONH blood supply is sectoral in nature.[4] Knowing how the neural and vascular tissue is distributed is critical in recognizing how characteristic patterns of tissue damage can occur in various ONH disorders.

less of disc size.[15,23] It is reported that larger disc areas physiologically have larger BMO areas, which contributes to thinner but stable BMO-MRW results. Therefore, guided progression analysis of RNFL and BMO-MRW is paramount to classify stability vs. progression over time.[15,24]

Tilted disc syndrome (TDS). This congenital condition is another anomalous optic nerve condition. It arises from malclosure of the embryonic optic fissure during the second month of gestation. This presents clinically with inferonasally rotated optic discs in tandem with juxtapapillary changes and retinal vascular abnormalities, including adjacent inferonasal crescent or PPA, retinal pigment epithelium and choroidal thinning, posterior staphyloma and situs inversus. The optic disc rotation is non-progressive but can impair the clinician's ability to visualize the true boundaries of the rim tissue and C/D ratio. The anatomical insertion may lead to a sense of sectoral cupping, but physiologically TDS does not exemplify rim thinning or laminar deformation.[9,25,26]

OCT sheds light on what can be clinically confounding when differentiating TDS from glaucoma. RNFL results associated with TDS depend on optic nerve morphology and torsion degree but typically show false RNFL thinning in nasal quadrants, which are stable over time on glaucoma progression analysis (GPA).

GCA and BMO-MRW provide better specificity than RNFL for TDS evaluation. GCA has more uniformity, with less confounding results due to anatomy, and shows intact, stable ganglion cell structure over time. BMO-MRW adjusts for individual ONH rim tissue orientation and in TDS, reveals intact, stable neuroretinal rim tissue over time.[27]

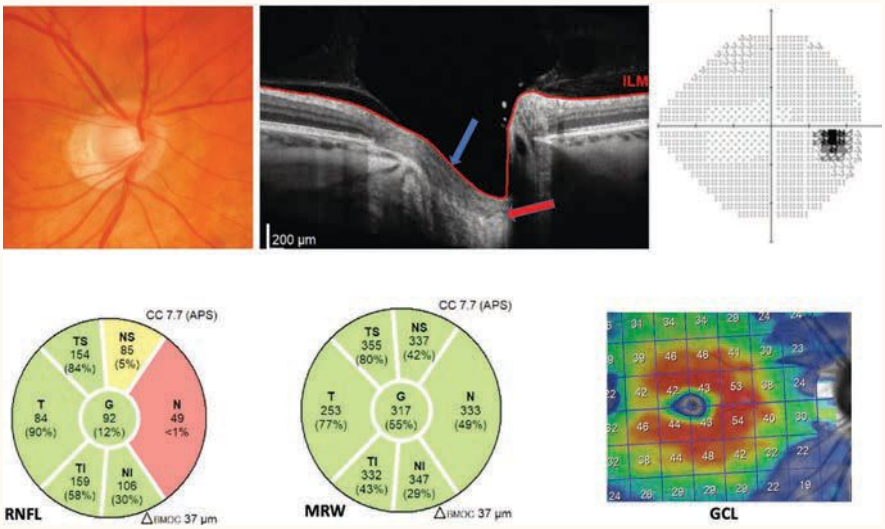
Visual field defects in TDS are most commonly found in the superior temporal quadrant (recall that inferonasal is the most common disc rotation), with reports of up to 33%.[28,29] These defects are non-progressive and do not necessarily respect the horizontal meridian, emphasizing how TDS physiology spares the nerve fiber layer and lamina cribrosa of thinning.[26,28,29]

Pseudopapilledema and ONH drusen (ONHD). The term pseudopapilledema encompasses clinical variations of anomalous ONH elevation, blurring or an indistinct margin appearance in the confirmed absence of RNFL edema. Congenital anomalies including tilted and malinserted discs, hypoplasia, myelinated nerve fiber layers, Bergmeister's papillae and ONHD may create the clinical appearance of an indistinct optic nerve, leading the primary and emergent differential diagnosis to be true optic disc edema.[30]

Pseudopapilledema impairs complete visualization of rim tissue. Serial fundus photography, visual field testing, GCA and OCT with BMO-MRW are valuable tools that, when assessed altogether, provide better visualization of the anomalous optic nerve structure and a baseline determination of function.[27] If GPA signals progress over time, the clinician will be able to identify glaucomatous patterns of change that may have been otherwise masked by the anomalous clinical appearance.[30,31]

ONHD is considered a subtype of pseudopapilledema and presents with acellular deposits consisting of calcium, amino and nucleic acids and mucopolysaccharides that form in the prelaminar ONH.[32] Caucasians and patients with a family history of ONHD have a higher prevalence, and the majority of ONHD cases are bilateral.[30,33] Researchers reported the best supported understanding of ONHD formation by using histological studies to determine the presence of abnormal axonal metabolism, leading to axonal disruption, extrusion of mitochondria into the prelaminar extracellular space and dysregulation of calcium deposition.[32,33]

Another study determined the angioarchitecture in ONHD differs from the normal optic nerve. The authors report that embryologic development of optic nerve vasculature is disturbed early on by the presence of ONHD, creating risk of vascular flow impedance at the level of the central retinal artery and its arterial bifurcations within the optic disc. Thus, while ONHD is considered a benign condition, there may actually be risk of vascular obstruction at the optic nerve, inducing chronic hypoxia and long-term RNFL damage.[33,34]



This -7.00D myope was previously classified as a glaucoma suspect. Note the malinsertion and intact pre-laminar tissue (blue arrow) found on the ONH cross-section. The lamina is marked by the red arrow. Additionally, the MRW is unremarkable and the nasal RNFL tissue is thin secondary to temporalized vasculature. Both the ganglion cell layer and the visual field are unremarkable.

TABLE 1. FACTORS ASSOCIATED WITH DIFFERENT ONH ABNORMALITIES

	OAG	NAION	AAION
ONH Morphology	Excavation causing deep cupping, laminar remodeling, localized or diffuse RNFL thinning, adjacent PPA	Ischemia causing sectoral RNFL atrophy without excavation, deep cupping or laminar remodeling	Inflammatory ischemia causing diffuse RNFL atrophy with shallow cupping and without laminar remodeling
Chronic Disease ONH Appearance	Progressive and significant deep cupping, potential for mild rim pallor	Stable, mild to severe sectoral > diffuse pallor, minimal or absent non-progressive sectoral shallow cupping	Stable, severe diffuse pallor with non-progressive shallow cupping
OCT RNFL	Progressive RNFL thinning correlating to areas of rim thinning that commonly involve the vertical quadrants initially, may approach "floor" values with chronic and unmanaged disease	Acute phase: sectoral > diffuse RNFL edema Chronic phase: non-progressive sectoral to diffuse RNFL atrophy nearing "floor" values	Acute phase: diffuse RNFL edema Chronic phase: non-progressive diffuse RNFL atrophy, typically severe at "floor" values
OCT GCA	Wedge or "squeegee" GCL defect with early disease that may progress to arcuate, altitudinal or gutted patterns of loss with chronic and unmanaged disease	Altitudinal gutting of IPL and GCL that is commonly superior, identifiable in acute and chronic phase, non-progressive	Generalized, diffuse gutting of GCA, identifiable in acute and chronic phase, non-progressive
Humphrey Visual Field	Correlates to pattern of GCL loss that is more significant with chronic disease, nasal step or early arcuate are common initially with progression in chronic and unmanaged disease	Altitudinal defect correlating to GCA results that is commonly inferior, identifiable in acute and chronic phase, non-progressive	Generalized depression, non-progressive

ONHD and glaucoma may present concurrently. Therefore, the first step in clinical evaluation is a thorough examination of the optic nerve, its vasculature and the surrounding RNFL. The more superficial the ONHD, the more "lumpy-bumpy" the disc margin, with a nodular, yellow-to-gray papillary color. Be aware that the peripapillary retinal vessels are never obscured in benign ONHD—an important distinction from papilledema. The indistinct or elevated disc margins caused by ONHD can mask the appearance of glaucomatous cupping, making funduscopic examination challenging. As a result, assessment for wedge defects with the red-free filter may be the best funduscopic clue for concurrent glaucoma.[30,33]

Confirmation of ONHD is a necessity, as misdiagnosis may burden the patient with costly over-management and needless treatment. Recognizing the presence of buried and/or superficial ONHD with appropriate technological work-up will not only rule out emergent diagnoses like papilledema but will also assist in the identification of non-glaucomatous vs. glaucomatous patterns. Fundus autofluorescence (FAF) of the optic nerve will show hyper-autofluorescent, superficial, calcified drusenoid bodies. FAF sensitivity to detect buried ONHD is reported to be as low as 12% to 18%.[33]

B-scan ultrasonography, the traditional gold standard for ONHD identification, will reveal hyper-echoic drusenoid bodies within the optic nerve, but it also has a lower detection rate for buried drusen, likely because they are less calcified.[35] So, the clinician may need enhanced-depth imaging OCT (EDI-OCT) or swept-source OCT (SS-OCT) of the optic nerve to visualize buried drusen.[33,36] EDI-OCT and SS-OCT have been suggested as the new gold standard for visualizing superficial and buried ONHD, which appear as hyporeflective bodies with surrounding hyperreflective borders.[36]

With confirmation of ONHD, the next step is to rule out neuropathic or glaucomatous damage. RNFL thinning is typically localized to the area with greatest ONHD aggregation. The nasal quadrant is the most likely affected section, and RNFL thinning stabilizes as the superficial movement of ONHD concludes. Progressive RNFL thinning over time, especially in the inferior and superior quadrants, may signal glaucomatous rim thinning.

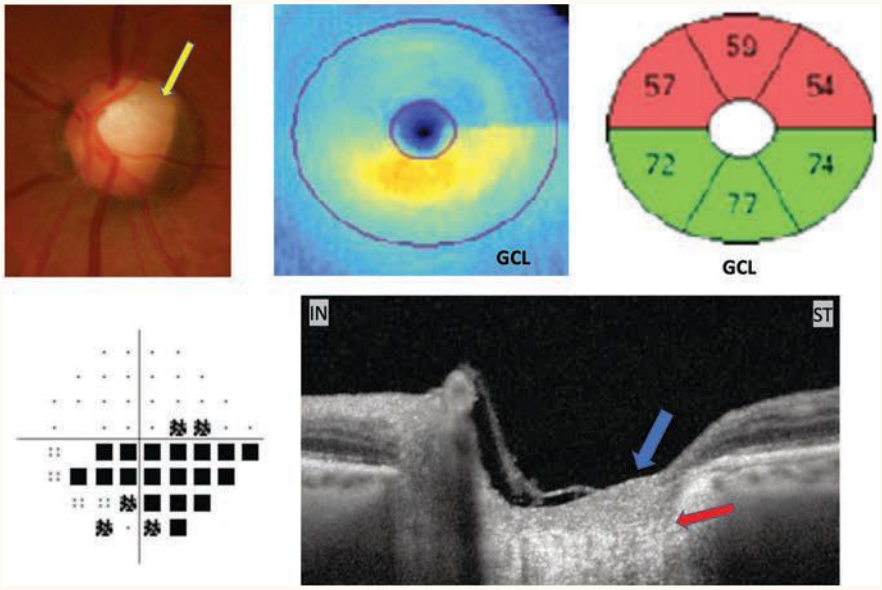
Monitoring the ganglion cell layer (GCL) is the most sensitive measure to examine for glaucomatous neuroretinal damage, as benign ONHD will not cause ganglion cell deficits.[33] Visual field results may be normal (48%), but defects can occur, particularly in superficial ONHD, with varying patterns including enlarged blind spot (30%) and incomplete arcuate or concentric narrowing defect (22%).[33,37] The slow, progressive nature of visual field defects in ONHD is debated, with etiologies of mechanical compression or vascular compromise currently under investigation.[33,36]

While ONHD pathophysiology differs from that of glaucoma, the ONHD structure-function relationship may involve ONH hypoperfusion and symptomatic visual deficiencies. Currently, there is no standard treatment for ONHD. Observation over time is preferred with the understanding that elevated IOP with progressive RNFL and GCL loss or visual field defects may signal concurrent glaucoma and thus warrant the initiation of IOP regulation.[33]

Papilledema. True papilledema is bilateral optic disc edema due to increased CSFP and subsequent increased intracranial pressure (ICP). It presents initially with indistinct, hyperemic and elevated rim tissue, potentially with vessel obscuration, hemorrhages, negative spontaneous venous pulsation and Paton's lines. Papilledema has various systemic etiologies, including compressive brain tumors, venous sinus thrombosis and idiopathic intracranial hypertension (IIH). Confirmation of papilledema from funduscopic and technological examination requires emergent intervention.[38]

Elevated CSFP in the optic nerve sheath will directly influence laminar position via increased translaminar pressure difference, which produces a net posterior force on the surface of the lamina and a hydrostatic pressure gradient within the prelaminar and laminar tissues. If unmanaged, this mechanical insult will lead to optic nerve swelling from axoplasmic flow stasis, peripapillary expansion and deformation and globe flattening. When ICP is decreased with appropriate treatment (*e.g.*, weight loss, oral acetazolamide, lumbar puncture, shunt surgery), the CSFP within the optic nerve sheath will also decline. Timely management of these cases is critical, as chronic edema can lead to irreversible damage.[39]

Differentiating papilledema from glaucoma comes into play clinically when managing the long-term effects of papilledema, which include structural optic nerve and ganglion cell atrophy and functional visual decline. Funduscopically, the optic nerve may appear diffusely pale with no excavation or cupping of the rim tissue. If RNFL atrophy is severe (approaching 40µm in thickness), the optic nerve may have overlying damaged astrocytes, which appear similar to glial tissue and may obscure the rim margin.[9,31] Post-edema RNFL values vary depending on the chronicity and severity of the papilledema, with some reports of a return to normal ranges and others of mild to drastic atrophy.



This patient was previously treated for moderate primary OAG OU. They exhibit superior temporal sectoral pallor (yellow arrow), hemispheric ganglion cell loss, inferior visual field defect and intact prelaminar (blue arrow) and laminar tissue (red arrow) shown on OCT through the superior temporal rim. This is consistent with AION, not glaucoma.

OCT can be very helpful in these cases, with certain parameters offering more information than others. One study determined elevated ICP is associated with increased ONH volume and decreased Bruch's membrane displacement volume. It found that with a decrease in ICP (after lumbar puncture) comes a decrease in ONH volume and in the ganglion cell complex volume, reinforcing how retinal atrophy is also a consequence of optic disc edema. Also of note was the stability of cup volume before and after lumbar puncture, which supports the funduscopic presentation of optic nerve atrophy without cupping.[38]

Other research examined IIH patients who were treated with acetazolamide or surgical intervention and discovered that ganglion cell loss occurred on average about two months after initial diagnosis, despite treatment, which correlated to the extent of final visual field loss and mean deviation reduction even up to one-year post-treatment. Thus, from a clinical standpoint, although visual field assessment has been considered the gold standard for monitoring functional loss in papilledema, doctors should now consider serial GCA to aid in detection of earlier patterns of neuropathic disease.[39]

Ischemic optic neuropathies. Anterior ischemic optic neuropathies (AION) initially present with optic disc edema from swelling of the RNFL and are classified as non-arteritic (NAION) or arteritic (AAION) based on presentation and etiology. NAION is the most frequent cause of unilateral optic disc edema in ages 45 to 70 and is characterized by a hyperemic, edematous nerve with flame hemorrhages and arteriolar narrowing that causes sudden, painless vision loss.[40]

AAION is an ocular emergency which arises from giant cell arteritis, a systemic vasculitis of medium- and large-sized vessels, with variable systemic symptoms such as jaw claudication and temporal headache. AAION presents with a chalky-white, edematous optic disc, with hemorrhages and severe, sudden, painless vision loss. If acute AAION is suspected, emergent intervention is necessary to reduce the risk of progression to bilateral blindness.[41]

Ischemic optic nerve damage is not governed by IOP-related tissue stress like glaucoma, but instead from

a hypoxic state from insufficient posterior ciliary artery circulation, which cascades retinal ganglion cell axonal damage to axonal death. Systemic or nocturnal hypotension and systemic vascular diseases such as diabetes and hypertension may initiate NAION optic nerve non-perfusion.[9,40,42]

NAION RNFL damage is greatest in superior disc sectors, especially the nasal and temporal sectors. This is likely explained by more tightly packed axons and thus higher oxygen demand, leading to a more rapid hypoxic state in these sectors.[43] AAION pathophysiology involves inflammatory occlusion of the short posterior ciliary arteries at the ONH in response to granulomatous vasculitis.[41]

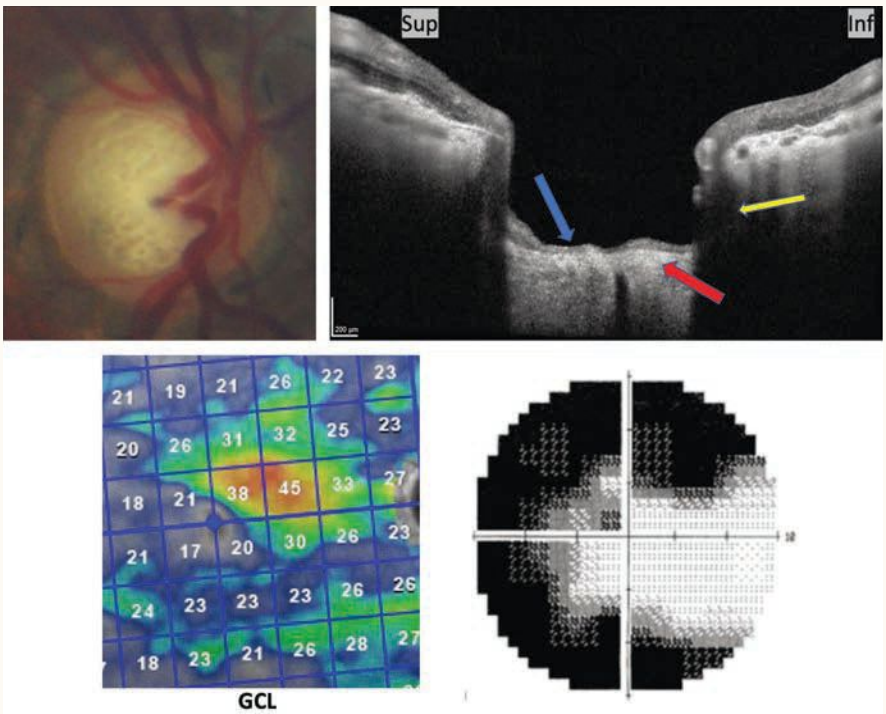
Regardless of etiology, ischemic insult to the optic nerve will induce pallor and RNFL atrophy without rim tissue excavation or laminar deformation.[9] This is clinically observed as "pallor greater than cupping," distinguishing the aftermath of ischemic insult from glaucoma.[42-44] Researchers examined end-stage AION for the presence of cupping and determined 2% of NAION eyes experienced cupping vs. sectoral pallor (72%) or diffuse pallor (28%), while 92% of AAION eyes experienced cupping.[43]

Optic nerve excavation may be lacking in NAION because the lamina cribrosa is spared from ischemic insult.[42] Further topographical investigation with scanning laser ophthalmoscopy shows a difference in ischemic vs. glaucomatous cupping; neither NAION nor AAION experience deep cupping like glaucoma. Research found glaucomatous cup volume was seven times greater than NAION and two times greater than AAION, confirming that while cup size in end-stage AAION and glaucoma may appear similar, it is the glaucomatous nerve experiencing progressive posterior excavation and laminar deformation.[44]

RNFL edema can vary in severity but is more significant and diffuse in AAION than NAION. As the ischemic insult enters its chronic phase (>six weeks), RNFL edema begins resolving and RNFL atrophy occurs. One study determined that a majority of acute NAION patients experienced superior-quadrant RNFL thickening, with RNFL thinning in chronic NAION predominantly occurring in the superior and temporal quadrants, with significant atrophy progressing for up to six months before plateauing. Compared with glaucoma, this RNFL atrophy is rapid and matches the clinical presentation of defined disc pallor, even approaching OCT "floor" micron values.

Acute and chronic NAION will present with characteristic GCA altitudinal loss, most commonly superior and identifiable before optic disc edema resolution.[45] Segmenting out the macular layers shows ganglion cell loss occurring as early as two weeks post-initial presentation, with progressive inner retinal thinning occurring until it levels out at around three months.[45] When comparing acute with chronic NAION, the internal plexiform layer (IPL) thinning was the most significant of the macular layers.[40,45]

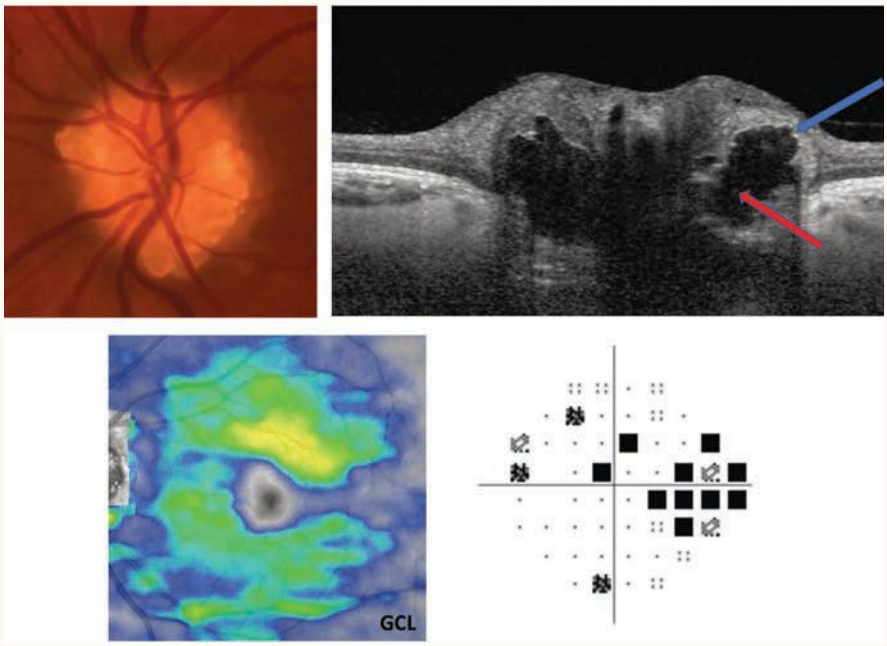
This is in direct contrast to glaucoma where less abrupt GCL thinning occurs, IPL thinning is less severe and macular RNFL thinning mimics optic disc RNFL loss.[46] Ischemic inner retinal loss corresponds functionally with altitudinal visual field loss, commonly inferior, another hallmark finding of acute and chronic NAION.[40,47] Generalized GCA and visual field depression is typically experienced in AAION; be mindful that a patient with severe vision loss >20/200 from either ischemic condition may not have the visual capacity to perform an accurate visual field test.[40]



This patient has advanced glaucoma. Note the posterior and sub-scleral excavation (yellow arrow) of prelaminar tissues (blue arrow) and lamina thinning (red arrow) on vertical OCT cross-section consistent with glaucoma. Advanced GCL loss and corresponding 10-2 grayscale are included.

Takeaways

A host of non-glaucomatous optic nerve conditions either confound examination or create a clinical appearance of glaucomatous mimicry. Taking a systematic clinical and technological approach, while addressing each patient's care on an individual basis, will help differentiate optic nerve conditions and reduce patient burden. ■



Pictured here is ONH drusen. Note the hyper-reflective borders (blue arrow) and hyporeflective core of the drusen (red arrow) on EDI-OCT cross-section. A myriad of presentations can occur with both visual fields and GCL. This patient's GCL is stable, and Humphrey visual fields show a partial arcuate defect, which is a common field result.

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OPTOMETRIC STUDY CENTER QUIZ

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1. What quadrants of OCT RNFL thickness may be increased in anatomically large optic nerves?
a. Superior and temporal.
b. Superior and inferior.
c. Inferior and temporal.
d. Temporal and nasal.

2. What ancillary tests are largely unaffected by tilted disc insertion, making them excellent options for concurrent glaucoma assessment?
a. BMO-MRW and GCA.
b. BMO-MRW and RNFL.
c. OCT RNFL and visual field.
d. OCT RNFL and GCA.

3. All of the following are pseudopapilledema conditions except _____.
a. Malinserted discs.
b. Optic nerve head drusen (ONHD).
c. Idiopathic intracranial hypertension (IIH).
d. Optic nerve hypoplasia.

4. Which ancillary testing best allows the clinician to identify buried ONHD?
a. EDI-OCT.
b. B-scan.
c. FAF.
d. OCT RNFL.

5. What are potential long-term effects of unmanaged papilledema?
a. Optic nerve excavation or cupping.
b. Optic nerve and ganglion cell atrophy.
c. Optic nerve atrophy with intact ganglion cells.
d. Increased ICP leading to chronic increased IOP.

6. What funduscopic presentation describes chronic NAAION?
a. Cupping > pallor.
b. Sectoral deep cupping.
c. Laminar deformation.
d. Sectoral pallor > cupping.

7. Which statement regarding cupping in chronic AAION is true?
a. Cupping does not occur in chronic AAION.
b. The cupping is shallow and non-progressive.
c. The cupping is deep from posterior excavation.
d. The excavation mimics glaucoma, which is why the cup volume is the same in AAION and glaucoma.

8. When comparing OCT RNFL results in NAAION and glaucoma, which statement is true?
a. RNFL loss is more rapid in glaucoma than NAAION.
b. Acute NAAION will present with RNFL atrophy that mimics glaucomatous damage.
c. Chronic NAAION commonly involves superior temporal RNFL thinning, which may rapidly approach "floor" values quicker than glaucoma.
d. RNFL loss is more rapid in NAAION than glaucoma, usually reaching "floor" values during the acute phase.

9. What optic neuropathy and GCA result is most accurate?
a. Glaucoma, early wedge or "squeegee" GCL loss.
b. NAAION, diffuse GCL loss.
c. AAION, inferior altitudinal IPL > GCL loss.
d. NAAION, inferior arcuate GCL > IPL loss.

10. Which ancillary testing result is a hallmark finding in NAAION?
a. GCL loss that is more significant than IPL loss on GCA.
b. OCT RNFL atrophy predominantly involving the nasal quadrant.
c. Sectoral OCT RNFL atrophy occurring rapidly before development of GCA atrophy.
d. Non-progressive, inferior altitudinal defect on visual field.

11. The most common form of optic neuropathy found in adulthood is _____.
a. Leber's optic neuropathy.
b. NAAION.
c. AAION.
d. Glaucoma.

12. The optic nerve head transmits stimuli between the neurosensory retina and the _____.
a. Pons.
b. Medulla.
c. Pituitary gland.
d. Lateral geniculate body.

13. The ONH primarily consists of how many components?
a. Two.
b. Four.
c. Six.
d. Eight.

14. The blood supply of the optic nerve comes mainly from _____.
a. Posterior ciliary artery.
b. Central retinal artery.
c. Vortex veins.
d. Cilioretinal artery.

15. Which of the following is not one of the five Rs?
a. Scleral ring.
b. RNFL.
c. Disc hemorrhage.
d. Disc color.

16. Optic disc hemorrhages can be found in all of these locations except _____.
a. Straddling the neuroretinal rim.
b. At the level of the lamina.
c. In the peripapillary retina.
d. Subfoveally.

17. Unlike in other optic neuropathies, tissue lost in glaucoma includes both _____.
a. Vascular and muscular.
b. Neural and connective.
c. Neural and muscular.
d. Muscular and connective.

18. Cupping in glaucoma is _____.
a. Shallow and involves prelaminar tissue.
b. Shallow and creates pallor.
c. Deep and involves excavation of lamina.
d. Deep and involves only prelaminar tissue.

19. All of the following are factors that affect the optic nerve in glaucoma, except _____.
a. IOP.
b. CSFP.
c. OPP.
d. Atmospheric pressure.

20. Glaucomatous optic nerves that were most likely to be misidentified as normal were _____.
a. Large nerves with large cups.
b. Average nerves with average cups.
c. Large nerves with small cups.
d. Small nerves with moderate cups.

Examination Answer Sheet

Understanding ONH Dynamics in Glaucoma and Beyond
Valid for credit through July 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.

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

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Answers to CE exam:

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

Post-activity evaluation questions:

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

21. Discuss the pathophysiology of optic nerve head disorders. ① ② ③ ④ ⑤
22. Explain how the optic nerve head functions. ① ② ③ ④ ⑤
23. Identify potentially suspicious optic nerve pathology. ① ② ③ ④ ⑤
24. Diagnose various optic nerve head disorders. 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
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31. The content was evidence-based.
32. The content was balanced and free of bias. ① ② ③ ④ ⑤
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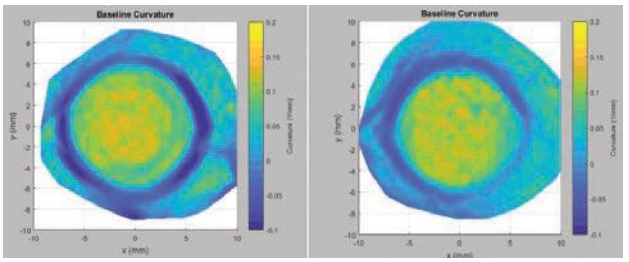
By submitting this answer sheet, I certify that I have read the lesson in its entirety and completed the self-assessment exam personally based on the material presented. I have not obtained the answers to this exam by any fraudulent or improper means.

Signature _____ Date _____ Lesson 121427 RO-OSC-0721

Shape-shifter

Monitoring scleral topography changes over time helps ensure a consistently optimized fit.

Q How significant is scleral shape change over time in scleral lens wearers? Will these patients eventually require lens parameter changes?



Curvature maps of a patient's sclera just after (left) soft disposable lens removal and 24 hours later.

A "The shape of the sclera has become an area of increasing interest with the growing popularity of scleral lenses," suggests Jason Jedlicka, OD, associate professor at the Indiana University School of Optometry and chief of the school's Cornea and Contact Lens Service. He notes that instrumentation can now measure the sclera in the hopes of achieving better alignment between the lens and the eye. This has raised the question of whether the shape of the sclera changes over time and, if so, if a lens refit is necessary to maintain an optimized fit.

Culprits

While more scientific literature is discussing the shape of the sclera, very little covers changes in this shape, according to Dr. Jedlicka. He says one key consideration to keep in mind when thinking of scleral shape is the overlying conjunctiva, which appears far more vulnerable to short-term changes in thickness, leading to variation in scleral imaging. So, when considering scleral shape change and its relevance to lens fitting, think of the shape of the underlying sclera and that of the overlying conjunctiva as two separate aspects.

Conjunctival thickness can change in the short term due to compression from contact lens wear, ocular surface inflammation and edema, among other reasons. Conjunctival changes can alter scleral topography, even if the underlying shape of the sclera remains stable.

The conjunctiva can be compressed and thinned due to both scleral and soft lens wear. This compression seems to resolve itself when contact lenses are discontinued for 24 to 48 hours depending on how the lens is fitted and how long it has been worn. The conjunctiva can also thicken due to edema, and this is often seen in corneal gas permeable wearers who show 3 and 9 o'clock staining and injection.

In the case of conjunctival thickness changes, to acquire accurate scleral shape imaging for the purpose of lens fitting, lens wear should be discontinued and consideration should be given to treating the ocular surface with topical lubricants or anti-inflammatories to improve conjunctival health.

Scleral shape can also change with corneal ectatic disease. Anatomically, the sclera and the cornea comprise one continuous layer of collagen that has different arrangements of fibers, so the idea that altering the structural stability of the cornea might have a ripple effect on the sclera seems logical.

The Scleral Shape Study Group published a review of 227 eyes with corneal ectasia vs. 115 eyes with normal corneas. The researchers found that the patients with corneal ectasia had more inferior steepening of the sclera with a quadrant-specific effect than healthy controls. While not directly proven by this study, the results seem to suggest that as patients with corneal ectasia progress, their scleral shape can change just as their corneal shape does.[1]

In addition, there is case-based evidence that collagen crosslinking, which strengthens an ectatic cornea and often creates a degree of flattening, can alter the scleral shape profile as well.[2] Therefore, it is reasonable to suspect that disease processes that alter corneal shape by weakening the tissue or procedures that help to strengthen it can also impact the shape of the sclera.

Takeaways

Scleral lenses rest on the conjunctiva, the shape and thickness of which vary noticeably, potentially even day by day. The conjunctiva overlies the sclera, the shape of which changes very little but can be altered over time. Combined scleral and conjunctival shape changes in the short and long term can impact the fit of scleral lenses. Addressing conjunctival health and shape during the fitting period and monitoring for scleral shape changes over time, particularly in the inferior quadrant, allows your scleral lens patients to maintain an optimal fit from the start and enjoy the best experience and outcome possible. ■

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About Dr. Shovlin

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

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REVIEW Education GROUP

Black and Blue and Red All Over

This rare subconjunctival hemorrhage formation required a tailored approach to stop the bleeding.

BY GRIFFIN CHRISTENSON, OD, AND LEONID SKORIN, JR., DO, OD, MS
FOREST GROVE, OR, AND ROCHESTER, MN

A 64-year-old female presented one day after receiving a 1.25mg intravitreal injection of bevacizumab (Avastin) in her right eye. The patient had developed a small subconjunctival hemorrhage and a "blue patch" on her right lower eyelid shortly after the procedure. The patch had reportedly enlarged throughout the previous day and now encompassed both the right lower and upper eyelids.

The patient's eye was red and swollen and tender to the touch. She stated her vision was blurry, noting intermittent double vision while watching television. She said dabbing her eye drew blood.

Ocular history included exudative age-related macular degeneration with choroidal neovascularization in both eyes, for which she was receiving intravitreal anti-VEGF injections every eight weeks. Additionally, the patient had grade 2+ nuclear sclerotic cataracts in both eyes. Her medical history was positive for polycythemia vera, protein S deficiency, erythema nodosum and type 2 diabetes mellitus. The patient was placed on a 10mg daily regimen of the direct oral anticoagulant (DOAC) medication Xarelto (rivaroxaban, Janssen Pharmaceuticals) for her protein S deficiency. Additionally, she was taking 3mg of prednisone orally for recent-onset pyoderma gangrenosum and receiving insulin injections of Lantus (Sanofi) 14 units daily and NovoLog (Novo Nordisk) 6 units with meals to manage her diabetes. Her polycythemia vera was treated with phlebotomies as needed.

Fig. 1. Ecchymosis of the upper and lower eyelids. Note the blood mixed in with the tears in the medial canthal area.

The patient's entering distance visual acuities were 20/60 OD and 20/40+ OS, with no improvement on pinhole testing. Her visual acuity had been 20/25 OD during the previous day's examination. She did not have a relative afferent pupillary defect, nor did she report pain upon eye movement, although she did have some difficulty moving her right eye.

Upon examination of the anterior segment, the patient's right eye displayed 2+ to 3+ ecchymosis of both the right upper and lower eyelids (*Figure 1*). The patient had a 3+ subconjunctival hemorrhage with persistent leakage of blood, and blood was visualized in the patient's tear meniscus (*Figure 2*). Her right eye was not proptotic. There were no signs of anterior chamber reaction, hypopyon, hyphema or infection, and intraocular pressures were within normal limits. Examination of the posterior segment, as well as of the left eye, was unremarkable.

Diagnosis

The examination was directed to differentiate between a severe anterior subconjunctival hemorrhage and a retrobulbar hemorrhage (RBH), which is an ocular emergency that poses a significant threat to vision.[1] Findings prompting suspicion of RBH include sudden onset of severe pain, proptosis, ophthalmoplegia, deterioration in visual acuity, subconjunctival hemorrhage, increased intraocular pressure and pupil abnormalities such as a relative afferent pupillary defect.[2,3]

Salient factors that helped rule out a diagnosis of RBH in this instance included normal intraocular pressure and the absence of pupil abnormalities, proptosis and pain on eye movement. The decreased visual acuity and intermittent diplopia was explained by the persistent blood in the tear lake and mild restriction of eye movement by the protruding subconjunctival hemorrhage. Furthermore, the location of the previous day's injection, paired with the visualization of the continued hemorrhaging, allowed for localization of the source of bleeding.

Anti-VEGF injections are performed at a relatively anterior location, 3mm to 4mm posterior to

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.

the limbus, so as to enter the pars plana of the ciliary body.[4] Conversely, the most common causes of RBH include retrobulbar anesthesia and blunt force trauma, neither of which our patient experienced.[5]

Treatment for acute RBH requires the release of pressure from within the orbit.[1] The transcutaneous transseptal orbital decompression approach allows for drainage of an orbital hematoma, while lateral canthotomy and inferior cantholysis allow for orbital decompression but do not evacuate pooled blood from within the orbit.[1]

Discussion

Subconjunctival hemorrhages are typically benign and self-limiting conditions that produce minimal symptomology. When seen in non-anticoagulated patients, intervention is usually not required.[6] Acute subconjunctival hematoma formation, as seen with our patient, is a rare occurrence.

Anticoagulation is indicated for treatment and prevention of thromboembolytic events for patients in a hypercoagulable state (such as protein S deficiency).[7] For nearly 70 years, heparin and vitamin K antagonists, such as Coumadin (warfarin, Bristol-Myers Squibb), have been mainstay pharmacological agents used for these patients.[7] While the benefits of these therapies have been well established in a wide array of thromboembolic disorders, limitations such as a narrow therapeutic range, food and other drug interactions and frequent laboratory monitoring of the international normalized ratio (INR) have encouraged efforts to develop more targeted therapies.[8] More recently, DOACs have risen to prominence with a purported goal of offsetting some of the limitations of warfarin and heparin (*Table 1*).

Currently, there is no FDA-approved equivalent to the INR for measuring the anticoagulant effect of DOACs. Qualitative coagulation assays such as activated partial thromboplastin time, thrombin time and prothrombin time are insufficient to assess the degree of anticoagulant effect as seen with INR for management of warfarin therapy.[8] Quantitative measures such as anti-factor Xa level, plasma drug concentration, dilute thrombin time and ecarin thrombin time are able to directly assess anticoagulation effects.[8]

Unfortunately, standardized therapeutic ranges for DOACs have not been established and quantitative test results have not been correlated with clinical outcomes.[8] If a patient taking Xarelto or Eliquis (apixaban, Pfizer) experiences uncontrolled bleeding, the anticoagulant antidote Andexxa (andexanet alfa, Portola Pharmaceuticals) can be administered.[9] Andexxa has been shown to rapidly reverse the anticoagulant effects of both of these medications.[9] Andexxa may also be effective in reversing the anticoagulant effect of other direct and indirect factor Xa inhibitors.[9]

Outcome

Due to the anterior location of the subconjunctival hemorrhage, a pressure patch was placed over the patient's right eye in an attempt to tamponade the bleeding. The patient was instructed to leave the patch in place for three hours before removing it to determine if the bleeding had ceased. After the appropriate amount of time had passed, the patient called to inform us that the bleeding had been controlled. She was instructed to use cold compresses to reduce swelling and keep her head elevated while sleeping. ■

Table 1. Currently Available DOAC Agents

Generic Name	Trade Name	Mechanism of Action
rivaroxaban	Xarelto	Direct factor Xa inhibitor
apixaban	Eliquis	Direct factor Xa inhibitor
edoxaban	Lixiana	Direct factor Xa inhibitor
betrixaban	Bevyxxa	Direct factor Xa inhibitor
dabigatran	Pradaxa	Direct thrombin (factor IIa inhibitor)

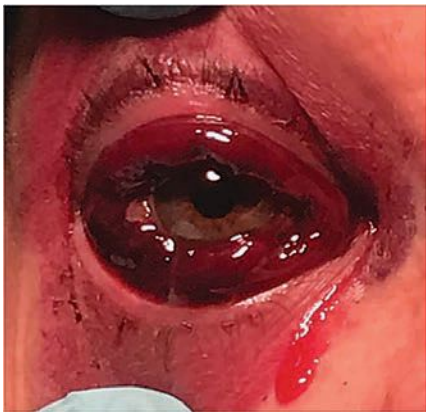

Fig. 2. Extensive protruding subconjunctival hemorrhage.

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Dr. Skorin is a consultant in the Department of Surgery, Community Division of Ophthalmology at the Mayo Clinic Health System in Albert Lea and Austin, MN. He has no financial interests to disclose.

BY BISANT A. LABIB, OD

Encircling the Arcus

While this finding usually shouldn't raise alarm, be aware of atypical presentations that could lead to disastrous consequences.

Corneal arcus is a regularly encountered condition on routine ophthalmic examination, as it is one of the most common ocular manifestations of aging. While this ubiquitous finding is most often benign, there are a few circumstances in which the practitioner should pause and consider a potentially life-threatening systemic etiology. In order to distinguish between these possibilities, it is necessary to understand what comprises a normal case of corneal arcus and how to identify atypical manifestations.

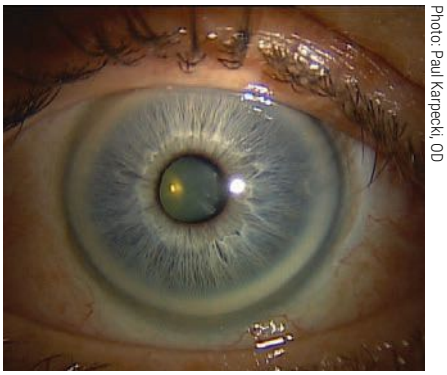


Photo: Paul Karpecki, OD

Age-related deposition of cholesterol and other lipids in the stroma occurs in response to increased permeability of the limbal blood vessels.

Squaring the Circle

Corneal arcus appears as a bilateral, white or grey opacification encircling the cornea. There is a 1mm clear zone known as the lucid interval of Vogt between the opacification and the limbus that distinguishes the condition from other peripheral corneal degenerations.[1,2] Corneal arcus usually begins to form around 6 or 12 o'clock and spreads until it comprises a full circle.[2] Histopathological studies have reported that corneal arcus is an accumulation of cholesterol, phospholipids and triglycerides from the systemic circulation that is then deposited near the limbus at the level of the stroma, Bowman's membrane and Descemet's membrane.[1,3,4]

It is unclear why the lipid deposition is limited to a circular peripheral ring around the cornea. It could be that due to the cornea's avascularity, the limbal blood vessels deposit material only on the outer regions. It has also been speculated that the temperature gradient of the cornea affects how lipids are deposited. Additionally, the precise arrangement of collagen and molecules within the corneal layers prohibits the movement of lipids into the center of the cornea.[2]

In elderly patients, corneal arcus is usually benign, without visual consequences or ocular complications. It is more commonly found in men than women and is also thicker and denser in appearance in men.[1] Its incidence rises with age due to the increase in permeability of the limbal blood vessels with aging, allowing for easier lipid deposition from the bloodstream.[4]

As the condition is associated with normal aging and usually not considered pathologic in the elderly population, it may be indicative of abnormal lipid metabolism if identified in non-elderly patients.[5] In the event that a middle-aged or younger patient presents with corneal arcus, studies have indicated a potential correlation with systemic hypercholesterolemia, specifically low-density lipoprotein build-up, and coronary artery disease.[1]

Corneal arcus arising in patients under 50 years of age, also known as arcus juvenilis, is a marker for lipid dysfunction and often yields chronically elevated serum cholesterol. It is estimated that males younger than 50 with arcus have a 6.4x higher risk of developing cardiovascular disease-related mortality. Treatment with a statin to lower cholesterol levels has been shown to result in a significant decrease in risk, though corneal findings remain unchanged.[5] One study suggested a possible correlation between arcus and alcoholism in males under 60 years of age.[6]

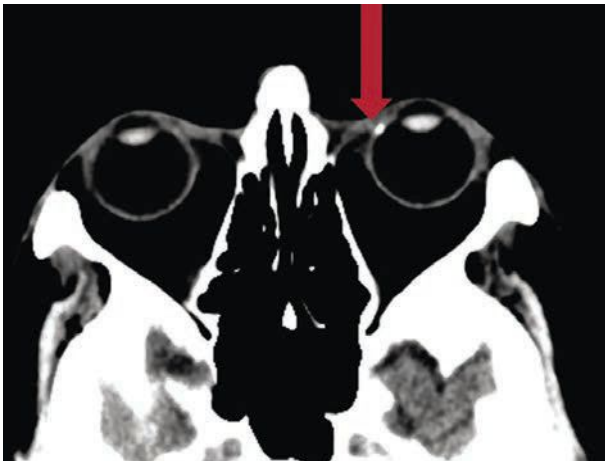
In rare cases, arcus may be a unilateral finding. Some studies report that this is due to severe carotid occlusive disease, with arcus presenting only in the contralateral eye. This suggests that in the event there is a large, unilateral carotid plaque, the lack of blood flow prohibits arcus formation in the ipsilateral eye, while it is not restricted in the contralateral eye with patent carotid.[7]

The presence of arcus can have ocular implications as well as the aforementioned systemic risks. Ocular conditions such as chronic hypotony may also lead to a unilateral or asymmetric presentation of arcus. In this case, however, the arcus is present or more pronounced in the affected eye. This is likely due to the fact that the inflammatory process associated with the breakdown seen in ocular hypotony leads to increased vessel permeability and increased likelihood of lipid deposition from the bloodstream.[8]

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.

Studies have considered the effect of arcus on the overall corneal composition and how it may affect the structure as it relates to measuring intraocular pressure (IOP) and hysteresis. It has been suggested that corneal hysteresis and resistance is lower in eyes with arcus when compared with eyes without.[9] Furthermore, a study evaluating central corneal thickness (CCT) and IOP measurements demonstrated that, on average, eyes with arcus had lower CCT and higher IOP but not glaucoma. It is unclear what the cause of these associations is, but this finding suggests that the presence of arcus has an effect on the overall biochemical properties of the cornea.[10]



Corneal arcus appears hyper-dense on CT scans (red arrow).

Takeaways

For most optometrists, there is rarely a clinical day that goes by where corneal arcus is not an encountered condition among our patients. Although it is most often a completely benign, age-related finding, all ODs should be aware of its atypical variants, as they may be more ominous. It is necessary to recognize these as the first signs of what could be a serious underlying health condition that requires intervention. ■

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Slit-Lamp Camera Captures Sharper Images

Attaching a camera to your slit lamp helps document pathology and aids patient education. To get the best resolution possible, Telscreen says the newest version of its EyeRes device closes the gap between digital and human image resolution.

The EyeRes Diamond system captures 137 million pixels per square inch, not far off from the peak color resolution of a human eye, according to the manufacturer, and a 3.3x gain in resolution over the company's previous top-of-the-line system, the EyeRes Platinum. Telscreen also notes that the system will be simple to use, promoting efficiency in the exam room. Just step on a foot pedal, perform a normal slit lamp exam and the system will produce clear, high-resolution images. The company also offers training on the principles of slit lamp photography, according to the company's website.

Like Telscreen's other slit lamp cameras, the EyeRes Diamond will allow you to share and store images and videos of pathologies, and its new image sensor might pick up conditions that are invisible on other devices, according to the company's website.

Fundus Cameras Elevate Imaging

Two new tabletop fundus cameras from Optomed, called Halo and Polaris, create crisp, high-res photos that can be easily stored or shared, according to the company.

Both devices can perform non-mydriatic fundus imaging with a 45-degree field of view through pupils as small as 4mm, company literature notes. Both also make use of a 12MP image sensor, include 10 fixation targets and use eye tracking for alignment and autofocus for image acquisition. Imaging options for the devices include color and red-free photography, built-in cup-to-disc measurement and the ability to take anterior segment photos, says Optomed.

The Halo (above) and Polaris (right).

The chief differences concern portability and operation. The smaller and more lightweight Halo has no on-board monitor; instead, the practice connects it to a Windows 10 PC. A tech with a laptop can be located up to 25 feet away from the patient during imaging, which can take place through a glass wall, a company press release explains. These social distancing capabilities could help you and your patients feel safer during appointments. The Polaris, slightly larger and heavier, has a built-in 10-inch touch screen display, according to product literature.

Halo and Polaris are DICOM compatible and offer various connectivity options for image sharing and integration with EHR. Optomed says the cameras require less operating time and minimal training compared with other devices for easier integration into practice workflows.

▶ CONTACT LENSES

Bausch + Lomb Extends Multifocal Power Range

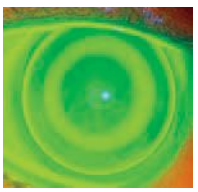
Presbyopic patients with higher-than-usual astigmatism have had to rely on custom-fit contact lenses if they wanted to try that modality—until now. Bausch + Lomb recently expanded the cylinder powers on its Ultra Multifocal for Astigmatism, adding powers of -2.25D and -2.75D to the range.

For some of the estimated 32 million people worldwide with both astigmatism and presbyopia, this means you may now be able to more easily offer contact lenses that accommodate their vision needs. The lenses offer the convenience of same-day in-office fitting during the initial exam.

Cylinder power parameters now include -0.75, -1.25, -1.75, -2.25 and -2.75 options. The other parameters remain unchanged: a sphere range of -6.00D to +4.00D with low and high add power varieties.

The lenses are made of samfilcon A material and have a base curve of 8.6mm and a diameter of 14.5mm.

New Ortho-K Option Debuts



The REMLens, named for its "rapid eye molding" effect while worn overnight, may help treat patients of all ages with low-to-moderate myopic refractive errors, says manufacturer X-Cel Specialty Contacts.

The lens, for use in patients with up to -5.00D sphere and up to -1.50D cylinder, features four independently adjustable fitting zones and five diameter sizes that each have three optic zone options. The edges of the lenses are specifically designed for optimal comfort, says the company. Doctors use an online calculator to store and load topography maps, fluorescein images and videos, reducing chair time on new fits or re-fits, X-Cel says. The calculator allows empirical fitting to ensure a proper and comfortable fit the first time around, according to the company, which could improve both efficiency and patient satisfaction.

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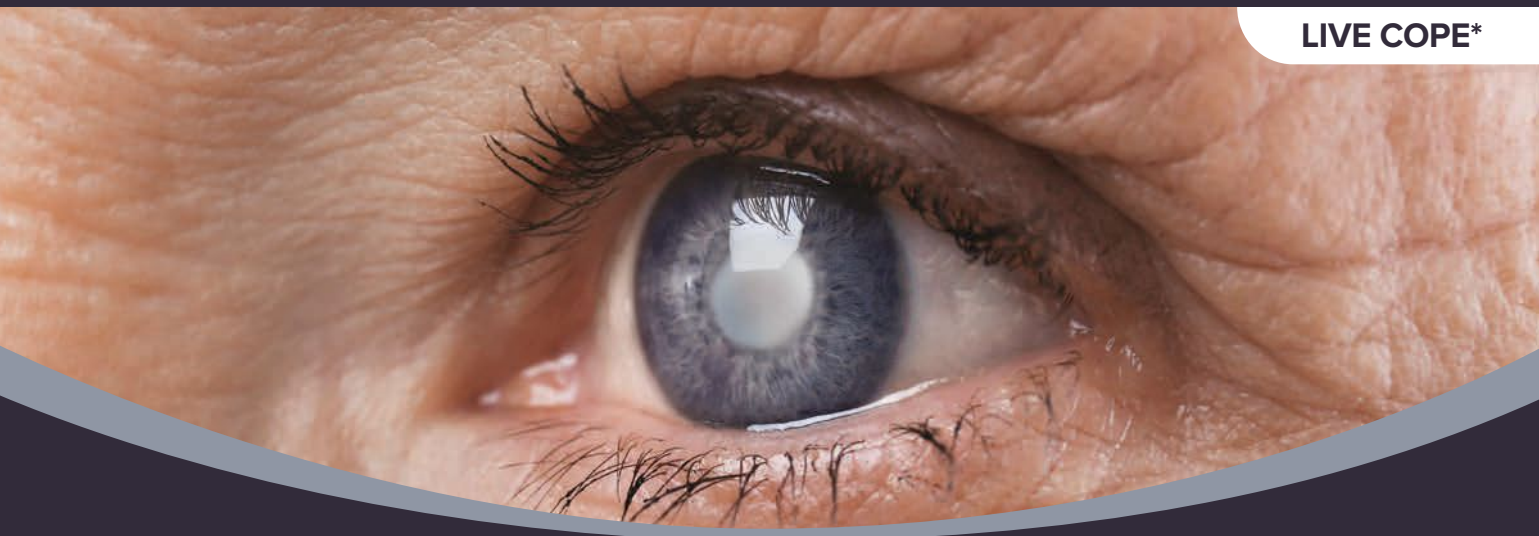

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BY ANDREW S. GURWOOD, OD

DIAGNOSTIC QUIZ

A Well-Red Patient

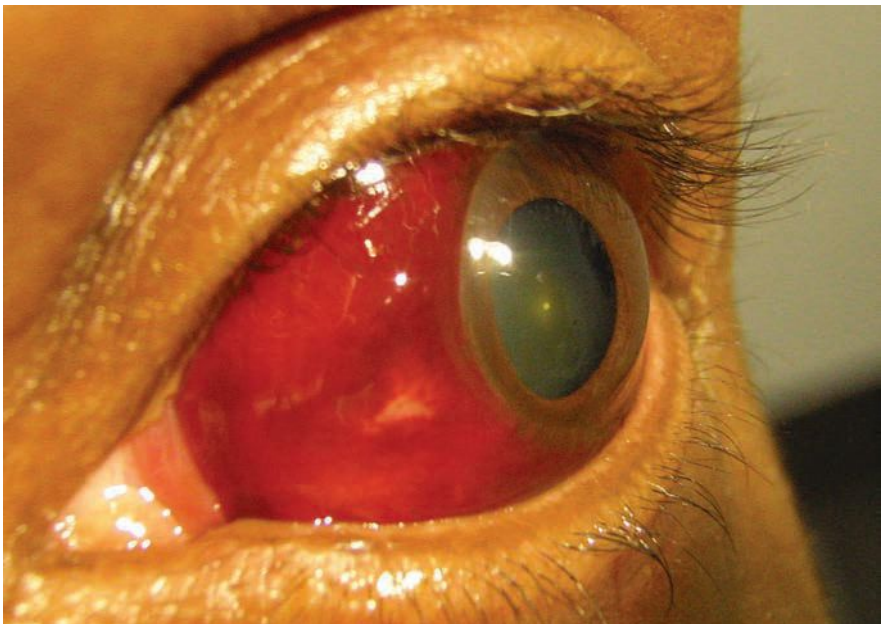
Eliciting a good case history can reveal much about the status of an acute hemorrhage and how you should manage it.

A 34-year-old man presented to the office with a chief complaint of a sudden-onset redness affecting one eye. He said he was not aware of it until someone pointed out to him that "his eye was bleeding."

He did not report any pain or vision loss. He also denied trauma, systemic disease or allergies of any kind.

Diagnostic Data

His best-corrected acuities were 20/20 OD and OS. The pertinent component of his external examination is demonstrated in the photo. There was no evidence of afferent pupillary defect. The remainder of his anterior, middle and posterior segment exam was normal. Goldmann tonometry measured 17mm Hg OU.



The patient was fairly unfazed by his appearance. Should the clinician be as well?

Additional Information

Efforts to obtain more information included asking about possible recent trauma (*e.g.*, sports, car accident), violent coughing, weight lifting, constipation and even any recent episodes of yelling.

In the absence of a mechanical vector elicited through discussion of history, lab work must be considered to rule out systemic diseases that could produce blood coagulopathy or hyperviscosity. Testing might include:

- complete blood count with platelets (CBC with differential and platelets)
- prothrombin time (PTT)
- activated partial thromboplastin time (APTT) and an international ratio (INR) to rule out overanticogulation in patients taking blood thinners
- homocysteine level, particularly in young men
- assay for natural anticoagulants such as protein S and protein C

Repeat events, evidence of additional bruising observed elsewhere or multiple events in a child or young adult must be considered suspicious for a shaking injury.

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. ■

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.

NEXT MONTH IN THE MAG

In August, we present our 45th annual contact lens report. Articles will include:

- How Do the Newest Contact Lens Options Fit In?
- Astigmatic Correction Tips and Tricks
- How to Improve Lens/Tear Interaction
- Contact Lenses for Drug Delivery: What to Expect

Also included in August:

- Comanagement Series: OD-to-OD Referrals
- What to Do When You See Uveitis

BAUSCH + LOMB ULTRA® MULTIFOCAL FOR ASTIGMATISM

FIT FOR SUCCESS

The results are in. Eye Care Professionals gave **Bausch + Lomb ULTRA® Multifocal for Astigmatism** high marks across the board thanks to its easy fitting process.

HERE'S WHAT YOUR PEERS HAD TO SAY ABOUT THIS LENS:

89%

agreed that it is easy to get a **successful** fit during the **first visit** with this lens.*

<5 mins

80% agreed that it took **no more than 5 minutes longer** to fit this lens compared to toric lenses.†

95%

agreed that this lens allows them to **fit patients who have previously dropped out** of contact lenses.‡

SCAN CODE FOR SUCCESS STORIES

*Results of an online survey with Eye Care Professionals who completed an evaluation program for Bausch + Lomb ULTRA® Multifocal for Astigmatism contact lenses (n=219). Survey results include Eye Care Professionals who reported that successful fitting was extremely easy, easy, or somewhat easy (on a 6-point scale) during the first visit, with a margin of error ±4.1%.

†Results of an online survey with Eye Care Professionals who completed an evaluation program for Bausch + Lomb ULTRA® Multifocal for Astigmatism contact lenses (n=219). Survey results include Eye Care Professionals who reported that it took less chair time, no extra chair time, 1-2 minutes of additional chair time, or 3-5 minutes of additional chair time to fit Bausch + Lomb ULTRA® Multifocal for Astigmatism with a margin of error ±5.3%.

‡Results of an online survey with Eye Care Professionals who completed an evaluation program for Bausch + Lomb ULTRA® Multifocal for Astigmatism contact lenses (n=219). Survey results include Eye Care Professionals who strongly agreed, agreed, or slightly agreed (on a 6-point agreement scale) with the surveyed statement, with a margin of error ±2.8%.

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References: 1. Alcon data on file, 2019. **2.** Kern JR, Kappell G, Trinh H, et al. Antimicrobial properties of a novel contact lens disinfecting solution, OPTI-FREE® EverMoist®. *Cont Lens Anterior Eye*. 2011;34(suppl 1):S30. **3.** Alcon data on file, 2010. **4.** Alcon data on file, 2011.

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