Optometry's Role in the Diabetes Epidemic, *p. 32*

REVIEW® OF OPTOMETRY

July 15, 2020

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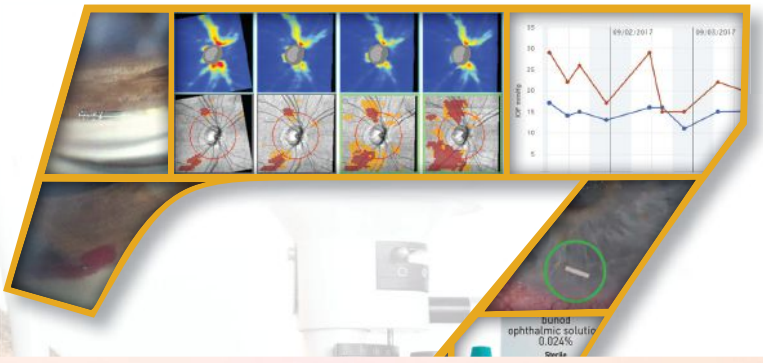
26th Annual Glaucoma Report



Optometry's Role in the Diabetes Epidemic, *p. 32*

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26th Annual Glaucoma Report

7 WAYS GLAUCOMA CARE IS CHANGING

Better drugs, safer surgeries, smarter diagnostics and new approaches are easing the burden on patients—and their ODs, ***p. 54***

Glaucoma: The Perils of Progression, ***p. 38*** • EARN 2 CE CREDITS – A Practical Approach to Angle-closure, ***p. 60***

ALSO: Satisfying the Complicated Presbyope, ***p. 24*** • What's Your Disc Diagnosis?, ***p. 46***

*In some patients with continued daily use. One drop in each eye, twice daily (approximately 12 hours apart).

†Xiidra is an LFA-1 antagonist for the treatment of dry eye disease. Pivotal trial data: The safety and efficacy of Xiidra were assessed in four 12-week, randomized, multicenter, double-masked, vehicle-controlled studies (N=2133). Patients were dosed twice daily. Use of artificial tears was not allowed during the studies. The study endpoints included assessment of signs (based on Inferior fluorescein Corneal Staining Score [ICSS] on a scale of 0 to 4) and symptoms (based on patient-reported Eye Dryness Score [EDS] on a visual analogue scale of 0 to 100).[3]

A larger reduction in EDS favoring Xiidra was observed in all studies at day 42 and day 84. Xiidra reduced symptoms of eye dryness at 2 weeks (based on EDS) compared to vehicle in 2 out of 4 clinical trials. Effects on signs of dry eye disease ICSS (on a scale from 0-4; 0=no staining; 4=coalescent) was recorded at each study visit. At day 84, a larger reduction in inferior corneal staining favoring Xiidra was observed in 3 of the 4 studies.[3]

Indication

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

Important Safety Information

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

Please see Brief Summary of Prescribing Information on adjacent page.

References: 1. U.S. Food and Drug Administration. Code of Federal Regulations, Title 21, Volume 5 (21CFR349). https://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfcfr/CFRSearch.cfm?CFRPart=349&showFR=1. Accessed April 17, 2020. **2.** Jones L, Downie LE, Korb D, et al. TFOS DEWS II Management and Therapy Report. *Ocul Surf.* 2017;15(3):575-628. **3.** Xiidra [prescribing information]. East Hanover, NJ: Novartis Pharmaceuticals Corp; November 2019.

NOVARTIS

Novartis Pharmaceuticals Corporation
One Health Plaza
East Hanover, New Jersey 07936-1080

5/20 XIA-1388813

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

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

1 INDICATIONS AND USAGE

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

4 CONTRAINDICATIONS

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

6 ADVERSE REACTIONS

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

- Hypersensitivity *[see Contraindications (4)]*

6.1 Clinical Trials Experience

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

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

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

6.2 Postmarketing Experience

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

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

8 USE IN SPECIFIC POPULATIONS

8.1 Pregnancy

Risk Summary

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

Data

Animal Data

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

8.2 Lactation

Risk Summary

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

8.4 Pediatric Use

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

8.5 Geriatric Use

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

Manufactured for:
Novartis Pharmaceuticals Corporation
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T2019-110

VOL. 157 NO. 7 ■ JULY 15, 2020

IN THE NEWS

Researchers recently reported that **increased exercise intensity is associated with decreased glaucoma risk**. A team assessed objective exercise intensity based on measurements from accelerometers worn by 1,387 adults over one week. With Rotterdam criteria, participants who spent the day standing or walking vs. sitting had 58% decreased odds of glaucoma, while each 10-minute increase in moderate-to-vigorous activity per day was associated with 38% decreased odds of glaucoma with disc image grading.

Tseng VL, Yu F, Coleman AL. Association between exercise intensity and glaucoma in the National Health and Nutrition Examination Survey. Ophthalmology. June 7, 2020. [Epub ahead of print].

Researchers in Australia found no correlation between quantitative values of vitamin A intake and refractive error. Their data suggests **increased vitamin A intake in childhood does little to stave off myopia in young adulthood**. Although they noted that those with adequate vitamin intake were less likely to have myopia, that association became insignificant after adjusting for cofounders such as ocular sun exposure level, educational level and parental myopia.

Ng FJ, Mackey DA, O'Sullivan TA, et al. Is dietary vitamin A associated with myopia from adolescence to young adulthood? Trans Vis Sci Technol. 2020;9(6):29.

Researchers recently discovered that **glaucoma patients with optic disc hemorrhage (ODH) experience faster visual field progression** than those without. Over an average of 64 months, they found eyes with ODH in two different disc sectors showed worse progression rates than eyes with either ODH in one sector or no hemorrhages at all. Sectors with one hemorrhage experienced a faster visual field progression rate than those with none.

An D, House P, Barry C, et al. Recurrent optic disc hemorrhage and its association with visual field deterioration in glaucoma. Ophthalmol Glaucoma. June 9, 2020. [Epub ahead of print].

Severe Sleep Apnea Leads to Corneal Changes

Intraocular procedures should be done with caution in these individuals.

By Jane Cole, Contributing Editor

Patients with severe obstructive sleep apnea-hypopnea syndrome (OSAHS) may have distinct differences in their endothelia compared with healthy subjects, a team of researchers from Greece suggests. Their study, published in *Cornea*, also found the low level of REM sleep typically seen in these patients may contribute to an increase in corneal thickness.

Specifically, the study found greater pleomorphism and polymegethism in the corneal endothelia of patients with severe OSAHS compared with normal eyes.

The comparative case series examined a total of 190 eyes, which included 102 eyes of patients with severe OSAHS and 88 eyes in the control group.

After a detailed eye exam, the researchers performed specular microscopy in all participants and compared corneal parameters between the groups. They also assessed the influence of the polysomnographic findings on corneal endothelial cell shape and central corneal thickness.

The researchers noted the central endothelial cell density and central corneal thickness were not significantly different between the groups. However, the variation of cell area was significantly higher and the hexagonal cell appearance was significantly lower in the OSAHS group. Additionally, the investigators observed a significant negative correlation between central corneal thickness and REM sleep.

"Our study highlighted the corneal endothelial alterations in patients with severe obstructive sleep apnea-hypopnea syndrome," says researcher Evangelia Chalkiadaki, MD. "This is the first time that increased pleomorphism and polymegethism of the central corneal endothelium were observed in apnea patients, probably as a result of chronic, intermittent hypoxia. Apnea patients with a lower percentage of REM sleep had increased corneal thickness—an indicator of poor corneal oxygenation."

The study suggests clinicians should be careful when dealing with the eyes of patients with severe OSAHS, especially with intraocular procedures such as cataract surgery.

Future controlled studies with larger sample sizes are needed to confirm the relationship between REM sleep and corneal thickness and to determine their clinical significance, the researchers suggest.

Chalkiadaki E, Andreanos K, Florou C, et al. Corneal endothelial morphology and thickness alterations in patients with severe obstructive sleep apnea–hypopnea syndrome. Cornea. June 10, 2020. [Epub ahead of print].

NEWS STORIES POST EVERY WEEKDAY MORNING AT www.reviewofoptometry.com/news

FTC Finalizes Hot Button CL Rule

Prescribers must document and confirm patients received their prescriptions.

Following four years of review and thousands of public comments, the Federal Trade Commission (FTC) recently voted to amend the Contact Lens Rule—referred to as the Final Rule—which "facilitates shopping for contact lenses by requiring prescribers to automatically provide a copy of a patient's prescription to the patient and to verify or provide prescriptions to third-party sellers."

Prescribers will also need to request patients' confirmation that they received their Rx, but the Final Rule provides some flexibility in the way the prescription and confirmation are provided, the FTC claims.

From 2015 to 2019, the FTC hammered out the Final Rule by considering public input, surveys, studies, analyses and other information about the evolving contact lens marketplace.

Under the Final Rule, prescribers will be required to do one of the following actions to confirm a patient received their prescription following a contact lens fitting:

- Ask the patient to acknowledge the receipt of the contact lens prescription by signing a separate confirmation statement.
- Ask the patient to sign a prescriber-retained copy of the prescription that contains a statement confirming the patient received it.
- Request the patient sign a prescriber-retained copy of the receipt for the exam that contains a statement confirming the patient received the prescription.
- Give the patient a digital copy of the prescription and retain evidence it was sent and received or made accessible, downloadable and printable.

An Extra Burden

While the update was expected, as it's been in the works since 2015, the timing of the decision in light of COVID-19 is disappointing, says optometrist Brian Chou of San Diego.

Currently, most optometrists are navigating a dramatically different practice landscape due to the pandemic, including increased administrative burdens with PPP loan accounting, greater costs for PPE and cleaning, re-staffing issues, additional time spent on safety measures and slowing schedules to enhance physical distancing, he says.

"It would've been nice if the FTC displayed greater awareness of the COVID-19 fallout on optometric practices by giving more lead time for implementation of these updates. The FTC definitely lost points with me by their insensitive timing," Dr. Chou says.

Bring on the Red Tape

Adding another hurdle for prescribers, they now must maintain proof they satisfied the confirmation of the Rx release requirement for at least three years. If a patient refuses to sign a confirmation, prescribers must note this and save the record to prove they are in compliance.

The Final Rule adds a new definition of the term "provide to the patient a copy," which will now allow the prescriber—with the patient's verifiable consent—to provide a digital copy of the prescription in lieu of a paper one.

When seeking a patient's consent, doctors will need to tell the patient the specific method of electronic delivery they will use and also retain a record of the patient's consent for three years. The Final Rule will also require prescribers to give patients or their designated agents an additional copy of their prescriptions on request within 40 business hours.

The Final Rule will put an additional administrative burden on optometric practices' staff and software, Dr. Chou says.

"This adds insult to injury during a time when optometric practices are recovering from closure and having to do more work than ever," he notes. "The ideal scenario is for the FTC to provision adequate time for the various optometric electronic medical records (EMRs) to catch up in development and release software builds that seamlessly document conveyance of contact lens prescriptions to patients. That way, staff don't need to obtain patient signatures and scan them into document management."

Unfortunately, EMR development takes time, likely up to 12 months, he adds. "I would recommend optometrists let their EMR companies know loud and clear that they need this enhanced functionality ASAP to help reduce the additional administrative burden of the Rule's update," he says.

Still, the FTC ruling has a bright side, Dr. Chou adds, since he believes it may force optometry to reduce its reliance on product sales and shift the profession further toward service. Patients may end up paying less for their disposable lenses but more for their service fees in part to subsidize meeting the update's administrative requirements, he suggests.

(Continued on p. 6)

ROCK Inhibitor Improves Fuchs' Outcomes

An ARVO abstract suggests that treatment with the rho-kinase (ROCK) inhibitor ripasudil may suppress the expression of genes responsible for abnormal extracellular matrix deposition and guttae formation in Fuchs' endothelial corneal dystrophy (FECD). Researchers took endothelial cell–Descemet's membrane (EDM) complexes from FECD patients during Descemet's membrane endothelial keratoplasty and from normal donor corneas unsuitable for transplantation. The team analyzed gene and protein expression with and without a dose of 30μM ripasudil.

They found the ROCK inhibitor caused significant downregulation of FECD-specific genes—both at the mRNA and protein level—compared with untreated controls. Suppressive effects were more pronounced in FECD specimens than in normal control specimens and were maintained for up to 72 hours of incubation. They observed discrete changes in the expression levels of a number of components of the signaling pathways upon treatment.

The study authors conclude that this approach could serve as a novel anti-fibrotic treatment in patients with early-stage FECD.

Kruse FE, Zenkel M, Tourtas T, et al. Inhibition of the rho-ROCK pathway modulates abnormal matrix production in Fuchs' corneal endothelial dystrophy. ARVO 2020. Abstract #1182.

ODs respond to Updated FTC Rule

(Continued from p. 5)

New Rules for Sellers

The Final Rule includes several new requirements for sellers as well. To address concerns about such services verifying Rxs by leaving incomplete or incomprehensible automated telephone messages with prescribers, sellers who use those services for verification must do the following:

- Record the entire call, and preserve the complete recording.
- Start the call by identifying it as a prescription verification request made in accordance with the Contact Lens Rule.
- Deliver the verification message in a slow and deliberate manner and at a volume that the prescriber can understand.
- Make the message repeatable at the prescriber's option.

Specialty Lens Changes

"Since my practice is skewed toward managing keratoconus and eye disease with specialty lenses, I am disappointed that the FTC has not yet educated consumers that their intent with this update is to improve competition in the soft disposable contact lens space, not custom medically-indicated lenses," Dr. Chou says.

The danger is that patients in medically indicated contact lenses for issues such as keratoconus, corneal transplantation and graft-vs.-host disease will mistakenly believe they can purchase their custom lenses through any online retailer and their doctor can readily perform lens exchanges in this manner, he adds.

"Not the case," Dr. Chou says. "Whether intended or not, the FTC is externalizing onto eye doctor offices the burden of explaining to patients that custom lenses cannot be filled through just any contact lens company." In effect, he explains, the doctor's office becomes the bearer of bad news, "whereas the FTC could instead be taking the leadership of preemptively educating consumers that medically-indicated lens designs can only be successfully prescribed when the doctor works directly with the contact lens laboratory."

The AOA Reacts

In a statement, incoming AOA president William T. Reynolds, OD, says, "The FTC was wrong four years ago when they first proposed this destructive plan, and they're wrong today in seeking to implement it. More than 100 US Senators and House members—Republicans and Democrats—have joined with the AOA since 2016 to fight back, and we will do what it takes to increase this support going forward. This is a completely misguided attack on law-abiding, frontline optometry practices that is coming at a time when we've been providing essential, primary care through every stage of the COVID-19 public health emergency."

Instead of responding to the pandemic by supporting small health care practices serving their communities and heeding the Federal directives to ease regulatory burdens, this government agency has chosen to attack doctors and penalize patients with a destructive new record-keeping requirement, the AOA noted.

The Rule changes go into effect 60 days after publication in the Federal Register notice. The Contact Lens Rule has been in place since 2004. ■

FTC Announces Final Amendments to the Agency's Contact Lens Rule. www.ftc.gov/news-events/press-releases/2020/06/ftc-announces-final-amendments-agencys-contact-lens-rule. Federal Trade Commission. June 23, 2020.

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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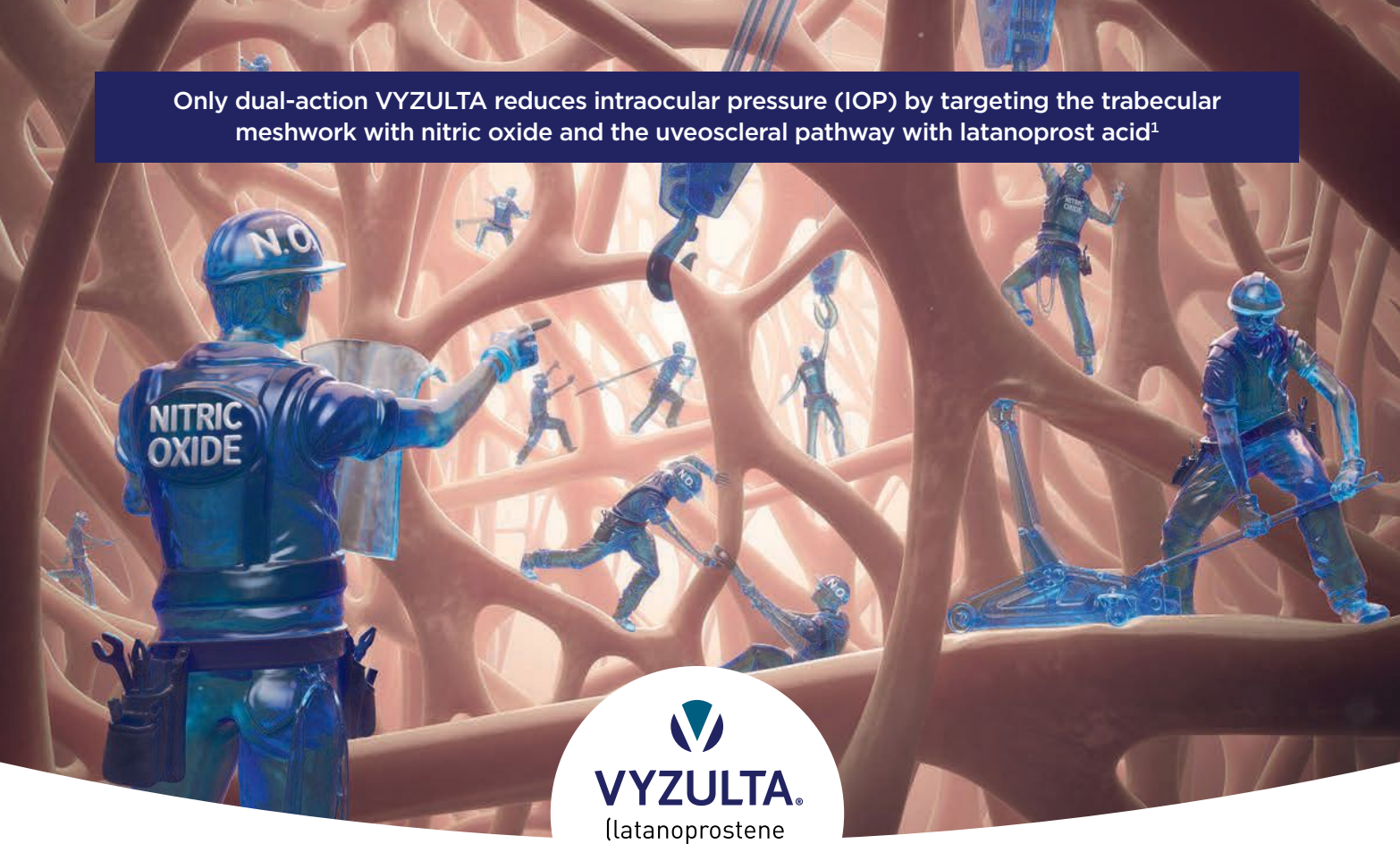

Only dual-action VYZULTA reduces intraocular pressure (IOP) by targeting the trabecular meshwork with nitric oxide and the uveoscleral pathway with latanoprost acid[1]

VYZULTA®
(latanoprostene bunod ophthalmic solution), 0.024%

EXPAND THE TRABECULAR MESHWORK WITH THE POWER OF NITRIC OXIDE[2-6]

VYZULTA achieved significant and sustained long-term IOP reductions vs Timolol 0.5% in pivotal trials[7]

P<0.001 vs baseline at all pre-specified visits over 12 months in a pooled analysis of APOLLO and LUNAR clinical trials (N=831)

VYZULTA demonstrated safety profile in clinical trials

Only 6 out of 811 patients discontinued due to ocular adverse events in APOLLO and LUNAR clinical trials[1,8,9]

Visit VYZULTANOW.com to see our efficacy results

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

IMPORTANT SAFETY INFORMATION cont'd

- 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 Prescribing Information on adjacent page.

References: 1. VYZULTA Prescribing Information. Bausch & Lomb Incorporated. **2.** Cavet ME. *J Ocul Pharmacol Ther*. 2018;34(1):52-60. DOI:10.1089/jop.2016.0188. **3.** Wareham LK. *Nitric Oxide*. 2018;77:75-87. DOI:10.1016/j.niox.2018.04.010. **4.** Stamer DW. *Curr Opin Ophthalmol*. 2012;23:135-143. DOI:10.1097/ICU.0b013e32834ff23e. **5.** Cavet ME. *Invest Ophthalmol Vis Sci*. 2015;56(6):4108-4116. **6.** Kaufman PL. *Exp Eye Research*. 2008;861:3-17. DOI:10.1016/j.exer.2007.10.007. **7.** Weinreb RN. *J Glaucoma*. 2018;27:7-15. **8.** Weinreb RN. *Ophthalmology*. 2016;123(5):965-973. **9.** Medeiros FA. *Am J Ophthalmol*. 2016;168:250-259.

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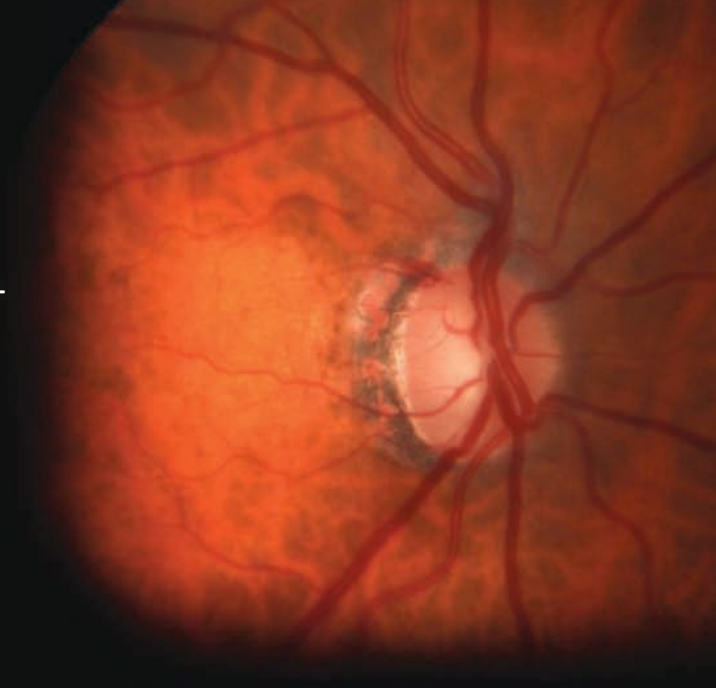

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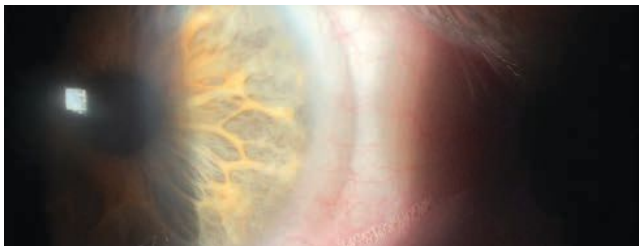

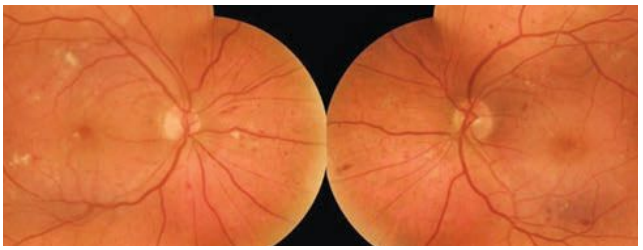

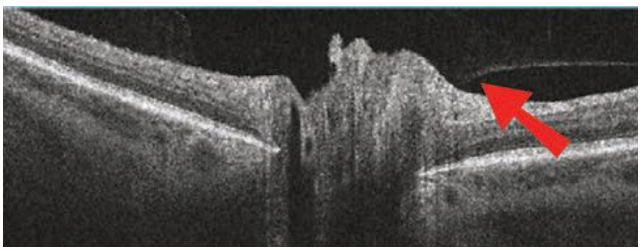

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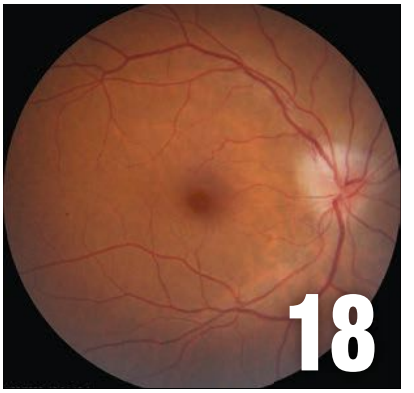

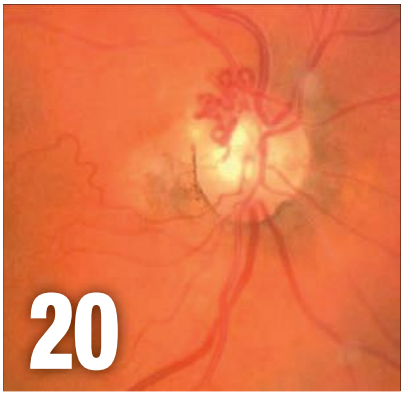

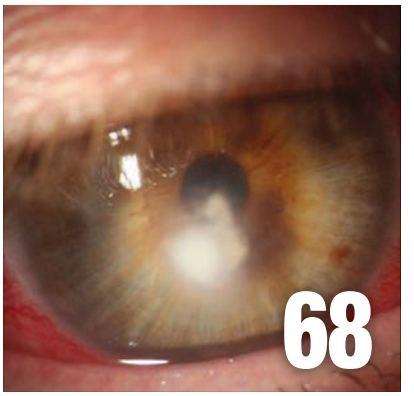

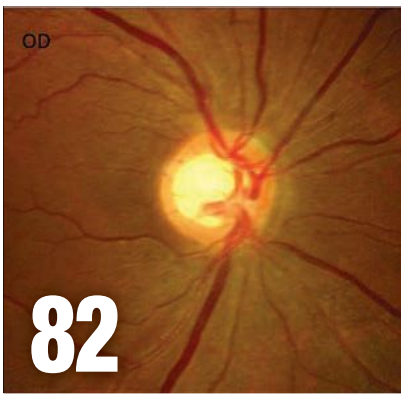

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Outlook

By Jack Persico, Editor-in-Chief

A Bitter Pill

Taking the profit out of contact lens sales looks to be devastating. But could it also be liberating?

I think we can all agree that the Federal Trade Commission's new Final Contact Lens Rule, adopted in late June after four years of debate, is a lousy deal for optometry. First on everyone's minds is the financial hit. A policy that mandates prescription release, and takes great pains to make clear that patients will know they have the freedom to price shop, will cause the bottom to drop out of many practices' materials sales.

Then there's the red tape. Practices are expected to provide the Rx, document this exchange, maintain proof of Rx release for at least three years *and* still deal with the litany of robo-calls for prescription confirmations from big-box sellers. If you have concerns about these verification requests from retailers, you're free to chase them down for clarification. Good luck with that.

Finally, there's the risk to patients. Putting price front and center in the minds of contact lens wearers is going to foster a mindset that cost concerns should drive their decisions. The prospect of cheap lenses delivered in 24 hours will hold much more sway than nebulous concepts like lens design, visual acuity, eye health, routine check-ups and all the rest. Prices are clear, unambiguous signals people use to evaluate their options. Quality of care is far less measurable.

Even in a good year, none of this would be met with enthusiasm. And this is certainly not a good year. But maybe the chaos of 2020 creates a perfect opportunity to start moving beyond reliance on product sales.

Think about it: practice finances are so off the rails this year anyway that it might be the best time in recent memory to rejigger your fee structure to value your skills more than your inventory.

That strategy has been advocated for decades, and the comeback has always been: *easier said than done*. Many practices simply need product revenue to survive (or at least maintain the expected returns). Since COVID-19 has forced most practices to make tough calls about changes to their staffing, supplies and services already, what's a little more? As Winston Churchill said, "If you're going through hell, keep going."

Raising your contact lens fitting fees wouldn't go down easy with established patients who are accustomed to what they've been paying. What could justify a sudden hike? It's not like you suddenly got 20% better at fitting lenses, right? They'll likely need a loyalty discount of some kind to prevent bad blood. But adding specialty services like scleral lens fitting and even a renewed push into multifocals (still a distressingly small portion of lens sales in most practices) could add to the complexity of your offerings and help justify a new approach to how you bill for contact lens services.

Product sales revenue won't go away overnight; it's too ingrained in most traditional optometry practices. But starting to wean off that reliance will add some distance between you and cut-throat retailers, who'll always have a price advantage. Focus instead on yours: clinical expertise. ■

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Glaucoma Updates Post-COVID

The pandemic hasn't slowed progress in new care options.

By Paul M. Karpecki, OD, Chief Clinical Editor

Despite the COVID-19 lockdown, the FDA has continued to approve new therapeutics, many of which will impact how we manage glaucoma. The approval of Durysta (bimatoprost implant, Allergan), for example, yields the first intracameral sustained-release implant to lower intraocular pressure (IOP). In two Phase III studies, Durysta lowered IOP by approximately 30%; though it dissolves in about three months, the effects continue for years. Many other changes are on the horizon:

Diagnostic advances. Hysteresis, measured with the Ocular Response Analyzer (Reichert), is becoming increasingly useful. Research shows this measurement may be a predictor of glaucoma progression risk.[1] For me, it's often the measurement that determines if I should or should not treat a borderline glaucoma patient, or helps me better understand why they are progressing.

Another new diagnostic tool for glaucoma is the Eyekinetix (Konan Medical). Most cases of glaucoma involve asymmetric nerve changes, and the device accurately and quickly measures pupils, including subtle relative afferent pupillary defect, overcoming the shortcomings of the swinging flashlight test.[2]

Treatment updates. Doctors are now closely addressing the ocular surface of glaucoma patients, as chronic preservatives combined with inflammation-inducing drops, such as prostaglandin analogs, can cause discomfort, quality of life issues and poor compliance. Now, more ODs are suggesting selective laser trabeculoplasty or preservative-free drops.[3]

Another potentially useful procedure is MIGS at the time of cataract surgery. At this year's AGS meeting, the four-year Hydrus (Ivantis) pivotal trial data was released, showing that 71.4% of patients (vs. 44.2% who had cataract surgery alone) who started the trial on one medication remain medication free post-op.[4]

Telemedicine

With COVID-19 mandating less time with and greater distance from patients, more clinicians have gone virtual. Not only that, reimbursements for telemedicine exams are now on par with live exams. While glaucoma may not seem to fit the usual virtual visit profile, many opportunities exist. For example, patients may come in for OCT, visual fields, hysteresis and an IOP check—and then schedule a telemedicine visit to discuss the findings.

Keep in mind that the patient must request the telemedicine visit, which means you need to educate them that you provide virtual care. Document the same way you would with a live visit but record the amount of time spent with the patient. Then, email them a video or voice recording of the discussion, follow-up and any medication instructions. I even include an animation pertinent to glaucoma (via Rendia) that provides them an archivable recording regarding drops and dosing, which decreases call backs and patient confusion.

The world of glaucoma is changing, in a good way. We have myriad new opportunities to improve the lives of our patients with this vision-threatening disease. ■

Note: Dr. Karpecki consults for companies with products and services relevant to this topic.

New Tools for DED

The FDA has also been busy approving new treatment options for dry eye:

The agency accepted the resubmission of the New Drug Application for Eysuvis (loteprednol etabonate ophthalmic suspension 0.25%, Kala Pharmaceuticals) for the short-term treatment of the signs and symptoms of DED.[1] Also, the iTear100 Neurostimulator (Olympic Ophthalmics) was approved as a non-drug, external neurostimulator to temporarily increase acute tear production.[2] Finally, Bausch + Lomb recently received approval for the Infuse daily disposable silicone hydrogel contact lens, made with a new material (kalifilcon A) designed with those who experience contact lens dryness.

1. Kala Pharmaceuticals resubmits New Drug Application for EYSUVIS for Dry Eye Disease. Business Wire. May 4, 2020.
2. Olympic Ophthalmics receives FDA clearance for iTear100 neurostimulator. PR Newswire. May 14, 2020.

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PPE: Tales From the Trenches

These safety measures have proven tricky, but I've got a few pointers.

By Montgomery Vickers, OD

Well, folks, we are almost all back to work by the time this prints. I think you will agree that the transition into the nouveau optometric practice has been a little smoother than we thought it would be.

Now, don't get me wrong. I, too, had to convince myself that I was unlikely to catch a potentially deadly disease while refracting a 10-year-old who was messing with his mask, but, day by day, it has become easier for me to ease into this new world.

But a lot has changed, and some of it has been surprising. For example, we hardly have any no-shows now. Oh, 20-something males still never show up, but that's expected. Everyone else is showing up. I have mixed emotions about that trend, since 83.4% of my humor is related to griping about no-shows in this column. Still, if almost every one of my usual no-shows actually show up, all things considered, it's a good thing, right?

Coverup Considerations

You will agree that the personal protective equipment (PPE) has taken some time to get used to. I'm learning what works for me, and I would like to share with you some practical PPE and other hygienic wisdom I have gained:

1. Do not put the mask on immediately after eating cheese. Trust me.

2. To avoid fogging up your glasses just as you are picking rust out of someone's cornea with a needle, go back to wearing your multifocal contact lenses, whether you can see with them or not.

3. If you have a reusable cloth mask, wash it, for goodness's sake! If you are wearing a disposable paper surgical mask, uh, dispose of it before the inside looks like a three-year-old's pull up.

4. Use a mask that's tight enough for a decent seal but not so tight that you look like Jeff Sessions.

5. Remember, your patient cannot tell you if are smiling, so clap or something. Also, laughing behind the mask must be handled delicately or they may think you are hacking your lungs out.

6. Do not automatically shake hands with someone who sticks his out to you. Me? I give the foot bump. Patients seem to think it is funny and laugh… or maybe they are hacking their lungs out.

7. When you wash your hands, make sure you do it in front of the patient—and be sure it's for 20 seconds. I've gotten called out on skipping a few seconds more than once by the hand-washing police.

8. Always remember that your patients are pretty freaked out these days. Maybe their first bifocal can wait a couple more months, unless, you have a licensed grief counselor on staff.

9. Your mask should not look like a skeleton's grin. Stick with puppies or Mick Jagger's lips or something.

10. People ask me, "What about wearing gloves?" Well, gloves are probably less sanitary than your carefully washed hands (see #7), but patients who come in with things stuck in their eyes that have to be removed by your filthy hands actually may like to see you in gloves.

11. Speaking of gloves, please (a) buy decent quality gloves so they don't split like a jilted boyfriend and (b) practice putting them on and off so you don't look like a dork. Being an optometrist is dorky enough.

I am keeping track of all things related to reopening in the COVID era. You can be sure that once patients start no-showing again, I will get back to being funny. ■

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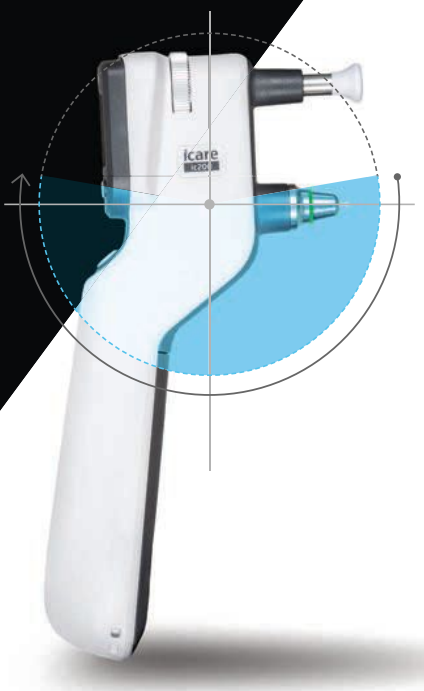

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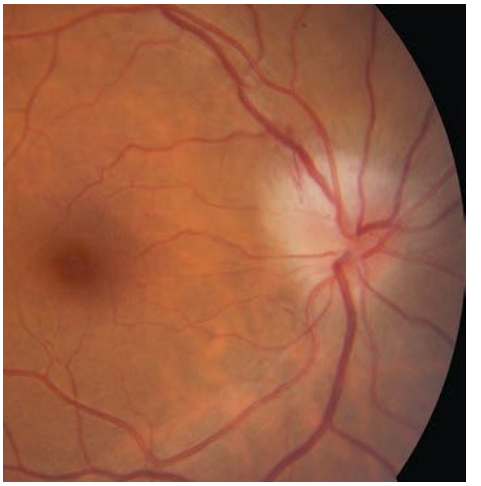
An Optic Neuritis Outlier

This condition, as well as MS, can still affect patients outside the typical age demographic. **Edited by Paul C. Ajamian, OD**

Q A 51-year-old presented as an emergency during the shelter-at-home period. She had complaints of a streak in front of her right eye, but her acuity was 20/20. A swollen nerve was noted without an afferent pupillary defect (APD). What is my differential diagnosis here?

A "When looking at the nerve—any unilateral swollen optic nerve—you have to consider optic neuritis," says Nate Lighthizer, OD, an associate professor at the Oklahoma College of Optometry. One of the most common causes of a unilateral swollen nerve is optic neuritis. And one of the most common underlying causes of optic neuritis is multiple sclerosis (MS).

"Usually, if you're asking for the typical demographics of optic neuritis that you see in all the textbooks and in the clinical trenches, those patients are in their 20s to 40s with a unilateral presentation of sudden vision loss," Dr. Lighthizer notes. While this patient was not within this age range, he didn't think she was too old for optic neuritis.



While not the case with this patient, an APD, decreased vision and pain on eye movement often accompany optic neuritis.

Ask Questions

"When you see unilateral nerve swelling in a patient no older than 50, have optic neuritis near the top of your differential," Dr. Lighthizer says. Optometrists should look carefully for an APD using a very bright light source, such as a binocular indirect ophthalmoscope. Also, ask about pain on eye movements. This patient was an exception on all counts: older age, and no APD or pain on eye movements.

"When a unilateral swollen optic nerve presents, ask if the patient has been diagnosed with MS," Dr. Lighthizer says. If the answer is no, they may be presenting to you with one of the classic signs of multiple sclerosis. To rule out multiple sclerosis, you will need to have neuroimaging done, according to Dr. Lighthizer. You can either order the scan yourself, or send the patient to a neuro-ophthalmologist or a neurologist," Dr. Lighthizer notes. In our case, the MRI helped confirm the suspicion of MS.

Check History

Dr. Lighthizer reminds us to review the med the patient is on, along with conducting a detailed medical history, to eliminate other potential causes of the swollen optic nerve. Hypertension can reach such elevated stages that it can cause bilateral swollen optic nerves, also known as malignant hypertension.

"If an older patient presents with a history of obesity, high blood pressure for an extended period of time or uncontrolled diabetes, then non-arteritic ischemic optic neuropathy (NAION) is a much more likely diagnosis because of their vascular history," Dr. Lighthizer says.

Treat Accordingly

The good news with optic neuritis is that it usually resolves even without treatment. However, high dose IV methylprednisolone for three days can accelerate healing and visual recovery. This is sometimes followed by oral steroids for about two weeks with a slow taper. "Usually the neurologist or the neuro-ophthalmologist will make the call to order the IV steroids," notes Dr. Lighthizer.

He recommends that optometrists follow their patients every other week once they are out of the hospital. It is especially important to monitor their vision, pupils, visual field and nerve status in these cases. Be sure to communicate your findings clearly and regularly with the specialist.

Optic neuritis and MS can happen at any time to anyone of any age. Keep it on your radar, and remember to advocate for the patient and get them into the neurology system in a timely fashion. Be mindful this is not always an easy task. ■

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RVOs: Detour Ahead

The formation of collateral vessels may help you understand the severity of the occlusion.

By Bisant A. Labib, OD

The vascular network that nourishes the retina is a complex and essential part of vision. Obstructions may occur within these blood vessels—namely, vein or artery occlusions—that impair the normal pathway of blood throughout the retina, potentially leading to devastating visual loss.

Much like many other parts of the body, the retinal vasculature attempts to troubleshoot these occlusions and form alternative routes to restore flow. In the case of venous occlusions specifically, these mechanisms may appear as collateral vessels, which are identifiable on clinical examination and may affect the prognosis or course of venous occlusion.

Collateral FAQs

When a retinal vein occlusion (RVO) occurs, collateral channels often form to bypass the site of occlusion and offer an alternative path for blood to nourish the affected retinal area. Unlike neovascularization, these vessels are preexisting deep within the capillary bed and only fill when necessary.

The filling of collateral vessels is thought to depend heavily on hemodynamic factors. Increases in hydrostatic pressure within the occluded area direct blood into areas of lower capillary pressure. This pressure gradient, resulting from hemodynamic stress, eventually leads to enlargement and filling of these already existing capillary channels in an effort to bypass the vein occlusion.[1-4]

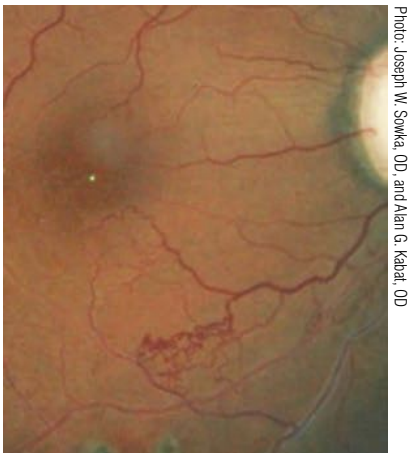


Photo: Joseph W. Sowka, OD, and Alan G. Kabat, OD

This patient's retina exhibits collateral vessels bypassing an occluded vessel.

Unlike other vascular responses in the retina, collaterals connect an obstructed vein to a neighboring unobstructed vein.[2,3] In contrast, a vascular shunt, for example, connects dissimilar blood vessels, such as arteries, to veins.[1,3]

The most common cause of collateral formation is either branch or central RVOs; of these two, branch retinal vein occlusions (BRVOs) more frequently lead to collateral formation.[1]

Because BRVOs and central retinal vein occlusions (CRVOs) occur in different locations, collaterals that form in each case will also appear different clinically.[4]

In a BRVO, the obstruction occurs at sites where the retinal artery crosses a vein, or an arteriovenous crossing.[5] The vein occluded at that site has surrounding, unobstructed channels that ultimately lead to the central retinal vein, where intraretinal collateral formation arises to restore blood flow in a BRVO, developing within weeks of the occlusion.[1,4]

On funduscopic examination, these areas appear as intraretinal tortuous blood vessels within the deep capillary bed, across the temporal raphe and other sites to bypass the occluded area. They may be difficult to distinguish from neovascularization; here, fluorescein angiography (FA) is useful. Initially following BRVO, there is minimal leakage of collateral vessels due to limited capacity and weak endothelial cells. With maturation, the vessels become larger and more stable.[5]

Conversely, CRVO occurs at the level of the lamina cribrosa, where all branches of the central retinal vein are affected. As such, these collaterals are not intraretinal but are usually located on or around the optic disc and use the choroidal venous system for drainage.[4]

These vessels appear tortuous, with slow blood flow and cross the horizontal raphe, but may straighten over time or disappear when the obstructed site reopens.[2,3,5] They take on the same caliber as a normal retinal vein, which is the type of vessel collaterals appear to replace.[5]

Warning Signs

Generally, the number or severity of collaterals seen in both types of RVO is associated with the extent of capillary nonperfusion; thus,

the identification of collaterals on clinical examination is a potential marker for the degree of retinal ischemia that has taken place secondary to the occlusive event.[2]

It is difficult to ascertain whether collateral formation yields an improvement in visual outcome in RVOs or if they simply serve as markers for severity.

In BRVO, additional clinical features are associated with collateral formation, including a greater length of time of macular edema. This is potentially due to the longer duration of the ischemic event, generally, and the subsequent opportunity to develop collaterals in that timeframe.

Patients with collaterals have a smaller area of retinal hemorrhages that can be attributed to chronicity and resorption of hemes. Research also shows a correlation between younger patients with BRVO and the formation of collaterals, likely due to their ability to more readily undergo vascular remodeling.[4]

Road to Recovery

The timing of laser therapy in the treatment of BRVO complications such as macular edema or neovascularization is critical for prognosis. Based on the Branch Retinal Vein Occlusion Study, laser treatment is considered three to six months following the BRVO. The typical time for collaterals to form and mature is approximately six to 24 months. Laser treatment in areas of nonperfusion prior to this can impair collateral vessel formation. However, using a laser to destroy collaterals that have formed can increase neovascular leakage.[5]

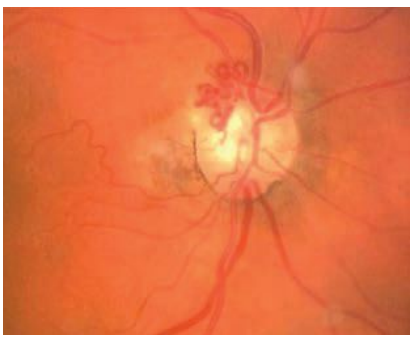
While collateral vessel formation is a natural mechanism to restore blood flow and reduce ischemic damage, research shows patients with CRVO and collaterals actually have poorer visual outcomes. One study found 52% of patients had acuity of 20/70 or worse, compared with 35% of CRVO eyes without collaterals.[2,4] Patients with collaterals also had macular edema for twice as long and a lower chance of visual improvement compared with those who didn't have collaterals.[4]

These factors indicate that CRVO with collateral formation is more of a marker for severity of ischemia. With the reduction in capillary density in severe cases, collaterals are more likely to arise, or patients who develop collaterals may have fewer available tributaries.[2]

In any vein occlusion case, identifying collaterals through clinical examination can be an important tool in determining the severity of the ischemic event and provide a clue as to the patient's ultimate visual prognosis. ■

OCT-A: Go with the Flow

Optical coherence tomography angiography (OCT-A) is a newer tool that allows for noninvasive visualization and analysis of retinal blood flow.[1] OCT-A images are created with successive images of OCT B-scans that detect flow of motion from red blood cells within the retinal vasculature.[6] The technology is faster, safer and less invasive than FA, and can also image deep capillary plexuses better than FA. Both FA and OCT-A can offer information on the location of ischemic retinal areas.[7] Quantifying the area of nonperfusion allows the clinician to predict the severity and outcome of the ischemic event.[6]



These collaterals formed over the optic disc in response to a CRVO.

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Collaterals vs. Neovascularization

	Origin	Appearance and Location	FA Testing
Collaterals	Pre-existing vessels that fill to bypass an occlusion.	Tortuous and larger caliber vessels around the peripapillary plexus.	Minimal leakage in very early stages; no leakage in mature vessels.
Neovascularization	New vessels formed by angiogenic cytokines and endothelial proliferation in response to ischemia.[1]	Fine meshwork of vessels at the level of the vitreous.[7]	Permeable to fluorescein.

Coding a Suspect

The rules differ when monitoring a patient who hasn't converted to glaucoma—yet.

By John Rumpakis, OD, MBA, Clinical Coding Editor

Evaluating a patient for the presence of glaucoma is part of the daily routine for any optometrist. Still, knowing current parameters on what constitutes a glaucoma suspect and the appropriate coding may require a refresher, since the ICD-10 code for a glaucoma suspect is invalid to use—and with the frequency it is used, it can spell trouble for your practice.

Setting a Diagnosis

Although 304 ICD-10 codes contain the word glaucoma, only one exists for glaucoma suspect (H40.0). Yet, it's not a proper code to use for diagnosis or for submitting to a carrier because it lacks specificity.

According to the American Academy of Ophthalmology (AAO), the diagnosis of a primary open-angle glaucoma (POAG) suspect is established by the presence of one of the following: consistently elevated intraocular pressure (IOP), suspicious optic nerve or abnormal visual field. It could also have associated risks of elevated IOP, family history of glaucoma or glaucoma suspect, thin central cornea, race, older age, myopia and type 2 diabetes.[1]

The diagnostic testing associated with a patient at risk, but not diagnosed, includes gonioscopy, pachymetry, tonometry, perimetry, careful optic nerve observation and ocular imaging. The term "ocular imaging" can include fundus photography and OCT based on the specific medical necessity of the patient.

These diagnosis codes (highest specificity only) can be used to pursue the necessary additional diagnostic tests and are the only codes to be used for proper diagnosis of a glaucoma suspect:

- H40.00X: Pre-glaucoma
- H40.01X: Open-angle with borderline findings, low risk
- H40.02X: Open-angle with borderline findings, high risk
- H40.05X: Ocular hypertension

The physician must specifically identify with the highest level of specificity the patient's type of "suspect." Using the same code for all suspects because of convenience or routine is inappropriate.

When testing, clinicians should map the appropriate ICD-10 code to the appropriate procedure they are performing. Although the list of procedures is broad, clinicians should not perform the same tests on every patient. Instead, they must consider each patient on an individual basis and only order clinically relevant and necessary tests.

Ongoing Care

Once the clinician establishes the diagnosis—whether a specific form of glaucoma or simply at risk—they then use that ICD-10 code on subsequent visits when performing follow-up tests to monitor progress and treatment effect.

According to the AAO's Preferred Practice Pattern for POAG, the ongoing clinical testing for a patient includes:[2] 92250 (stereo photography), 92133 (OCT of optic nerve) and 92083 (visual fields, threshold).

The frequency of testing is now based on two criteria: the patient's condition and the insurance carrier's guidelines. Most carriers allow one to two OCTs per year, generally alternated with a visual field. For stereoscopic photos, clinicians must establish necessity in the medical record each time they take a photo. Thus, if there is no change in the optic nerve noted in the physical exam, there is no necessity to photodocument "no change."

If clinicians must perform an extended optic nerve exam, the new (January 2020) code is 92202: ophthalmoscopy, extended; with optic nerve or macula drawing and I/R, unilateral or bilateral.

The additional criteria clinicians must meet stem from the National Correct Coding Initiative, or CCI edits. These rules stipulate what procedure codes can or cannot be performed on the same date of service.

Too often, practitioners ignore the insurance carriers' and the CCI edits' rules. This leads to inappropriate use of modifiers (specifically -59) because claims get rejected. This is highly scrutinized by carriers, and doctors are fined for inappropriate clinical and coding procedures.

The proper identification of glaucoma is a vital part of the clinical evaluation, and clinicians must understand the CPT and ICD-10 rules it requires. ■

Send your coding questions to rocodingconnection@gmail.com.

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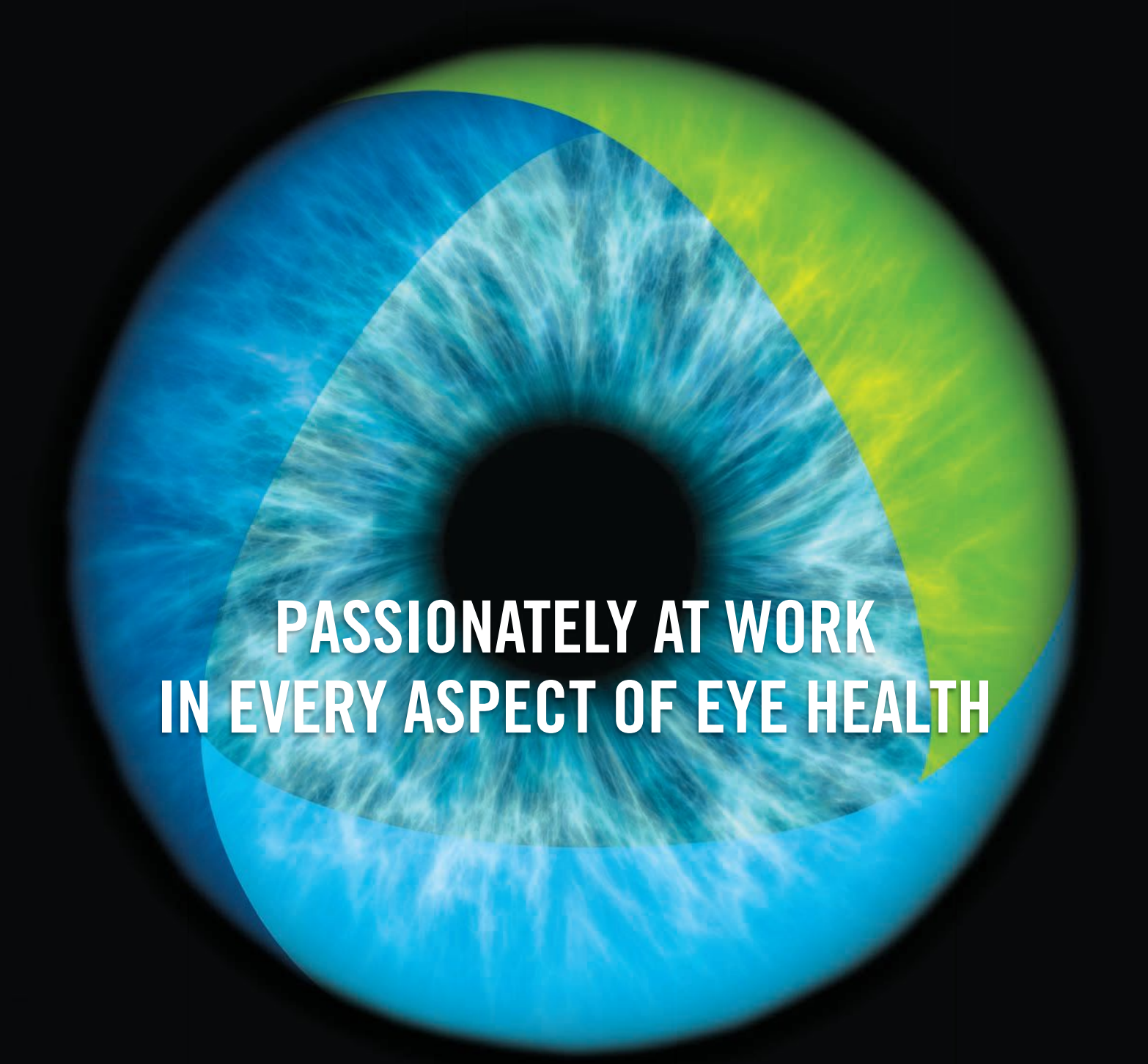

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Satisfying the Complicated Presbyope

Concurrent issues such as dry eye, prior surgery or binocular disorders can compromise visual quality. Optometrists can help restore it.

By Christopher Luft, OD, and Gregory Barbush, OD

When patients in their early to mid-forties remark, "Doc, I used to always have great vision, but now I feel as though I can't see anything without my glasses," any optometrist knows they're likely dealing with presbyopia. This pervasive condition affects roughly a quarter of the entire global population.[1] Unfortunately, 826 million are likely to have limited daily function because they don't have adequate management or correction.[1]

Optometrists can make the difference and offer patients corrective lenses and other solutions.[2,3] But that's not the end of their problems. Presbyopia can complicate, and be complicated by, a number of other conditions. Optometric physicians will need to consider the impact of their patient's visual condition when fighting conditions such as dry eye, cataracts, retinal disease or prior refractive surgery. This article helps ODs navigate the necessary considerations when managing presbyopia in complex situations.

Prism and Progressive Lenses

Purposeful prism can certainly be introduced into progressive lenses for presbyopic patients with diplopia, strabismus and other binocular vision conditions.[2,4] Success with prism requires a delicate balance of objective and subjective tolerances; thus, doctors must tailor parameters for prescribing to the individual patient, taking into account their age, systemic and ocular histories, and common patterns of visual demands.[2-4]

While cover test and prismatic implementation into a trial frame can be rudimentary, there are some pearls to keep in mind when fitting progressive lenses. Prism appears in spectacles whenever the thickness of the lens varies between two points.[5,6] Keep this in mind as we move into progressive lenses that combine multiple powers across one lens surface.[6,7] As we have learned with Prentice's rule, the prismatic effect on any point of a lens is directly proportional to the power of the lens, and the distance of that point from the optical center.[5-7] Measuring the interpupillary distance is very important in progressive lenses and needs to be done with the head properly aligned. Remember to maintain normal head posture while performing cover testing as well as while measuring for spectacles.[7] Any head tilts or turns can throw off the desired prismatic effect and may cause unwanted diplopia in itself.[6,7]

When considering the binocular prismatic consequence of spectacles, we focus on the net prismatic effect between the right and left lenses.[5-7] This is known as the prismatic imbalance, which affects binocular fusion. For vertical prism, bases oriented in the same direction between the two eyes have canceling effects, while the opposite is true for horizontal prism.[6,7] Make sure the amount of prism can be tolerated by the fusional vergence system by using a trial frame, along with the correct orientation of prism between the two eyes.[6,7]

Fresnel prisms can accommodate high ranges of prismatic correction.[8]

They consist of continuous thin, narrow prisms arranged on a plastic sheet.[8] Because their design is dependent upon prismatic angle vs. thickness of the lens, they are thin, flexible, and discrete on the surface of spectacles.[8] Fresnel prisms come in powers up to around 40 diopters.[8] They are a good trial prism for patients using prism for the first time or in cases with a large change in prismatic correction. Fresnel prism, while very useful, does tend to degrade image quality and can be noticeable to the naked eye. Once the patient reports good success and comfort with stick-on Fresnel, prism can then be ground into lenses, a more permanent modality to prismatic correction.

Some diplopic patients with strabismic amblyopia may not achieve single vision with any amount of prism and may warrant patching to resolve symptoms.[5,7,8] The same may be true for other types of diplopia secondary to neurologic concurrent pathologies causing progressive amblyopia over time. Some patients may also have some latent strabismus and may need multiple follow-up visits to ensure proper compensation and resolution in symptoms.

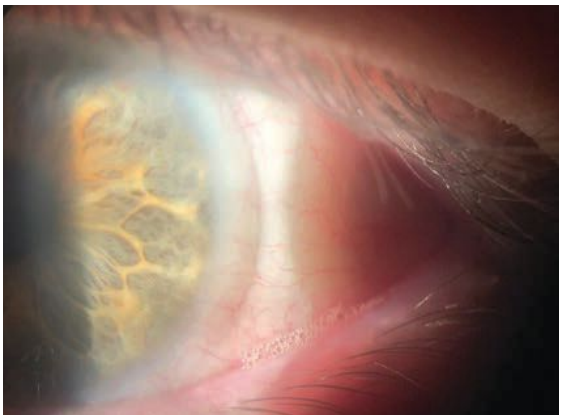
Adaptation to progressive lenses can take time and requires patience in addition to careful consideration. In a study, patients who couldn't adapt to progressives demonstrated slower peak velocities in convergence responses, a weaker ability to modify convergence responses, a reduced rate and magnitude of phoria adaptation and a reduced vergence facility compared to successful wearers.[4] These results suggest that when the accommodative system decreases in presbyopic subjects, the adaptive role of vergence and phoria systems may become critical when adapting to new visual environments such as those created when using progressive lenses.[4] The ability to change convergence peak velocity had the greatest sensitivity and specificity compared to the other parameters.[4]

The trial frame is a valuable clinical tool that allows our patients to see what we intend on prescribing, and feel how the prescription acts on their visual system. Prism and astigmatism can cause disruption to the vergence system and should be tried at all ranges before prescribing. This is an underused technique that all optometrists should rely on for most refractive and prismatic exams. Here, the patient is holding a near card with her prescription in the trial frame at the desired focal point. She is new to reading glasses and was given the "wow" factor before ordering her glasses.

The Ocular Surface

In all varieties of dry eye, especially in cases of severe ocular surface disease, presbyopia adds a lay\er of complexity to the already compromised patient. According to 2018 data from the US Bureau of Labor Statistics, 65% of the civilian labor force is age 35 or older, and this category is projected to maintain that average through 2028.[9-11] This places eye care physicians in a pivotal position. Our aging patient population is learning to battle functional, progressive visual changes and adapting to these changes on top of the use of glasses can be difficult to navigate.

As stated by the epidemiology report from the Tear Film and Ocular Surface Society's second Dry Eye



A bandage contact lens used on a keratoconjunctivitis sicca patient with significant epithelial staining, after which symptoms resolved and redness subsided. Note the frothing along the lower lid margin, indicative of meibomian gland dysfunction and reduced tear quality. Patients with ocular surface compromise often experience visual fluctuations that reduce the quality of multifocal contact lens wear.

Workshop report (DEWS II), for all subgroups analyzed the prevalence of dry eye increased significantly and showed a linear association with age.[12] Research suggests that multiple factors, including uncorrected presbyopia, are associated with both ocular and nonocular symptoms.[13] In fact, a 2017 study showed an increase in dry eye disease in patients who are presbyopic.[14]

Environmental factors such as pollen or dander allergies, prolonged digital screen time and contact lens wear can worsen dry eye signs and symptoms. In advanced stages of the condition, the severity of dry eye damage may become sight-threatening.[15] And the medications that patients use—including antihistamines, hormonal replacement therapy and androgen therapy—can worsen dry eye symptoms, too.[10]

According to DEWS II, 18 classes of drugs can negatively impact dry eye.[12,15,22] Polypharmacy, where multiple medications are used concurrently, may also exacerbate dry eye symptoms. Researchers note that people older than 60 engage in polypharmacy at a rate of approximately 37%.[15]

The main strategy we try to employ with presbyopes that suffer contact lens discomfort related to dry eye disease (DED) or allergy is early detection and management. If we can detect clinical signs of ocular surface disease in the early stages, we can reduce patient symptoms, chair time and contact lens dropout.[16,17] Prior to initiating lens wear for a new presbyope, look for clinical signs of meibomian gland dysfunction, lid wiper epitheliopathy, injection and any reduction in tear-film break-up time (TBUT).

Dry eye treatment plan discussions are often a fluid blend of clinical and therapeutic recommendations along with lifestyle modifications. Preservative-free artificial tears can reduce contact lens discomfort by reducing friction at the ocular surface that can lead to the initiation of the inflammatory cascade.[18] Managing meibomian gland dysfunction with therapeutic warm compresses and lid hygiene effectively improves TBUT and lid health.[18]

Existing and new technologies like Lipiflow (Johnson & Johnson Vision), iLux (Alcon), TearCare (Sight Sciences) and intense-pulsed light (Lumenis and others) offer in-office opportunities for patients of all ages but especially those of mature age with conceivable ability or willingness to pay for these premium services. These hands-on options are also excellent considerations for those preferring a practitioner-involved process to service chronic lid margin disease.

Anti-allergy drops prior to or after lens removal, allergen avoidance and daily disposable lens wear are all modalities we employ to aid in the varying degrees of patients' red, itchy eyes suffering from ocular allergies.

Objective improvement indicators cited in the DEWS reports that ODs should look for at the slit lamp include improved corneal and conjunctival staining, prolonged TBUT (improved over baseline) and improved quality of meibomian gland presentation with less capping and increase in lipid secretion quality.[12,15,22] Clinical testing outside the slit lamp that indicates improvement would be a decrease in tear osmolarity detectable using a clinical osmometer (like that from TearLab).[20] In 2014, researchers determined that osmolarity appears to be the best marker across all levels of disease severity as well as in different subtypes of dry eye disease.[21]

Restoring the function of the meibomian glands, improving the clinical corneal presentation and increasing tear film stability will allow for initial and long-term success.[17] If we are able to identify the combination of therapy given each presbyopic patient's clinical findings, ideally at an early phase, we can open their options up to different modalities of clear, stable vision at multiple ranges.

Contact Lens Options

Monovision contact lenses correct one eye for distance and the other for near ranges (or a modified version of this); patients who are able to tolerate the disparity do well without the need for additional near spectacle help. One disadvantage of monovision is the lack of depth perception and binocularity. We

find monovision works preferably in patients who have notable one-eye dominance, amblyopia or other conditions that already limit binocularity.

Some patients are not good candidates for this option or are unable to adapt well; for them, consider multifocal contact lenses. The advantages of this modality are numerous, including the ability to provide simultaneous vision and binocular function at all ranges. Gas permeable, hybrid and scleral lenses with multifocal optics exist in several different designs, but can usually be incorporated into current user's lenses. In the case of someone with keratoconus and prominent apical scarring, de-centering optics or varying zone sizes can be a triumph for these patients if we are able to adjust the optic zones accordingly where the impact of the scarring is minimized in a multifocal design.

Soft toric multifocals can be a solution for the unmet need of our presbyopic patients who have otherwise not had success secondary to their astigmatism. These manufacturers offer a broad range of parameters to correct or help significant toricity while performing well at near and intermediate ranges with stable vision. Beyond the standard available toric multifocals (Ultra Multifocal for Astigmatism, Bausch + Lomb), custom lens labs also offer a wide variety of soft lens designs and prescription parameters to tailor the optics for each patient.

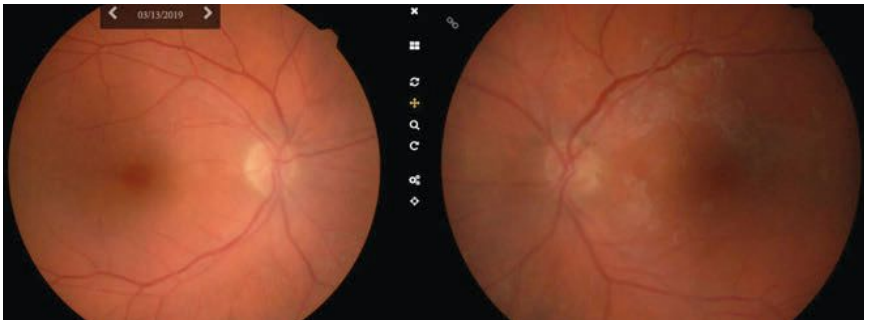
Scleral lenses can also provide stable, clear vision while alleviating symptoms of dry eye disease.[10] The vault of the lens over the cornea allows for a fluid reservoir ("moisture bath") that acts to optically neutralize corneal irregularities and keep the ocular surface hydrated during wear.[21]

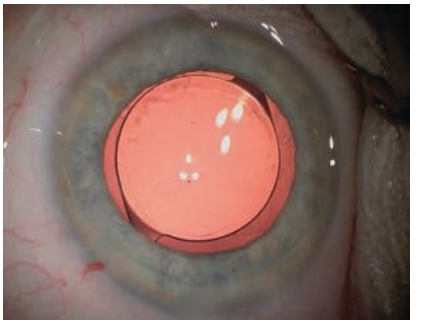
The DEWS report from 2013 recommended scleral lenses if other conservative treatment options such as artificial tears, lid therapy, topical pharmaceuticals or punctal plugs were inadequate in controlling ocular surface disease.[22] This was upheld in DEWS II as a therapeutic consideration for patients with moderate to severe dry eye.[12,15,22] Sclerals may help prevent or delay the patient from having procedures like amniotic membrane transplantation, tarsorrhaphy, mucous membrane or salivary gland transplant, or other lid surgeries.[20-22] Their many advantages include protecting the ocular surface from further desiccation, providing continuous hydration to the cornea to repair underlying epithelial pathology (e.g., punctate corneal staining), allowing for best correctable vision and improving the patient's quality of life.[10,22,23]

Scleral lenses also protect the ocular surface from irregular lid margins or, in cases of entropion that create exposure keratopathy and neurotrophic changes, lead to increased patient comfort.[22,23] Research shows that scleral lens therapy promotes healing of surface epitheliopathy while reducing pain and photophobia.[24,25] It is especially notable in cases refractory to standard treatments involving patients with ocular pain, burning, stinging, foreign body sensation, blurred vision and photophobia resulting from keratoconjunctivitis sicca secondary to chronic graft-vs.-host disease.[23-25] No longer are reading glasses the only presbyopic option for contact lens wearers; scleral lenses present an opportunity to treat ocular surface disease while providing satisfying optical correction.[9,25,26]

Retinal Concerns

Preoperative management is a keystone of optometric care with regard to ocular and visual health,





Left & middle: **Fundus photos taken in the office of a 73-year-old woman sent for cataract surgery. These photos were sent along with the patient's chart, helping the surgeon to visualize the underlying RPE mottling and surrounding atrophy most dense superior nasal to the fovea OD and a widespread epiretinal membrane OS.** ***Right:*** **We recommended a monofocal implant, seen here with retroillumination, as the retinal pathology would cause too much variability and thus undermine the success of a multifocal IOL.**

in addition to personality type and priorities. Notes to the surgeon that reflect patient habits, prior success with monovision or multifocal contacts or any clinical findings unique to the patient are helpful in the surgical process. Any pathology can result in decreased contrast sensitivity, decreased acuity and an unhappy patient experience.[27-29] Document in detail epiretinal membranes, macular degeneration or any form of retinal pathology that may affect vision. As such, a proper dilated exam and well-written summary to the surgeon are imperative to success of any cataract surgery. A macular OCT before cataract surgery is very important and helps in the diagnosis of any suspecting and subtle pathology.[27-29]

Studies show no single type of IOL for these patients is completely without complications; therefore, we try to keep our explanations simple.[27-29] Research also shows that it is not possible to infer a direct relationship between cataract surgery and age-related maculopathies; instead, we must use our clinical judgment and that of the surgeon to determine if cataract surgery will help the patients' quality of life.[27]

Diffractive and refractive optics (or both) with multifocal IOLs cause light interference or light refraction through the implant, respectively.[27-29] In patients with concurrent pathology, the way light is bent and strikes a diseased retina typically produces low rates of success with problems of dysphotopsia, worsening higher order aberrations and poor overall image resolution.[27,28] Due to the fragile grasp we have on good vision with these patients, we typically recommend single vision (monofocal) lens implants to keep the visual system as balanced as possible.[28,29]

Any time we have any form of posterior segment pathology, visual potential will change as retinal disease progresses, which may be exacerbated by premium IOL options.[27,29] We usually will discuss daily activities and what zone of clear vision each patient values the most. From there we can make sure to maximize their visual potential at their desired focal point in order to create a solid foundation for a positive visual outcome.

For those patients who have mild cataracts and mild retinal pathology, we may even steer them into a non-surgical option for the time being. This way we can still modify their prescription while letting mother nature run her course with possible progression of these concurrent etiologies.[29]

Prior Refractive Surgery

Here is another scenario where presbyopia gets the best of us, no matter what procedure we had to correct our distance vision in the past: "Doc, I had LASIK so I wouldn't need glasses for distance; now you are telling me I need them again to read? You must be joking… right?"

According to the refractive surgery council, the number of refractive surgery cases has grown just over 6% since 2017 alone.[30] This is a true test to the advances in technology and high success rates for the industry, though it does affect our patients' response to presbyopia and tends to complicate matters when calculating IOL powers.[30-32] Because of the improved technology and high success rates, these patients are so used to seeing well that any change in vision will be noticeable, putting more pressure on the optometrist and ophthalmologist for superior visual outcomes without spectacle correction.[31,32]

The best thing to do here is to keep our patient's thinking as positive as possible and manage their expectation for all viewing distances. Highlight how their experience is a lot easier to manage post refractive surgery vs. prior. It has been a blessing that they have seen

so well for so long, reiterating that they only need to wear correction for part of the day because their distance vision is still so clear.

The goal of any lenticular implant is to maximize clear vision at the patient's specific desired ranges while reducing glare and minimizing post-surgical distortion.[31,32] Patients with previous refractive surgery are already more likely to have a higher risk for post-operative dryness and higher-order aberrations.[31,32] While there is no cure-all implant for our patients, historical refractive data is very important for the surgeon prior to IOL calculation.

Just as presbyopia motivates patients into the exam chair, it can also motivate them to put their trust in their eye care providers. The importance of dialogue cannot be stressed enough; simply knowing how to talk to the patient in your chair can be the difference between management success and failure. Knowing your audience is indispensable, and using this can help patients better absorb the science of presbyopia correction. The solutions are as ever changing as the problem itself and this realization is imperative to our patient's progress.

There is not one right answer when satisfying near vision demands, but knowing the process and how to manage each type of presbyopic patient will spell continuous financial and practice growth for years to come. ■

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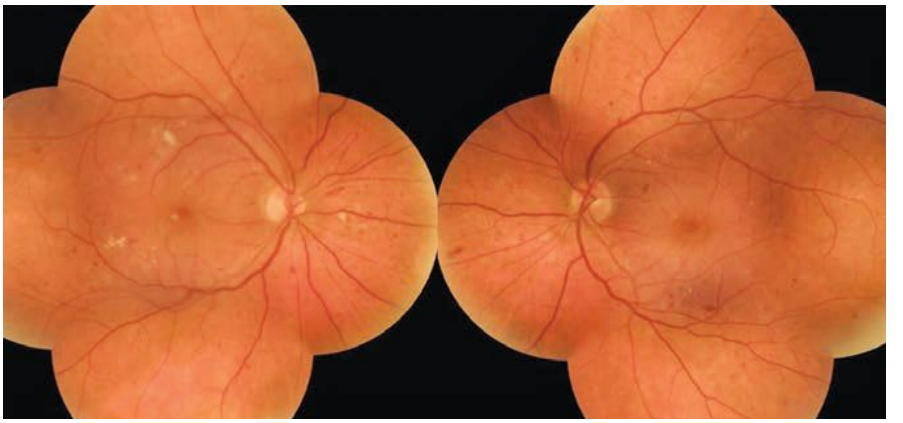
Optometry's Role in the Diabetes Epidemic

If you can get all patients—but especially those at risk—to focus on these five lifestyle modifications, the benefits would be immense. By Kevin Cornwell, OD

Over the past several decades, optometrists have moved from the sidelines into a more integral role when it comes to providing care for diabetes patients. Given optometrists' position as frontline healthcare providers, our involvement in diabetes management should begin, not end, at a patient's initial diagnosis.

Many patients with prediabetes or type 2 diabetes believe they have an irreversible hereditary condition. While this may be the case for those with type 1 diabetes, a completely different story exists for patients with type 2 diabetes.

This article discusses the optometrist's expanding role in managing type 2 diabetes (and prediabetes) and offers different lifestyle recommendations that can help patients manage their health and overcome potential long-term diabetic ocular complications. While these interventions do not replace standard-of-care diabetes management, they can prevent the onset and slow the progression of diabetic retinopathy (DR) and serve as an adjunct to the breakthrough treatments we're continuing to see.



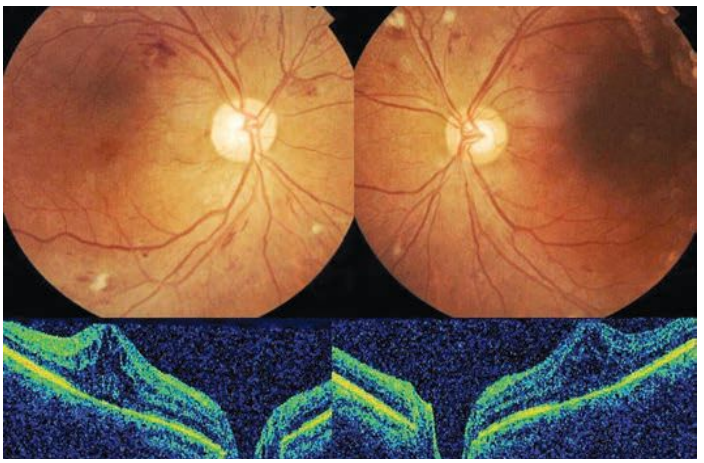
A 58-year-old female presented for her first eye exam without knowing she had type 2 diabetes and was found to have diabetic retinal changes OU.

Prevalence and Cost

According to the CDC, more than 34 million US adults have diabetes, seven million of whom are undiagnosed.[1] The number of Americans diagnosed with diabetes has almost doubled over the past 20 years, with 95% of cases being type 2 diabetes.[1] Optometrists alone diagnose type 2 diabetes in more than a quarter-million patients each year based on our eye exams.[2] After considering the fact that one in three adults has prediabetes, we can see that almost half of the US adult population is at risk of sight-threatening retinopathy, morbidity and mortality, as diabetes is among the leading causes primarily due to its association with cardiovascular disease.[1,3]

Approximately one in three diabetic patients have some form of DR, with up to 24,000 new cases occurring each year—a number that is expected to increase 40% by 2050.[4-6] DR is the leading cause of vision loss among US adults.[7]

Each year, the US healthcare system spends $327 billion to manage diabetes, its complications and the resulting loss in productivity.[8] Healthcare costs for diabetes patients are more than double those of unaffected patients.[8] Regardless of changes to US healthcare policy, no amount of reform can indefinitely sustain this growing economic burden.



A 66-year-old female had a history of uncontrolled type 2 diabetes and severe nonproliferative retinopathy with diabetic macular edema OU.

Given these staggering statistics, this is an all-hands-on-deck scenario for healthcare providers who interact with patients who have type 2 diabetes, prediabetes or metabolic syndrome. It is no longer solely the responsibility of the primary care provider (PCP) or dietician to discuss the importance of lifestyle intervention.

Start the Conversation

When discussing lifestyle intervention with patients, it's crucial to meet them where they're comfortable and tailor the conversation in a way that is manageable so they can engage in and benefit from the conversation. Asking open-ended questions can help start and continue the dialogue in a non-threatening way. For ODs with access to a patient's lab work, mentioning their last A1c and including specific results can be another beneficial conversation starter. Have printed resources available to patients that they can reference on their own time.

Patients who are more motivated to make the necessary dietary and lifestyle changes are likely to respond more proactively and positively to discussion. Some patients may feel uncomfortable with change or frustrated with conflicting information or slow results and find it harder to commit to a healthier lifestyle. Motivate them to stick it out and put in the work now so it pays off later.

Start by identifying one area for the patient to focus on and provide tangible steps for them to work toward. Encourage habits that are feasible for them, and ask them to determine how a healthier lifestyle would benefit them personally. Making this connection could ultimately lead to a more positive outcome.

It can also be useful to have patients keep a journal of their fasting and postprandial blood sugars so they can track exactly what works for them (and what doesn't). This can help provide clarification for those who may otherwise be overwhelmed or unsure of where to start.

These patients must understand that improving their retinopathy, blood sugar and overall health is a slower process, with diabetic retinal changes taking more than six weeks to improve in most cases, but failing to do so could have destructive effects. It can help to check in with patients periodically to recap previous conversations and provide continued encouragement, all while building a good rapport.

Patients should be advised to collaborate with their PCP prior to engaging in any dietary or lifestyle intervention, as modification to insulin dosage or other medications may be necessary.

Here are five changes to advocate for all patients, but vulnerable patients in particular:

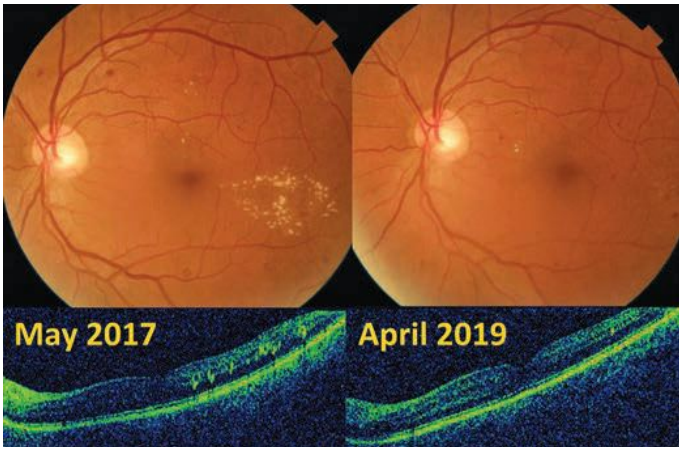
1. Cut Sugar Consumption

One of the first topics to address is excessive sugar consumption, including fructose and artificial sweeteners.[9-14] In 1822, consumption of added sugars in the American diet was equivalent to one 12oz can of soda every five days.[11] Today, the average American consumes this amount of sugar every seven hours.[11] Do not assume patients have already addressed excess sugar consumption in an effort to control their diabetes and optimize their health.

Fructose consumption often flies under the radar for patients trying to make healthy dietary changes. Average fructose consumption in the United States exceeds 50g per day and is higher among adolescents.[12] Excess fructose consumption is directly associated with elevated fasting blood glucose levels, hyperinsulinemia, metabolic syndrome and cardiovascular disease.[13,14] Limiting daily fructose intake to less than 20g per day (the equivalent of 1.5 apples) and avoiding products containing high-fructose corn syrup are good starting points for patients with metabolic problems, including type 2 diabetes.[13]

2. Limit Eating Time

Intermittent fasting and time-restricted eating (TRE) were common practices over 100 years ago in the pre-insulin days that were used by doctors to optimize health and longevity for their diabetic patients. Newer research shows that TRE remains promising as an effective adjunct therapy for controlling blood glucose levels in type 2 diabetes.



Mild diabetic retinopathy OS with early parafoveal macular edema in a 62-year-old male improved with TRE and carbohydrate restriction.

TRE can optimize insulin resistance, fasting blood glucose level, body composition and circadian rhythm.[15] TRE also improves cardiovascular biomarkers, such as total cholesterol, triglycerides, blood pressure and high sensitivity C-reactive protein (hs-CRP). The recent literature on meal timing is so promising that the American Heart Association advocates for it to optimize cardiometabolic health.[16]

The typical daily feeding window exceeds 15 hours on average.[17] In TRE, meal-timing is limited to an eight- to 12-hour window. Essentially, the patient's daily caloric intake remains the same, but breakfast is pushed later and dinner is pushed forward. Only one in 10 adults habitually maintains a 12-hour fasting window every day.[15]

A recent study looking at TRE in patients with metabolic syndrome found that timing meals within a 10-hour window (allowing a 14-hour nightly fast) over 12 weeks had the most favorable outcomes on many cardiometabolic markers.[15] These included improved insulin resistance, body mass index, low-density lipoprotein cholesterol and blood pressure.[15] Hemoglobin A1c was reduced by almost 1%, and liver enzymes, which are classic in non-alcoholic fatty liver disease, were reduced by roughly 10%.[15] Diet quality and physical activity remained stable.[15] No adverse events were reported.[15]

TRE may arguably be the easiest lifestyle intervention for patients to understand and implement, as they do not have to learn and adhere to a new diet or meal plan. This is the best option for patients who would rather change not what they eat, but when they eat.

3. Cut the Carbs

This is probably where you'll encounter the most resistance, but it cannot be ignored. The literature on the efficacy of dietary intervention in type 2 diabetes is vast and conflicting at times. Regardless of which nutritional approach patients adopt, studies seem to consistently demonstrate a direct relationship between carbohydrate restriction (a daily glycemic carbohydrate intake less than 45% of total calories) and improvements in insulin resistance and A1c.[18] Cardiovascular risk factors, including total cholesterol, triglycerides, blood pressure and hs-CRP, also significantly improve with carbohydrate restriction.[19] Tracking apps, such as "MyFitnessPal," are useful tools for helping patients understand their daily intake and identify where their calories are coming from.

One of the most heavily researched nutritional interventions for carbohydrate restriction is the Mediterranean-style dietary approach. By definition, the Mediterranean diet is lower in carbohydrates and higher in healthy fats.[20] Studies consistently show improvements in glycemic control, weight loss, hemoglobin A1c and other cardiovascular risk factors with this diet.[20] The Mediterranean diet is more efficacious than both low-fat and vegetarian-style dietary interventions for type 2 diabetes.[21] Other popular low-carb approaches include the paleo, whole30 and ketogenic diets.

Using telemedicine to educate patients with type 2 diabetes on the benefits of sustainable carbohydrate restriction, one company's recent two-year trial reported a remission rate in diabetes of approximately 7% and an average A1c reduction of 0.9%.[19] This is encouraging, given that less than 2% of patients with type 2 diabetes achieve long-term remission with current standard-of-care management.[22] By contrast, one in three patients with type 2 diabetes who undergo bariatric surgery achieve long-term remission.[23]

4. Optimize Sleep Schedule

Consistent, quality sleep is one of the most underrated factors when it comes to metabolic health. Getting seven to eight hours of sleep

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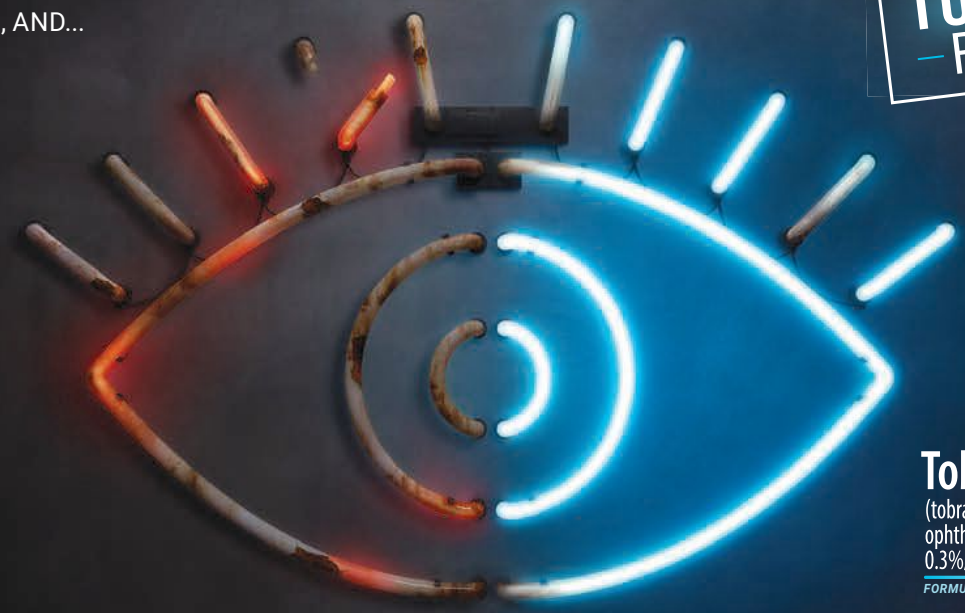
AND INFECTION, AND...



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Indications and Usage

For steroid responsive inflammatory ocular conditions of the palpebral and bulbar conjunctiva, cornea, and anterior segment of the globe and chronic anterior uveitis, corneal injury from chemical, radiation or thermal burns, or penetration of foreign bodies for which a corticosteroid is indicated and where the risk of superficial bacterial ocular infection is high or where there is an expectation that potentially dangerous numbers of bacteria will be present in the eye.

Important Safety Information

CONTRAINDICATIONS:
Most viral disease 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 disease of ocular structures. Hypersensitivity to any components of the medication.

WARNINGS & PRECAUTIONS:

- **IOP increase** – Prolonged use may result in glaucoma with damage to the optic nerve, defects in visual acuity and fields of vision. IOP should be monitored.
- **Aminoglycoside sensitivity** – Sensitivity to topically applied aminoglycosides may occur.
- **Cataracts** – Posterior subcapsular cataract formation may occur.
- **Delayed healing** – May delay healing and increase the incidence of bleb formation. Perforations of the cornea or sclera have occurred. Slit lamp biomicroscopy, and fluorescein staining should be conducted.
- **Bacterial infections** – May suppress host response and increase secondary ocular infections. In acute purulent conditions, steroids may mask infection or enhance existing infection. If signs and symptoms fail to improve after 2 days, the patient should be re-evaluated.
- **Viral infections** – Use with history of herpes simplex requires great caution. The course and severity of many viral infections of the eye (including herpes simplex) may be exacerbated.
- **Fungal infections** – Fungal infections of the cornea may occur and should be considered in any persistent corneal ulceration.
- **Use with systemic aminoglycosides** – Total serum concentration of tobramycin should be monitored.

ADVERSE REACTIONS:
The most frequent adverse reactions (<4%) to topical ocular tobramycin are hypersensitivity and localized ocular toxicity, including eye pain, eyelid pruritus, eyelid edema, and conjunctival hyperemia.

The reactions due to the steroid component are increased intraocular pressure with possible development of glaucoma, and infrequent optic nerve disorder; subcapsular cataract; and impaired healing.

The development of secondary infection has occurred. Fungal infections of the cornea may occur. Secondary bacterial ocular infection following suppression of host responses also occurs.

Non-ocular adverse events (0.5% to 1%) included headache and increased blood pressure.

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

[a]Randomized, investigator-masked, active-controlled, parallel-group trial conducted at 7 private practice clinical sites in the United States with 122 adult patients who had moderate to severe blepharitis/blepharoconjunctivitis.[1]

[b]Multicenter, double-blind, parallel-group, single-dose study of 987 patients receiving a single dose of TOBRADEX ST or TobraDex ophthalmic suspension.[2]

References: 1. Torkildsen GL, Cockrum P, Meier E, et al. *Curr Med Res Opin.* 2011;27(1):171-178. **2.** Scoper SV, Kabat AG, Owen GR, et al. *Adv Ther.* 2008;25(2):77-88.

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Brief Summary

This Brief Summary does not include all the information needed to use TOBRADEX ST safely and effectively. Please see Full Prescribing Information for TOBRADEX ST at MyTobraDexST.com.

INDICATIONS AND USAGE

TOBRADEX ST is a topical antibiotic and corticosteroid combination for steroid-responsive inflammatory ocular conditions for which a corticosteroid is indicated and where superficial bacterial ocular infection or a risk of bacterial ocular infection exists.

Ocular steroids are indicated in inflammatory conditions of the palpebral and bulbar conjunctiva, cornea and anterior segment of the globe where the inherent risk of steroid use in certain infective conjunctivitides is accepted to obtain a diminution in edema and inflammation. They are also indicated in chronic anterior uveitis and corneal injury from chemical, radiation or thermal burns, or penetration of foreign bodies.

The use of a combination drug with an anti-infective component is indicated where the risk of superficial ocular infection is high or where there is an expectation that potentially dangerous numbers of bacteria will be present in the eye.

DOSAGE AND ADMINISTRATION

Recommended Dosing: Instill one drop into the conjunctival sac(s) every four to six hours. During the initial 24 to 48 hours, dosage may be increased to one drop every 2 hours. Frequency should be decreased gradually as warranted by improvement in clinical signs. Care should be taken not to discontinue therapy prematurely.

CONTRAINDICATIONS

Nonbacterial Etiology: TOBRADEX ST 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.

Hypersensitivity: Hypersensitivity to any component of the medication.

WARNINGS AND PRECAUTIONS

IOP increase: Prolonged use of corticosteroids may result in glaucoma with damage to the optic nerve, defects in visual acuity and fields of vision. IOP should be monitored.

Aminoglycoside sensitivity: Sensitivity to topically applied aminoglycosides may occur.

Cataracts: May result in posterior subcapsular cataract formation.

Delayed healing: May delay healing and increase the incidence of bleb formation after cataract surgery. In those diseases causing thinning of the cornea or sclera, perforations have been known to occur with the use of topical steroids.

Bacterial infections: May suppress the host response and thus increase the hazard of secondary ocular infections. In acute purulent conditions, steroids may mask infection or enhance existing infection. If signs and symptoms fail to improve after 2 days, the patient should be re-evaluated.

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

Fungal infections: Fungal infections of the cornea are particularly prone to develop with long-term use. Fungal invasion must be considered in any persistent corneal ulceration.

Use with systemic aminoglycosides: Use with systemic aminoglycoside antibiotics requires monitoring for total serum concentration of tobramycin.

ADVERSE REACTIONS

The most frequent adverse reactions to topical ocular tobramycin (TOBREX®) are hypersensitivity and localized ocular toxicity, including eye pain, eyelids pruritis, eyelid edema, and conjunctival hyperemia. These reactions occur in less than 4% of patients. Similar reactions may occur with the topical use of other aminoglycoside antibiotics.

Non-ocular adverse events occurring at an incidence of 0.5% to 1% included headache and increased blood pressure.

The reactions due to the steroid component are: increased intraocular pressure (IOP) with possible development of glaucoma, and infrequent optic nerve disorder; subcapsular cataract; and impaired healing.

Secondary Infection.
The development of secondary infection has occurred. Fungal infections of the cornea are particularly prone to develop with long-term use. Fungal invasion must be considered in any persistent corneal ulceration. Secondary bacterial ocular infection following suppression of host responses also occurs.

USE IN SPECIFIC POPULATIONS

Pregnancy and Nursing Mothers

There are no adequate and well controlled studies in pregnant women. TOBRADEX® ST ophthalmic suspension should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus. Caution should be exercised when TOBRADEX® ST is administered to a nursing woman.

Pediatric Use: Safety and effectiveness in pediatric patients below the age of 2 years have not been established.

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

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per night is crucial for patients looking to manage their type 2 diabetes and overall health.[24] The risk of developing or worsening diabetes increases with sleep durations outside this range.[24]

When patients report poor quality sleep, it opens the door for optometrists to discuss potential underlying issues, such as sleep apnea and excessive exposure to blue light at night. Sleep apnea can be a common comorbidity in metabolic syndrome, diabetes and glaucoma, so referring appropriate patients for a sleep study could have a significant impact on their health. Explaining that blue light can cause circadian rhythm disruption and melatonin suppression can open the conversation up to the importance of blue-blocking lens technologies or apps for use before bedtime if screen time is unavoidable.[25,26]

5. Value Consistent Exercise

The discussion would be incomplete without addressing the importance of regular exercise and movement. Some patients may feel discouraged by or overwhelmed with a new fitness routine. It is important to convey that even basic exercises, such as walking for 20 minutes per day, can significantly benefit patients' metabolic health.[27] Consistency matters more than intensity of exertion.

Comorbidities (e.g., rheumatoid arthritis, obesity) may prevent patients from engaging in prolonged periods of weight-bearing movement. Non-weight-bearing exercises such as swimming, aqua aerobics and stationary cycling are viable alternatives. Remind proactive patients that whatever they enjoy doing to stay active will likely be the most sustainable routine for them moving forward.

Depending on practice modality, office location and insurance plan, some patients may even be eligible for discounted or free gym memberships. Share this with patients so they know their options.

As integral members of the healthcare team, optometrists have been encountering a growing number of patients presenting with uncontrolled (and undiagnosed) metabolic diseases, such as type 2 diabetes. No longer can we defer to a patient's other healthcare providers to discuss the importance of evidence-based behavioral changes in managing their health. By educating patients on the risk of permanent vision loss due to their diabetes, we can help them lessen the risk of or altogether avoid long-term ocular complications. ■

Dr. Cornwell graduated from the New England College of Optometry in 2015 and completed a residency in ocular disease with the Indian Health Service. He works at a rural community health center in northern California where he provides eye care to populations in need and helps patients manage ocular manifestations of various systemic diseases.

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26th Annual Glaucoma Report

Glaucoma: The Perils of Progression

Controlling this disease requires a long-term, fluid management plan. These six tips can help you navigate the complicated road ahead.

By Brian D. Fisher, OD, David W. Johnson, OD, and April J. Fisher, OD

Open-angle glaucoma (OAG) is a chronic and visually devastating disease with minimal symptoms until it reaches the advanced stage. The goal throughout treatment is to stave off progression and ensure a lifetime of preserved vision.[1]

But once progression is detected, the practitioner is faced with a challenging decision: re-educate the patient on the current regimen to boost medication adherence or change the treatment course. Thoroughly educating patients about the progressive nature of glaucoma and its treatments can help patients understand the importance of medication compliance.

If compliance is not the issue, clinicians should ensure the patient is using proper drop instillation techniques, as some patients may struggle with dexterity.

Once clinicians address these issues, they can then reconsider the efficacy of the medication regimen prescribed.

Before considering a change in the current management, clinicians must evaluate the risk factor profile for progression, target intraocular pressures (IOPs), and medication adherence and burden. Other important considerations include the potential benefits and risks of surgery and the risks of functional vision impairment if left untreated vs. age-related decline.[2] This article discusses six considerations to help clinicians navigate the complicated management decisions necessary once a patient shows signs of glaucoma progression.

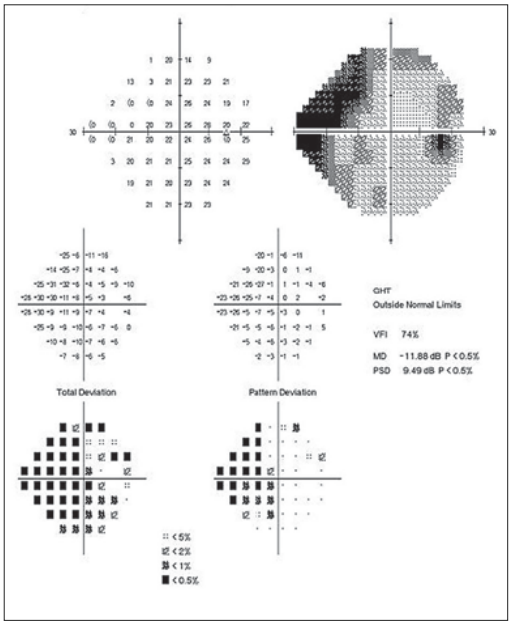

Fig. 1. This patient's 24-2 field shows moderate visual field damage with central involvement.

1. Assess Risk Factors

Unfortunately, disease progression isn't always cut-and-dry. Many patients progress slowly with little impact on their vision while others progress rapidly with devastating consequences.[1] To help detect rapid and severe progression, clinicians should perform three visual fields in the first year (i.e., at diagnosis, six months and 12 months); in year two, patients should have one visual field every six months. If the examination rules out rapid progression (i.e., greater than 0.5dB/year on mean deviation or pattern standard deviation), clinicians can scale back to one visual field per year if the patient remains stable (i.e., 0.1dB/year).

Thus, one of the most important factors in advancing glaucoma is the rate of progression. Ancillary testing

with optical coherence tomography (OCT) and visual fields can help clinicians document structural and functional changes associated with progression (*Tables 1-3*).

Once progression is determined, clinicians must consider the patient's age, general health status, life expectancy and expected rate of decline with current treatment to design the best adjunctive therapeutic approach based on each patient's risk of visual decline.[1,2] For example, patients aged 70 or older with slowly progressing glaucoma likely require less intense treatment or sometimes no additional treatment at all, while young glaucoma patients with fast progressing disease require quick action, an aggressive approach and possibly surgery.

Certain optic disc features can indicate a higher risk of visual decline in glaucoma patients. These include an increasing vertical cup-to-disc ratio with preferential rim loss to the inferior, inferotemporal, supratemporal and superior regions, the presence of Drance hemorrhages, increasing size of the parapapillary beta zone and new localized retinal nerve fiber layer (RNFL) defects.[3]

Other important clinical features putting the patient at risk for further disease progression include severe staging at the time of diagnosis, type of glaucoma (i.e., pseudoexfoliation, pigment dispersion) and large mean deviation (<-12.00dB) on perimetry. Higher peak and average IOPs at baseline, higher mean IOP or large IOP variation also put the patient at a higher risk for visual decline.[1,2]

2. Be Wary of Target IOPs

Studies show each 1mm Hg of increased IOP is associated with a 10% to 19% increased risk of progression.[4] The best IOP for each patient isn't necessarily a static number—it's a balance between the risk of decreased vision related quality of life due to glaucoma and the risks of treatment.[5] While insufficient evidence shows setting target IOPs is associated with better clinical outcomes, clinicians must consider the risks and benefits before establishing a target IOP for each patient.[5] Considerations include short and long-term IOP fluctuations, inter-observer

Table 1. OCT RNFL Progression[19-25]

- No current reference standard on limit of RNFL thinning that confirms progression.
- Event-based change: repeatable inter-visit change in average thickness ≥5µm.
- Trend-based change of global average thickness loss equaling 2µm to 3µm/year.
- Widening of existing thinning and defect on guided progression analysis. Inferotemporal widening and thinning is more common than supratemporal.

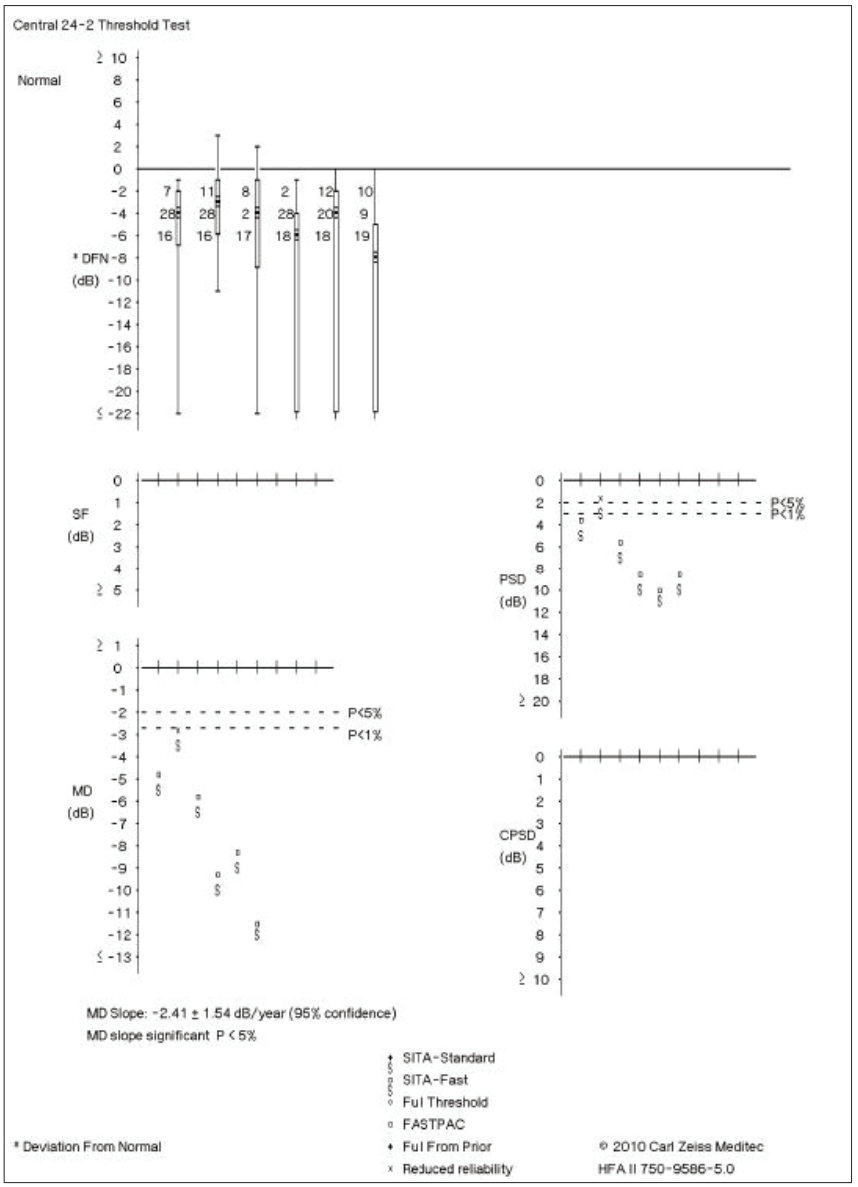

Fig. 2. This visual field readout shows trend-based change, which indicates visual field progression.

variability, patient life expectancy and treatment adherence.

When resetting target IOPs after adjusting a patient's glaucoma regimen, clinicians must evaluate the amount of glaucomatous damage, the average range of IOPs at which glaucomatous damage is occurring and the status of the fellow eye.[5]

Researchers suggests target IOPs may be particularly useful for patients at high risk of substantial vision loss and blindness.[5] For those with low risk for visual loss, clinicians may do better focusing on reducing treatment side effects rather than achieving a particular IOP.[5]

To complicate matters further, a patient's target IOP will likely change over time, especially if they experience accelerated progression with the current target or if the fellow eye's visual status becomes significantly reduced.[4]

Target IOPs are useful broad guidelines in OAG therapy but should not be used in isolation from other information. Serial ancillary testing can help clinicians highlight progression and modify therapeutic measures when indicated.

With an appropriate IOP target range and continuous reassessment, glaucoma progression can be considerably slowed to reduce the probability of decreased vision-related quality of life.[4]

Shedding LiGHT on SLT

The LiGHT Study Group conducted a large, prospective, randomized controlled trial with 718 patients (1,235 eyes) to compare standardized 360° SLT with eye drops in treatment-naïve patients. The majority of patients in each treatment arm were diagnosed with either OHTN or mild OAG—approximately 30% and 50%, respectively. Prostaglandin analogs were offered as the primary topical agent followed by adjunctive therapy with β-blockers, then carbonic anhydrase inhibitors or α-agonists. The patients were monitored for three years.[7] SLT was not associated with any serious adverse events, but approximately one-third of patients experienced transient effects such as discomfort, blur, photophobia and ocular hyperemia.

The SLT-first group experienced fewer drop-related side effects (5.7%) compared with the medication-first arm (20.2%), likely secondary to the reduction in the mean number of drops necessary in the former group.[7] This is consistent with reports from pooled analyses comparing SLT with eye drops for OAG, including data from the LiGHT Study Group, demonstrating that SLT is effective at significantly reducing the number of topical medications necessary for adequate IOP control.[8] The percentage of visits at target IOP was slightly higher for the SLT group when compared with the medication-first group: 93% vs. 91.3%.

Fewer treatment escalations occurred in the SLT-first arm, none of which led to trabeculectomy compared with 11 eyes in the medication-first group. Ultimately, 74% of patients treated with SLT first were stable at three years without using any topical therapy. A second SLT was necessary in 25.7% of eyes. There were no significant differences in visual acuity, IOP or mean deviation loss on visual field testing between the two groups at the study's conclusion.[7]

The LiGHT Study Group did not report data on medication adherence or persistence, which can significantly impact treatment escalations and outcomes. Studies show as few as 33% to 39% of patients persist with the initially prescribed medication at one year.[28]

The LiGHT trial design is clinically relevant due to its individualized treatment approach in which patients with more severe disease were assigned a lower initial target IOP with modifications made according to widely accepted and implemented clinical guidelines. Furthermore, it measured SLT success as controlling progression of neuropathy, rather than a percentage of IOP reduction. By stratifying these data based on disease stage, it showed a single SLT was far more likely to result in a controlled status without drops at three years in patients having either OHTN (72.8%) or mild OAG (64.3%) when compared with eyes with moderate (33.3%) and severe (9.6%) OAG.[7]

That is not to say SLT was ineffective in lowering IOP in more advanced stages. The mean treatment effect was similar among all stages (approximately 8mm Hg). It more likely reflects a standalone inability to meet the more stringent IOP goals newly diagnosed advanced disease warrants.[6]

In another study, 180° SLT was successful in 50% of eyes with advanced OAG when measured against the criteria of 30% IOP reduction from pre-treatment value and <18mm Hg.[29] However, randomized controlled trial studies comparing SLT with medication-only treatment groups largely include milder cases of glaucoma, so further research is necessary to elucidate the role of SLT in advanced cases; for now, filtration surgery remains the standard in the context of progressive neuropathy.[8,29]

3. Set New Baselines

Once new therapy is initiated, clinicians must establish new baselines for perimetry, OCT RNFL and ganglion cell analysis (GCA), and photo documentation. The practitioner does not need to perform additional tests when setting these new baseline parameters. Instead, they can reference the last two tests performed to set a new baseline.[1,2] Furthermore, guided progression analysis will support the analysis for trend-based change with these tests.

4. Consider SLT

Selective laser trabeculoplasty (SLT) was approved by the FDA in 2001 and has since proven itself an effective method for lowering IOP.[6] SLT is often considered in cases of inadequate IOP reduction with medications, intolerance, allergy or poor adherence to medications (e.g., due to cost, cognitive decline, insufficient dexterity or tremor) and may be recommended at various points in the

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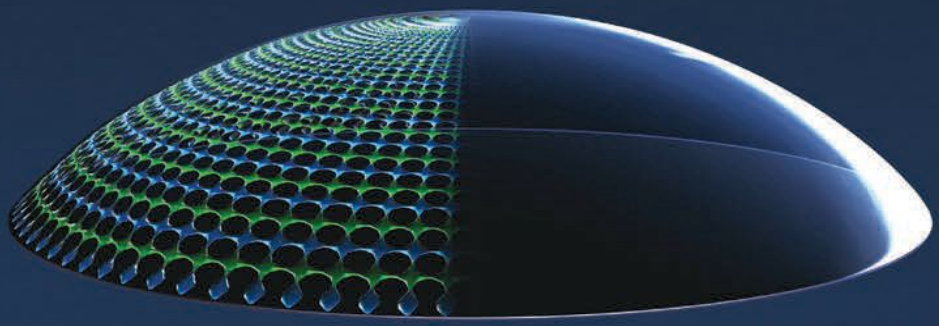

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Table 2. OCT GCA Progression[19-25]

- No current reference standard on limit of GCA thinning that confirms progression.
- Event-based change: repeatable inter-visit change in average thickness ≥4µm.
- Trend-based change of global average loss equaling 1µm to 1.5µm/year.
- Widening of existing ganglion cell-inner plexiform layer thinning and defect. Inferotemporal more common than supratemporal due to inferotemporal axons projecting to the macular vulnerability zone.

treatment arc, including as the initial treatment option.

Currently, SLT is less commonly offered as first-line therapy compared with topical medications for ocular hypertension (OHTN) or OAG. Recent evidence suggests SLT should be considered as a safe, effective alternative to medication as a primary therapy for a large subset of these patients.[7,8]

The Laser in Glaucoma and Ocular Hypertension Trial (LiGHT) Study Group found SLT could successfully arrest progression in 74% of patients with OHTN and newly diagnosed OAG for a period of at least three years without medications—a finding that should encourage providers to consider SLT as first-line therapy.[7]

Clinicians must consider many factors before recommending SLT to a patient, but they have fewer factors to consider if the goal is an attempt to eliminate glaucoma medication burden. It is well-established that a high baseline IOP positively correlates with the conventional measure of SLT success of ≥20% IOP reduction.[9]

The LiGHT Trial shows us that patients with OHTN and mild OAG are the most likely cohorts to achieve drop-free disease control at three years.[6] Patients with more advanced disease or lower baseline IOP can still benefit from SLT but may need adjunctive medical therapy; however, it is likely fewer drops will be necessary—relatively sparing the ocular surface and potentially improving the patient's medication adherence.[7,8]

5. Do Your MIGS Research

In particular circumstances, minimally invasive glaucoma surgery (MIGS) may be a good option for mild to moderate glaucoma patients undergoing cataract surgery. A recent study shows 22% of cataract surgeries performed by glaucoma specialists in 2016 included a MIGS procedure.[10] Numerous MIGS procedures exist, and they are minimally traumatic to the surrounding tissue and exhibit minimal tissue disruption. The various safety profiles are excellent compared with incisional surgery and glaucoma drainage device implantation.[11-13] Wound healing is rapid with good preservation of vision.[11-13]

Furthermore, MIGS are combined with cataract surgery, and efficacy shows moderate to high IOP-lowering capabilities. One meta-analysis shows a decrease in IOP and a reduction in glaucoma medications after MIGS surgery with low complication rates.[14]

This therapeutic option could allow a significant number of OAG patients to reduce their medication burden with a lower risk of complications.[3,14]

6. Prepare for the Last Defense

Medical therapy is effective for the majority of glaucoma patients, but surgical means are recommended when patients experience fast rates of functional and/or structural progression, central visual field loss, suboptimal hypotensive IOP control with medical therapy and SLT, or they have uncontrolled moderate-severe disease.[15]

Incisional glaucoma filtration surgery includes trabeculectomy and glaucoma drainage devices such as the Ahmed glaucoma valve or Baerveldt glaucoma implant.[11]

Despite an increasing safety profile over the years, these techniques have higher postoperative risks compared with nonincisional surgeries, such as late-onset bleb infection and endophthalmitis, hypotony maculopathy, choroidal effusion or hemorrhage, flat anterior chamber, corneal damage and cataract.[11]

Because of these risks, incisional

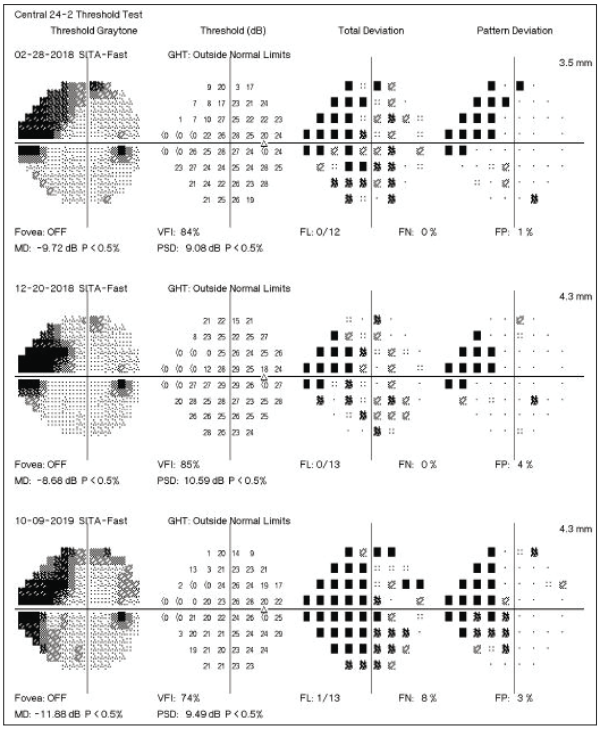

Fig. 3. This visual field readout shows event-based change, indicating visual field progression.

Table 3. Visual Field Progression[26]

- Requires at least three visual fields.
- The last two consecutive visual fields must be reliable and repeatable.
- Event-based change of new defects in a previous normal area showing:
  - 1-point with greater than 10dB regression.
  - Within the central 10° with two or more points with more than 5dB regression.
  - Outside the central 10° with three or more points with more than 5dB regression.
- Event-based change within existing defects:
  - 1-point with greater than 15dB regression.
  - Within the central 10° with any point with more than 10dB regression.
  - Outside the central 10° with three or more points with more than 10dB regression on two consecutive fields or more than 5dB on three consecutive fields.
- Trend-based change on guided progression analysis of:
  - Slope of p<1% on visual field index.
  - "Likely progression."
- Glaucoma Rate Index[27]
  - Newer method to detect long-term visual field progression in glaucoma.
  - Studies show it can provide earlier detection compared with point linear regression and guided progression analyses.
  - Validation with future studies for generalized use are still needed.

surgery is reserved for those with rapidly progressing glaucoma regardless of stage, those with severe glaucoma who failed with medical and noninvasive therapies and those with risk of visual impairment due to progressing central visual field loss.[16,17] Furthermore, incisional surgery requires intense postoperative healing, wound modulation and strict follow-up exams to manage postoperative complications.

There can be more disadvantages than benefits in the mild to moderate glaucoma patient or for patients who want to reduce the medication burden because therapeutic efficacy can gradually decrease over time, requiring repeat surgery.[17]

Despite these disadvantages, incisional surgery does provide an IOP reduction of 30% to 50% and should be strongly considered when the benefits of surgery outweigh the risks.[18] Therapeutic surgical management should not only maintain the patients' visual field and functional vision but also preserve their quality of life and independence.[12,13] Clinicians must weigh the risks and benefits of incisional surgery and only recommend these options when absolutely critical to stabilize aggressive glaucomatous progression.

Optometrists' primary goal in the management of glaucoma is to ensure a lifetime of visual function to meet patients' visual demands. No perfect formula exists to determine which therapeutic approach is best. By evaluating patients' risk for visual decline, medication adherence and burden, and the pros and cons of surgery, clinicians can individualize a therapeutic plan to address any apparent progression—and preserve vision as long as possible. ■

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Dr. April Fisher is an optometrist at Ocala West VA Community Based Outpatient Clinic, Ocala, Fla.

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What's Your Disc Diagnosis?

These cases can help you better differentiate tough optic disc abnormalities.

By Ashley Kay Maglione, OD, and Kelly Seidler, OD

Accurate evaluation of the optic disc is a critical part of optometric practice. When a disc is not "perfused, healthy, distinct and flat," it can be difficult to differentiate between anatomic variations and pathology. Clinicians must take a systematic approach to optic disc evaluation, carefully assessing the margins, color of the neuroretinal rim, cup-to-disc ratio and overall size of the nerve. This case-based review provides photos and clinical pearls to help you enhance your assessment of optic disc abnormalities.

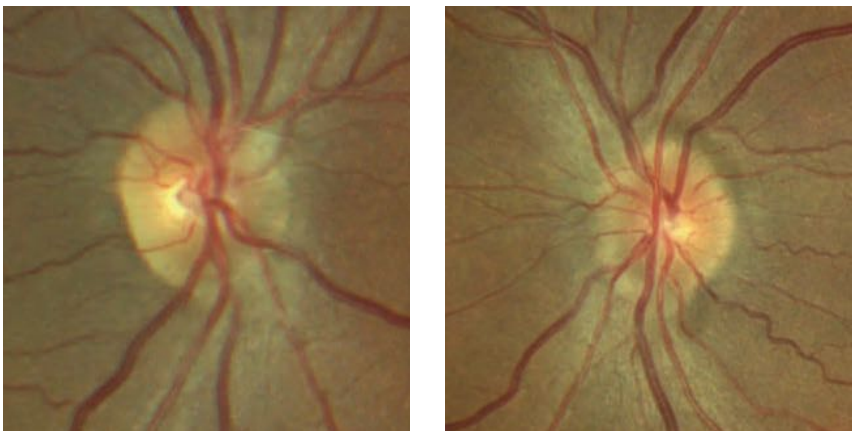

Fig. 1. Despite unremarkable entrance testing with no visual complaints, the patient's optic disc evaluation shows mild elevation with sectoral blurred margins nasally and superiorly OU.

Case 1: Surprise Elevation

A 43-year-old African American female presented for her annual exam without any visual complaints. Her health history was remarkable for hypothyroidism, asthma and iron-deficiency anemia. Entrance testing was unremarkable with 20/20 vision uncorrected OD and OS, full confrontation visual fields bilaterally and no relative afferent pupillary defect.

She exhibited 90% of normal abduction bilaterally with no diplopia and a 2 to 4 prism diopter comitant esophoria in all gazes. Her anterior segment exam was remarkable for palpebral conjunctival pallor, consistent with her history of anemia. Intraocular pressures (IOPs) measured 16mm Hg OD and 15mm Hg OS. Her blood pressure was elevated at 142/86mm Hg RAS. She was above her ideal body weight at 240lbs. Her dilated fundus exam revealed subtle elevation more so in the left eye than the right (*Figure 1*).

Discussion. The optic disc photos demonstrate elevation OS>OD, raising the question of papilledema vs. pseudopapilledema (*Table 1*). When asked about symptoms of increased intracranial pressure—such as headaches, pulsatile tinnitus, nausea, vomiting, diplopia and transient visual obscurations—the patient reported occasional headaches and "seeing stars" when she bent down. She denied any other symptoms or use of tetracyclines, vitamin A derivatives or oral

contraceptives. She exhibited no spontaneous venous pulse (SVP). In addition, upon analysis of the vitreoretinal interface on optical coherence tomography (OCT), she demonstrated subtle peripapillary wrinkles in the left eye, suggestive of mild papilledema (*Figure 2*).

Due to her ocular findings, she was sent for urgent neuroimaging that included magnetic resonance imaging (MRI) of the brain and magnetic resonance venography of the head to rule out a mass and venous sinus thrombosis. MRI is the preferred neuroimaging modality due to superior soft tissue resolution and better visualization of particular findings consistent with intracranial hypertension such as optic nerve sheath distension, empty sella and posterior globe flattening.[1] Imaging showed no evidence of intracranial mass or venous sinus thrombosis; however, she did exhibit low-lying cerebellar tonsils concerning for Chiari I malformation.

Chiari 1 malformation is eight times more common in patients with pseudotumor cerebri than the general population, suggesting a relationship.[2,3] The coexistence of Chiari 1 malformation and pseudotumor cerebri in patients with papilledema can create diagnostic and treatment dilemmas. In patients with papilledema, Chiari I malformation may be considered causative if there is obstruction of cerebrospinal fluid (CSF) flow.[2,3]

In this patient, lumbar puncture was deferred due to cerebellar ectopia; however, CSF flow was considered patent. Therefore, she was scheduled for follow-up with neurology on an outpatient basis for presumed idiopathic intracranial hypertension. Treatment was initiated with acetazolamide and weight loss was recommended.

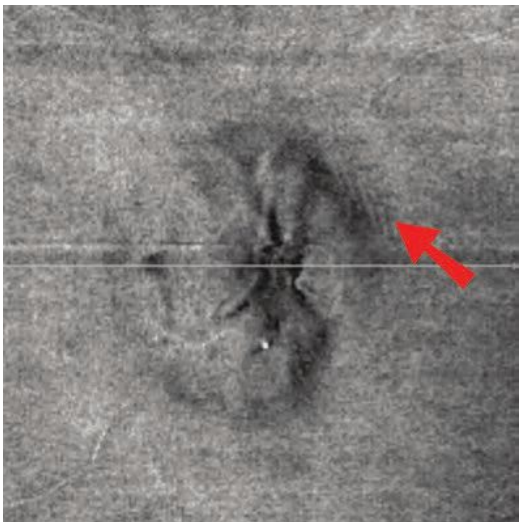

Fig. 2. In this patient's *en face* OCT vitreoretinal interface image of the left eye, note the wrinkles superior temporal, consistent with Paton's lines or peripapillary wrinkles that are not readily visible funduscopically.

The presence of peripapillary wrinkles on the vitreoretinal interface was important in raising the suspicion of papilledema on initial exam. Subsequent close optometric monitoring with assessment of the afferent system, dilated fundus exam and serial OCT scans is now indicated to assess the effectiveness of treatment and thus verity the working diagnosis.

Case 2: Problem Rising to the Surface

A 17-year-old Caucasian female presented for a routine examination. Her health history was unremarkable, and her only medication was oral contraceptives. She denied symptoms of increased intracranial pressure.

Her best-corrected visual acuity was 20/15 OD and OS. She exhibited a subtle, 0.3 log unit relative

Table 1. Papilledema vs. Pseudopapilledema

Differentiating these clinical entities can be quite challenging. Causes of pseudopapilledema are relatively benign and include buried optic disc drusen and small, anomalous and/or hypoplastic discs. Papilledema is, by definition, optic disc edema in the setting of increased intracranial pressure and is a medical emergency. Several tests can aid in the evaluation:

- OCT of the peripapillary RNFL can be particularly helpful, as the en face images can reveal subtle peripapillary wrinkles that would otherwise be difficult to view funduscopically.
- Fundus autofluorescence may help to highlight optic disc drusen, which appear hyper-autofluorescent. However, patients with optic disc drusen may also have overlying edema, and the buried disc drusen will not hyper-autofluoresce.
- B-scan remains the standard for assessing presence of buried optic nerve head drusen.
- As technology improves, OCT is increasingly used in the diagnosis of optic disc drusen. The ODDS Consortium recommends the use of enhanced-depth OCT imaging for adequate visualization of disc drusen. With OCT, the optic disc drusen appear as hyporeflective structures with a hyperreflective margin. Additionally, no RNFL thickness value reliably differentiates papilledema from pseudopapilledema.
- The presence of an SVP viewed upon direct ophthalmoscopy may suggest that an elevated disc is not secondary to increased intracranial pressure. Clinicians should view several rhythmic beats of a vein, as eye movement can occasionally mimic the appearance of a non-sustained pulse.

afferent pupillary defect in the left eye. Humphrey automated visual fields revealed a normal field in the right eye and a superior nasal defect with an enlarged blind spot in the left (*Figure 3*). Efferent testing was unremarkable with no abduction deficit. She exhibited no proptosis or ptosis. Pressures were 14mm Hg bilaterally.

Her dilated fundus exam revealed significant findings (*Figures 4 and 5*).

Discussion. While also exhibiting indistinct margins of the optic disc as in case 1, this patient's presentation was attributed to optic disc drusen. Fundus autofluorescence (FAF) was an important examination element in this case, as it helped to confirm the presence of superficial drusen in the left eye.

In the absence of drusen, the optic disc appears dark on fundus autofluorescence whereas superficial drusen appear bright, or hyper-autofluorescent. The Optic Disc Drusen Studies Consortium (ODDS) found that the majority of eyes with one superficial druse also had at least one buried druse.[4] Deeply buried drusen are not visible with FAF, but B-scan ultrasonography can be used to detect buried drusen and is often indicated.[5]

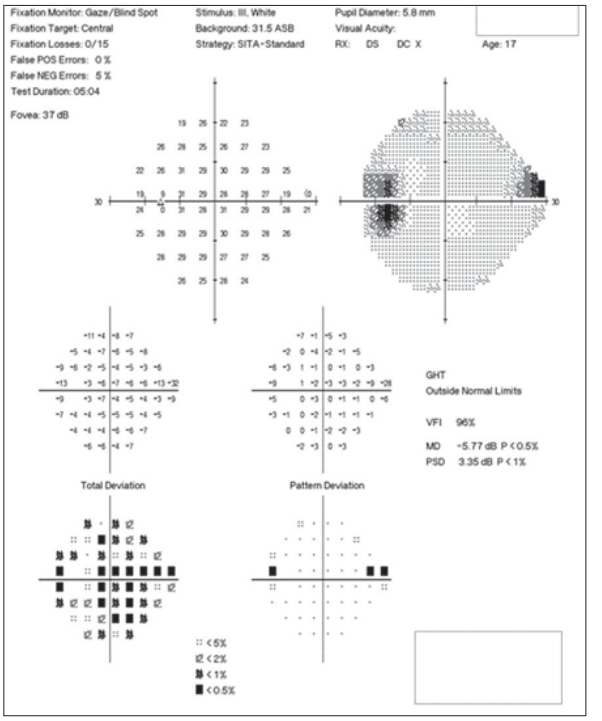

Fig. 3. The patient's 24-2 visual field OS demonstrates a small superior nasal step and enlarged blind spot.

Optic disc drusen are small, calcified deposits that become more apparent with age. They are typically buried during childhood and may initially appear as an optic disc with indistinct margins. With age, they gradually become more superficial and present with a bumpy appearance.

Optic disc drusen may predispose a patient to visual field defects, as seen in this patient, nonarteritic anterior ischemic optic neuropathy (NAION), subretinal hemorrhages and peripapillary choroidal neovascular membranes.[6]

Table 2. True Disc Edema vs. Traction

Unilateral Disc Edema	Vitreopapillary Traction
Decrease in afferent function.	Afferent function is largely intact in isolated VPT.
Sectoral or diffuse elevation of optic disc.	Tractional elevation of disc seen on OCT.
Often associated with vasculopathic, infectious and inflammatory etiologies, which should be assessed with appropriate blood work.	Often without associated pathology but may be seen in diabetic and other retinopathies.

Clinicians must also consider the possibility of a simultaneous presentation of disc edema and disc drusen; however, in this case overlying papilledema was considered less likely given the presence of a definite SVP and the absence of symptoms associated with increased intracranial pressure. Nonetheless, close serial monitoring of the disc appearance, afferent system and OCT is indicated to ensure no atypical progression.

Case 3: Systemic Suspicion

A 76-year-old Caucasian male presented with a complaint of a progressively worsening "cloud" over his vision OS. He had seen another eye care provider approximately two months prior and the previous fundus photo demonstrated diffuse disc swelling OS. His ocular history was otherwise remarkable for a traumatic retinal detachment OD with resultant poor vision.

His medical history was remarkable for seropositive generalized myasthenia gravis. He was diagnosed four months prior and was treated with pyridostigmine as well as prednisone and intravenous immunoglobulin during exacerbations. His history was also significant for orthostatic hypotension.

His best-corrected visual acuity was counting fingers at one foot OD and 20/40- OS. The exam revealed a 3+ afferent pupillary defect OD. Confrontation visual fields were severely restricted OD and exhibited inferior constriction OS (*Figure 6*).

His anterior segment exam was unremarkable OS. Upon dilated

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fundus examination, the disc was flat and distinct OD, while the disc showed significant findings OS (*Figure 7*).

Discussion. The patient was diagnosed with sectoral disc edema in the left eye evidenced by blurred hyperemic disc margins inferiorly.

Note that there is no longer swelling of the superior neuroretinal rim as documented by previous examination, and it appears that he has subsequently developed pallor with a corresponding inferior visual field defect.

Potential differentials of unilateral disc edema include arteritic and non-arteritic AION and optic papillitis. Arteritic AION was considered less likely as the patient did not present with symptoms of giant cell arteritis (GCA) such as headache, jaw claudication, scalp tenderness, weight loss, reduced appetite, fatigue, amaurosis fugax or pallid disc swelling. However, given his age and the potential devastating consequences, testing to rule out GCA—in the form of serum platelet, ESR and CRP studies—was indicated and ordered.

Non-arteritic AION may be considered in this case given the patient's age and history of orthostatic hypotension; however, given his monocular status and risk of further vision loss, laboratory workup to rule out any potential etiology of papillitis was indicated.

Patients with papillitis often present with complaints of vision loss, and the examination will demonstrate a corresponding decrease in visual acuity, visual field loss and an afferent pupillary defect. Optic papillitis can be caused by inflammatory conditions, such as sarcoidosis, and infectious diseases, such as Lyme disease and syphilis.[7]

The patient was asked to complete laboratory testing that included ESR, CRP, FTA-ABS, RPR, ACE, Lyme titer and ANA.

If suspicion for sarcoidosis is high, consider ordering chest imaging, as ACE can be falsely low. Results were remarkable for elevated Lyme disease IgG and IgM antibodies on Western blot.

While causation may be difficult to establish with certainty here, it was important to identify his systemic Lyme infection, which could, if untreated, lead to further vision loss in a patient with already significant vision impairment.

Case 4: Gaining Traction

A 52-year-old Caucasian male presented for evaluation of binocular vertical diplopia that began following a recent stroke. His medical history was remarkable for type II diabetes, hypertension, hypercholesterolemia, a right thalamic stroke, a myocardial infarction, asthma, sleep apnea, anxiety, depression and schizophrenia.

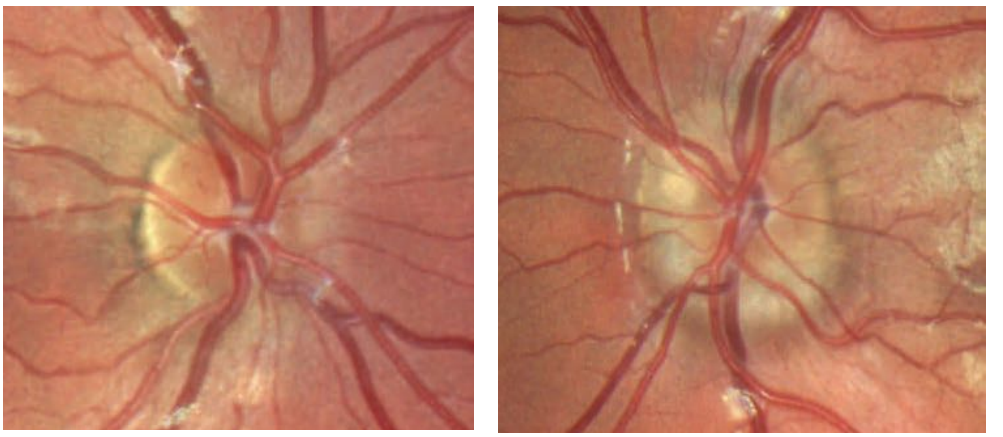


Fig. 4. These color fundus photos of the optic discs show that the margins of the right optic disc, at left, are indistinct nasally but are otherwise preserved temporal. The left optic disc, at right, has more indistinct margins with a notable superficial druse superior nasal.

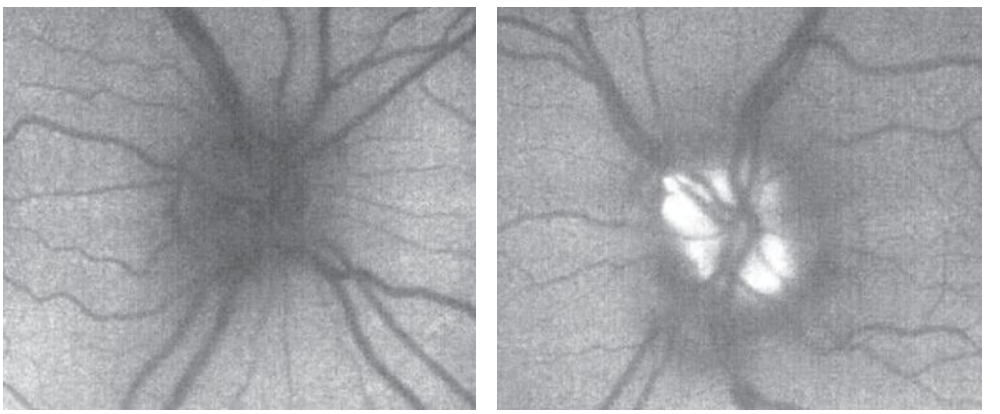


Fig. 5. The patient's fundus autofluorescent photos demonstrate significant autofluorescence of the left optic disc, suggestive of more prominent drusen than what is evident funduscopically.

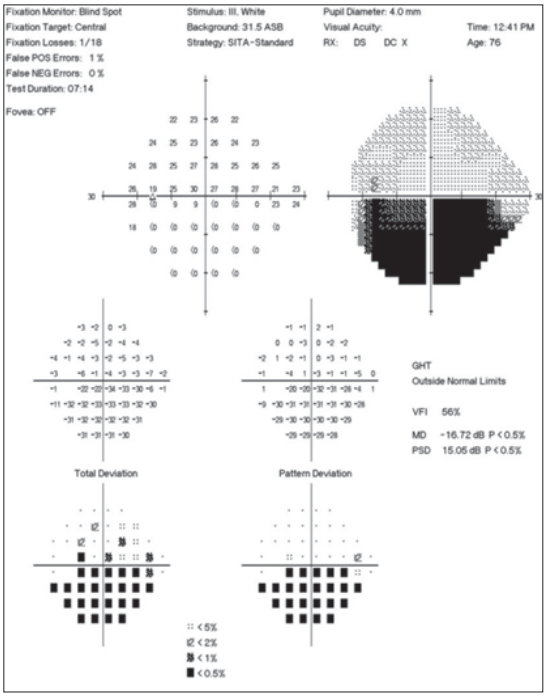


Fig. 6. The 24-2 automated visual field demonstrates an inferior defect OS.

Table 3. Pallor vs. Pseudo-pallor

Pallor	Pseudo-pallor
OCT typically shows loss of peripapillary RNFL and GCL.	OCT may show sloping of the neuroretinal rim, scleral crescent or large cupping without loss of RNFL.
Corresponding afferent dysfunction (reduced visu-al acuity, dyschromatopsia, visual field defect, af-ferent pupillary defect, reduced brightness sense or red desaturation).	If afferent testing is intact and pseudopallor is suspected, serial monitoring can be used to confirm the diagnosis.
May be associated with infectious, inflammatory, compressive and toxic/nutritional etiologies, which should be assessed with appropriate blood work and neuroimaging.	Associated with tilted discs, high myopia, pseudophakia, large physiologic cupping.

The patient's best-corrected visual acuity was 20/20 OD and OS. There was no afferent pupillary defect and confrontation visual fields were normal, as was color vision.

Efferent testing demonstrated a vertical misalignment diagnosed as skew deviation attributed to his history of known right thalamic stroke. His dilated fundus exam was unremarkable in the right eye. His left eye showed significant changes in optic disc appearance and OCT imaging (*Figure 8*).

Discussion. As with case 3, this patient also exhibited sectoral disc elevation; however, the etiology is not true disc swelling but is instead tractional in nature. Vitreopapillary traction (VPT) is a condition caused by adherence of a fibrotic membrane or incomplete posterior vitreous detachment that raises the optic disc margin. This patient's tractional elevation, induced by partial detachment of the posterior hyaloid face, can be visualized on the 5-line OCT raster.

In addition to elevation of the optic disc, VPT can result in indistinct optic disc margins and peripapillary hemorrhage, making it difficult to differentiate from true optic disc swelling, such as in AION and optic papillitis (*Table 2*).[8]

Therefore, clinicians must rule out these etiologies with serum lab testing. As such, CBC, ESR, CRP, ACE, ANA and RPR were ordered and unremarkable in this case. His negative blood work results, along with normal afferent function, helped to support the diagnosis of VPT as afferent visual function is often affected in cases of AION and optic papillitis.[9]

VPT has been described in both eyes without ocular pathology, as well as in eyes with pathology that may result in fibrotic membrane proliferation such as diabetic retinopathy and vein occlusion. Given the presence of telangiectatic vessels on the optic disc and systemic history, concurrent diabetic papillopathy could be considered in this case; but ultimately, long-term follow-up was helpful in excluding this diagnosis.[10,11]

This patient's case demonstrates the importance of considering VPT in optic disc elevation and looking closely at the vitreoretinal interface on OCT.

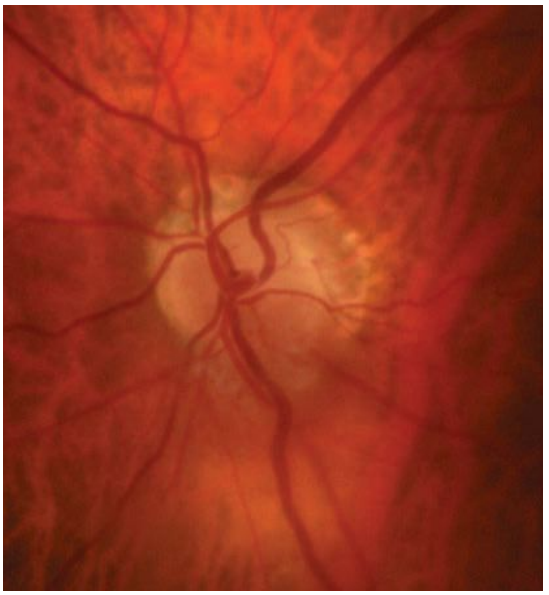
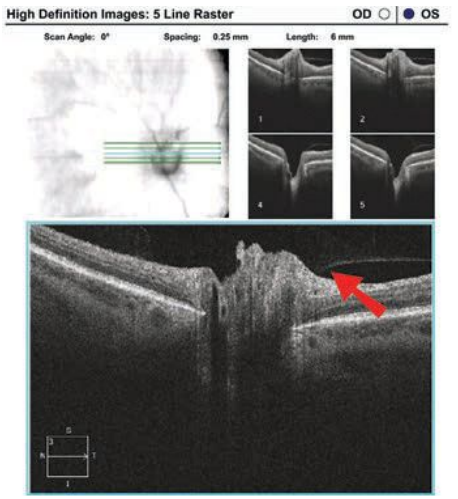


Fig. 7. The patient's optic disc photo demonstrates inferior sectoral elevation with hyperemia. Additionally, there is sectoral pallor of the superior neuroretinal rim OS.



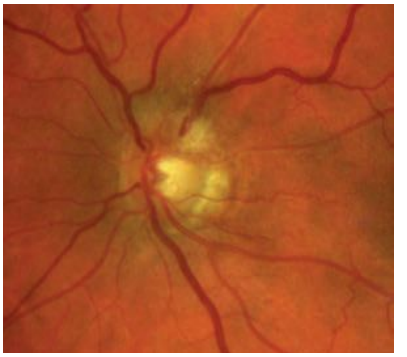


Fig. 8. Imaging reveals superior neuroretinal rim elevation with telangeictatic vessels. The OCT 5-line raster, at left, demonstrates vitreopapillary adhesion.

Case 5: A Pale Masquerader

A 15-year-old African American male presented for re-evaluation of his optic disc OS. He was seen one year prior by another provider and was diagnosed with refractive amblyopia OD and suspected pseudo-temporal pallor OS. He denied any visual complaints or changes. He denied any history of trauma or neurologic symptoms such as headaches. His medical history was remarkable for asthma.

His best-corrected visual acuity was stable at 20/40 OD, 20/20 OS. Pupils were equal, round and reactive to light with no afferent pupillary defect OS. Confrontation and automated visual fields were full without defects OU and color vision was normal. Refraction was remarkable for amblyogenic hyperopia OD. His posterior segment exam and OCT measurements were repeated and compared to findings from one year prior, with significant findings (*Figures 9 and 10*).

Discussion. The diagnosis of stable pseudo-temporal pallor OS was made based on normal afferent function in the left eye and OCT.

While some anomalous discs may exhibit associated afferent findings, the lack of any abnormalities OS was significant in this case, as true pallor is typically associated with afferent pupillary defect, color vision loss, visual field defect or a combination of all three (*Table 3*).

OCT of the optic disc and peripapillary retinal nerve fiber layer (RNFL) is a valuable tool to look for anatomic variations or anomalies that can give rise to the appearance of pseudo-pallor. For example, note in the patient's OCT the asymmetric disc diameter with the left disc (the one in question) being notably smaller than the right. This is important in this case, as small/hypoplastic discs may appear pale temporally, especially if there is a concurrent scleral crescent

Additionally, tilted optic discs can have a similar appearance and, in this case, a subtle tilt of the disc can be appreciated by viewing the horizontal tomogram on the OCT in which the temporal neuroretinal rim is lower and sloped.

However, interpreting OCT of the peripapillary RNFL for thinning can be complicated by anatomical variation, such as shifted RNFL bundles, or even pathology, such as disc swelling. Ganglion cell layer (GCL) analysis, in contrast, may not be as affected by anatomical difference/swelling and can be a valuable adjunctive tool for detecting retinal ganglion cell death, implicating an optic neuropathy.[12,13] Therefore, GCL analysis was beneficial as this ruled out any thinning or loss suggestive of an optic neuropathy.

Other conditions that a clinician may be confronted with that can mimic pallor include pseudophakic pallor, which is caused by change in the lens optics, and large physiologic cupping.[14] In addition to a thorough afferent evaluation and RNFL/GCL OCT, repeat evaluation to ensure stability is helpful in confirming pseudo-pallor, as opposed to pallor caused by an active process.

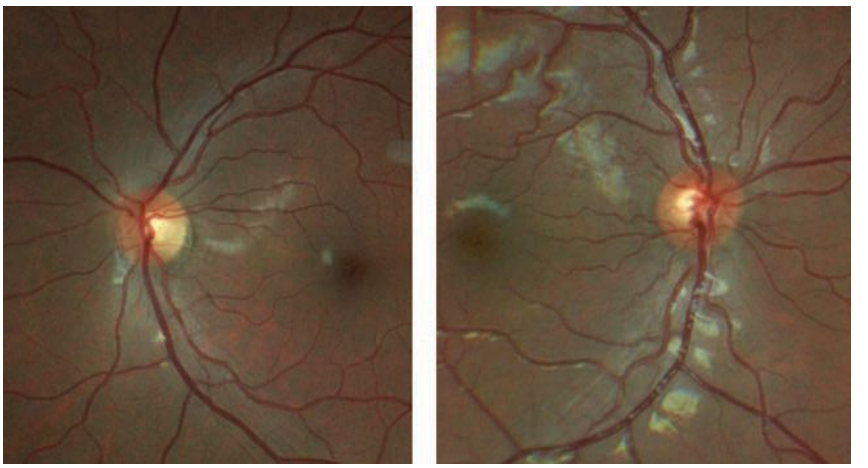


Fig. 9. This patient's fundus photo suggests a pale temporal neuroretinal rim in the left eye.

Ultimately, while these tools can help differentiate true pallor from pseudo-pallor, if the judgement cannot be made with confidence, further work-up to rule out potentially treatable causes of optic neuropathy may be indicated.

Careful clinical examination in conjunction with ancillary testing such as OCT and visual fields are important in differentiating benign processes from potential neuro-ophthalmologic emergencies. Critical assessment of the peripapillary region and optic nerve head for neuroretinal rim thinning, pallor and elevation is important in all patients to identify subtle disc anomalies and make the correct diagnosis. ■

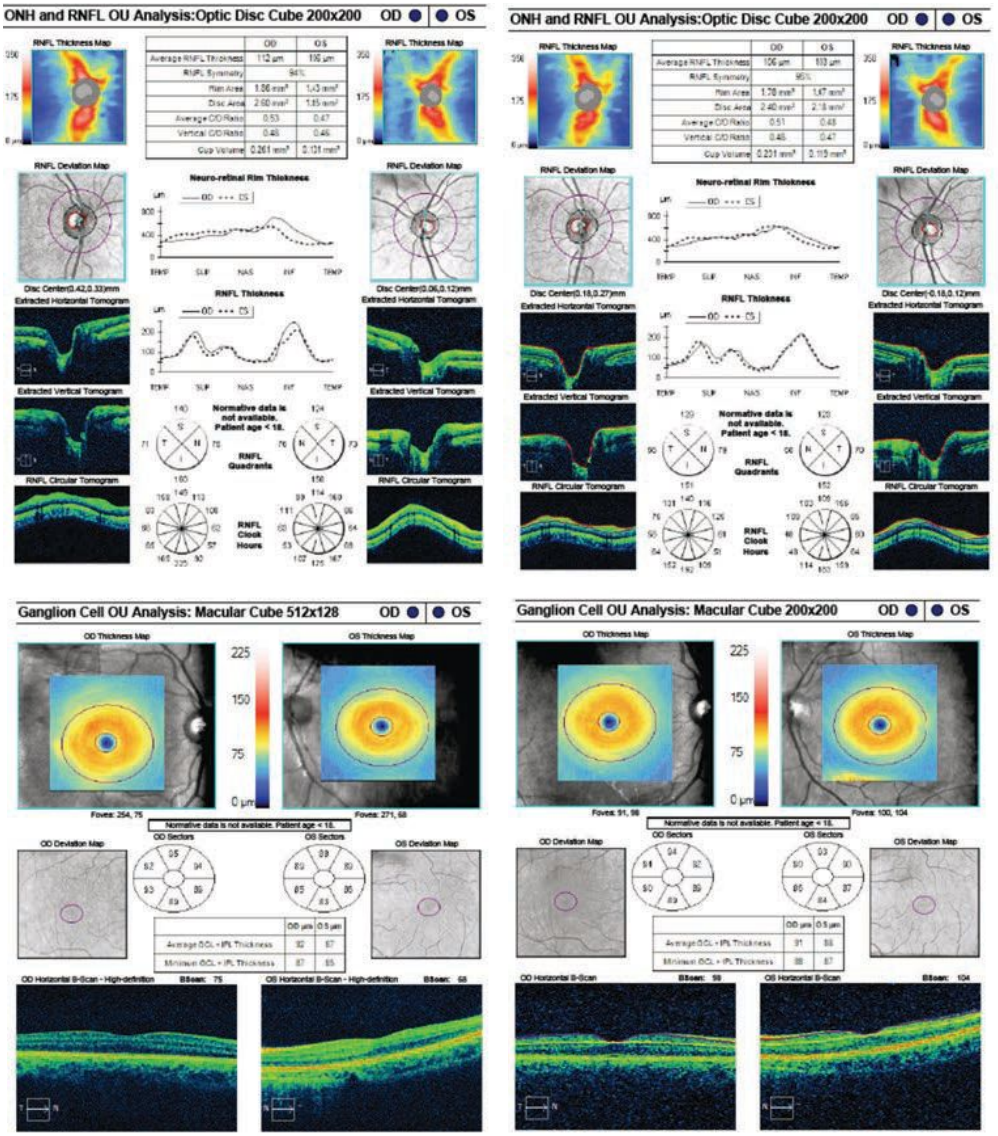


Fig. 10. These are the patient's OCT images upon initial exam, at left, and one year later, at right. The OCT demonstrates an intact RNFL and GCL in both eyes. Note the small disc area OS compared with OD, as well as gradual sloping of the temporal aspect of the cup OS compared with the symmetric margins of the cup OD.

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26th Annual Glaucoma Report

Seven Ways Glaucoma Care is Changing

Better drugs, safer surgeries, smarter diagnostics and new approaches are easing the burden on patients—and their ODs.

By Michael Chaglasian, OD, and Sarah B. Klein, OD

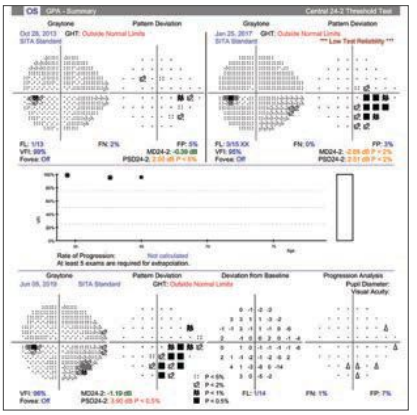
In the ever-changing health care landscape, optometrists have to constantly adapt to new needs and responsibilities. Glaucoma in particular is emblematic of our evolving role, as advances in technology, pharmaceuticals and research provide opportunities to expand our management of this sight-threatening condition. With the number of glaucoma patients growing steadily, even some ophthalmologists agree that optometrists have a vital role to play in glaucoma management and comanagement.[1]

To do so, we need to stay on top of improvements in glaucoma care so that we can make the best, evidence-based recommendations for our patients and be a crucial part of a team whose ultimate goal is to preserve sight for years to come. Lowering IOP is still the only known modifiable risk factor for glaucomatous progression, and the average optometrist has a crucial role in determining the best way to do so, whether it be through pharmaceutical intervention or surgical recommendations. With that, any chosen method of IOP control necessitates regular monitoring, which is more available to ODs than ever before, and allows us to effectively manage and comanage these patients with our ophthalmology colleagues.

Here, we take a look at seven vital advancements that have helped put patient care into the hands of the primary care optometrist and how we can use these technologies and techniques to our patients' advantage while moving the needle on scope of practice expansion.

1 *MIGS elevates the role of surgery.* Minimally invasive glaucoma surgery (MIGS) has exploded over the past few years as one of the fastest-growing treatment categories for mild to moderate glaucoma, due to an improved safety profile and decreased risk of complications compared with traditional glaucoma surgery options.[2] While efficacy may be modest as a whole compared with trabeculectomy, so too are side effects. Therein lies the category's chief strength: the risk/benefit balance is decisively in its favor.

MIGS is also an excellent option for patients who are noncompliant with drops or have not responded well to procedures such as selective laser trabeculoplasty (SLT).[3] Among the top players in this arena currently are the iStent Inject (Glaukos), the Hydrus Microstent (Ivantis), the Xen gel stent (Allergan) and various canal-based procedures such as iTrack (Ellex) and the Omni Surgical System (Sight Sciences). Though ODs should be well-versed in all options, for the sake of brevity we'll review the iStent and Hydrus here.



This patient's glaucoma was progressing despite being on two medications. He then developed an early cataract that was causing glare while driving at night. A MIGS device (iStent Inject) was used in conjunction with his cataract surgery. Post-op IOP was 16mm Hg after stopping one of his two meds.

The iStent and iStent Inject are tiny trabecular stents made of a biocompatible titanium that provides an excellent safety profile with minimal complications.[4] The original iStent device, which was 1mm/0.3mm in size, was implanted manually into the trabecular meshwork (TM) in combination with cataract surgery, with some technical difficulty and learning curve effect.[5] The newer iStent Inject boasts an even smaller size (360/230µm) and is now the smallest medical device implantable in the human body. Two stents are present in each preloaded applicator, and they are placed perpendicularly into the TM two to three clock hours apart with relative technical ease for the surgeon.

The two stents placed in this fashion improve access to aqueous collector channels and improve the chances of reaching an episcleral vein, therefore improving the potential for IOP reduction.[5] Research shows the original iStent reduces IOP significantly compared with cataract surgery alone, with an even greater decrease with two iStents, as well as allow for a reduction in the number of postoperative IOP-lowering medications needed to achieve goal IOP.[6]

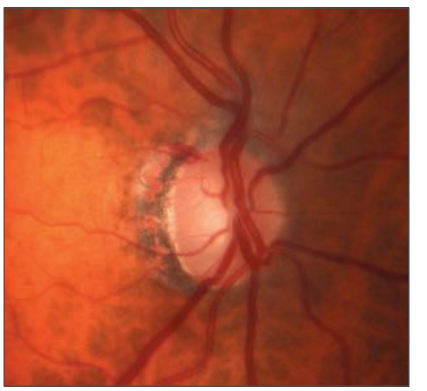
Postoperative care is similar to that of cataract surgery alone, with no additional visits or medications needed, and is therefore straightforward for the comanaging optometrist.[7] However, you may be able to start discontinuing topical glaucoma medication(s) as early as the day-one post-op visit, adding them back as necessary depending on the result.[8]

Proper patient selection is always the key to success with any procedure. Due to its excellent safety profile, iStent Inject can be confidently recommended for patients with ocular hypertension or mild to moderate open-angle glaucoma (OAG) who have concurrent visually significant cataracts and healthy, open angles, in the absence of inflammation, neovascular glaucoma or other innate or acquired angle abnormalities. They would need to be educated on the risks (nearly none), benefits (potential for reducing drop dependence) and cost, which varies based on insurance coverage and copays.

The Hydrus Microstent (Ivantis) is a small, flexible drainage device inserted in the TM parallel to Schlemm's canal; this procedure is also combined with cataract surgery. Once inserted, it causes scaffolding of the TM and increases outflow, with increased likelihood of targeting collector channels due to its 90-degree span in the anterior chamber angle. Compared with phaco alone, Hydrus has been shown to reduce IOP another 2.3mm Hg and med use following surgery by 30% through 24 months, with an average reduction in IOP of 7.6mm Hg at two years.[9] Post-op care is again similar to phaco alone and can be easily performed by the comanaging OD.

2 ***New drugs target different IOP-lowering mechanisms.*** After a 15-year drought in the United States without the approval of any glaucoma therapies, several new once-daily topical IOP-lowering medications have become FDA-approved over the past several years. As prescribing IOP-lowering medications is in the domain of the optometrist in nearly every state, this is exciting news that gives us additional treatment options that do not require comanagement.

The first category involves the advent of the long-awaited rho kinase (ROCK) inhibitor netarsudil 0.02%, an entirely new class of glaucoma drug that works by decreasing episcleral venous pressure, decreasing trabecular meshwork resistance and possibly reducing aqueous production.[10] It is available packaged alone as Rhopressa (netarsudil 0.02%, Aerie Pharmaceuticals) or in combination with latanoprost as Rocklatan (Aerie Pharmaceuticals),



This patient was on two topical meds for her glaucoma, a prostaglandin analog and a fixed-dose combination (brimonidine/timolol), and had SLT within the last year. IOP was 20mm Hg when this disc hemorrhage was noted in the right eye. The patient declined surgical options. Rocklatan (netarsudil/latanoprost) was prescribed (one drop every evening) as a substitute for the PGA. The IOP was reduced to 16mm Hg.

both for once-daily dosing. Netarsudil has been proven effective alone, lowering IOP up to 5mm Hg in its clinical trials, and in fixed combination with latanoprost, it showed a statistically superior IOP reduction over latanoprost and netarsudil alone at every measured time point.[11]

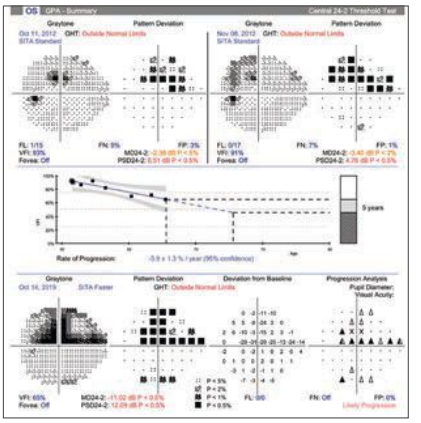
It has a unique side effect profile, with no serious systemic adverse events reported.[10] The main ocular side effect is conjunctival hyperemia, reported in 53% of patients on netarsudil alone and up to 59% of patients using Rocklatan. In clinical practice, however, we have seen that the hyperemia is most noted within the first few days of using the drug, and is worse immediately following administration, and therefore is recommended at bedtime.

Launched in early 2018, Vyzulta (latanoprostene bunod, Bausch + Lomb), a nitric oxide-donating prostaglandin analog, is another relative newcomer to the market. It lowers IOP by a dual mechanism of enhancing uveoscleral outflow while also enhancing TM/Schlemm's canal outflow by inducing trabecular cytoskeletal relaxation.[12] Research shows Vyzulta is more effective than latanoprost alone, with an additional 2mm Hg or more of IOP-lowering ability in 42% of patients, and was proven to have a greater IOP reduction than timolol at nearly all time points measured.[13-15] The side effect profile is minimal and comparable to earlier generation prostaglandin analogs.

All three of these drugs are dosed once daily, which is always ideal for compliance. Insurance coverage is improving across the country as well.

3 ***Sustained-release drug delivery eases compliance burden.*** Many new and exciting sustained-release drug delivery systems are in the pipeline for the treatment of glaucoma, ranging from intracameral implants (Travoprost XR/ENV515, Envisia) to punctal plugs (OTX-TP travoprost insert, Ocular Theraputix) to scleral implants (iDose, Glaukos). However, only one has achieved FDA approval at this time. Durysta (bimatoprost implant 10mcg, Allergan), a biodegradable intracameral implant, gained FDA approval in March 2020.[16]

Durysta is a sustained-release drug delivery system injected through a clear corneal incision into the anterior chamber and rests in the inferior chamber angle. It slowly releases bimatoprost and dissolves over time. Durysta's efficacy is comparable to topical bimatoprost, with an IOP-lowering effect that lasts up to six months.[17] The FDA approval is based on results from the two 20-month Phase III ARTEMIS studies evaluating safety and efficacy in 1,122 subjects vs. twice-daily topical timolol drops in patients with OAG or ocular hypertension. In these studies, Durysta reduced IOP by approximately 30% from baseline over the 12-week primary efficacy period.



This patient demonstrates rapid progression in the left eye. One of the main reasons is her inability to remain compliant with topical medications. She is also fearful of surgery and has declined several options. She is being considered for a drug delivery system such as Allergan's Durysta.

Durysta's side effect profile is similar to topical bimatoprost and other prostaglandin analogs, but causes minimal to no ocular surface irritation due to its presence in the anterior chamber. However, given its physical location, it is contraindicated in patients with Fuchs' dystrophy, prior corneal or endothelial cell transplant, and in the absence of a posterior lens capsule or posterior capsular tear.[18] Considering the well-documented statistics regarding poor patient compliance with topical glaucoma meds, this implant will take the responsibility out of the hands of the patient at least for a period of time, and will likely prove a reliable treatment option going forward.

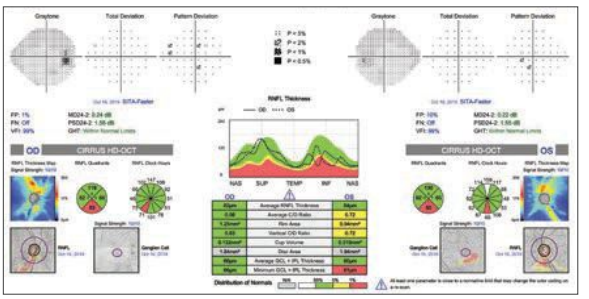
4 ***SLT gets the green LiGHT.*** SLT has long been recognized as an effective treatment for IOP lowering in mild to moderate glaucoma patients, since its original approval in 2001. SLT's predecessor, argon laser trabeculoplasty (ALT), has been extensively studied and demonstrated efficacy comparable to medical therapy as an initial treatment for glaucoma.[19]

ALT and SLT have similar efficacy but, as SLT is less destructive histopathologically, it has the benefit of being able to be repeated.[20] However, in the US and other countries, IOP-lowering medication is still the primary treatment offered in most cases for early glaucoma and ocular hypertension.

This conventional wisdom is now in question, and for good reason. In 2019, the results from the Laser in Glaucoma and Ocular Hypertension (LiGHT) study were released, and may lead to a paradigm shift in glaucoma treatment with more patients being offered SLT as an initial treatment option. The results of this observer-masked, randomized controlled trial performed in the UK

support the theory that SLT is just as, if not more, effective than medication for maintaining goal IOP.[21] In fact, at 36-month follow up, 74.2% of SLT eyes required no drops to maintain goal IOP and were within target at more visits (93%) than in the medication group (91.3%).[22] None of the SLT patients required glaucoma surgery to maintain goal IOP during the follow-up period vs. 11 patients in the eye drop group.[22]

This efficacy, along with a favorable side effect profile and improved cost effectiveness compared with that of topical meds, makes SLT a great choice for first-line therapy.[21] It also eliminates the issue of compliance, which is a constant struggle that's frustrating to ODs everywhere when trying to manage glaucoma with topical medications that are left in the hands of the patient. This pivotal study should influence our decision making going forward when considering initial treatment for glaucoma and ocular hypertension. It forces us to have a conversation with our patients about their initial treatment options, and at the very least to consider referral to a glaucoma specialist capable of performing SLT—including ODs in some states.



This POAG patient's pre-treatment IOP was 24mm Hg OD and OS. OCT shows RNFL loss with a clear inferior bundle defect in the right eye. The visual field is just starting to show some abnormal points. After discussing all treatment options with the patient, he elected to have SLT to avoid the topical side effects of medical therapy and the challenges of being compliant. At nine months post-SLT OU his IOP is 17mm Hg OU.

5 *Home-based monitoring reduces dependence on the exam room.* If the last few months have taught us anything, it's that times are changing. With the onset of COVID-19 and the evolution of how we are living our daily lives in healthcare and beyond, we clinicians may be asked (or forced) to adapt our methods. We are questioning the most safe and sanitary way to check IOP in the office with the debate vacillating between disposable Goldmann applanation tips (Tonosafe, Haag Streit) to Tonopen to iCare. There are also some interesting home care options for glaucoma management now available that may take on a larger role in months and years to come if we find our patients avoiding the office setting due to safety concerns related to the pandemic.

The iCare Home measurement report gives the highest and lowest IOP for each eye, as well as the day and time. It provides clinicians a quick overview of IOP measures outside of office hours.

The iCare Home is a "rebound" tonometer that patients can use at home to measure their own IOP, which can be helpful in monitoring the status of their disease and the risk for progression. It is a handheld device with a disposable probe that gently touches the eye (without the need for anesthetic) and takes six rapid measurements. The machine does not display the IOP readings to the patient but rather saves them internally; they are retrieved later on by the eye care provider.

iCare Home has been shown to give a helpful clinical picture of diurnal IOP fluctuations, especially when taken over a seven-day period, and has demonstrated what we think we know already about diurnal flux, with IOP measurements tending to be highest in the early morning and lower later in the day.[23] Although it has been accused of correlating poorly with Goldmann applanation tonometry readings, it can be a useful tool to gain the bigger picture in patients that may be progressing despite showing normal readings in the exam room.[24] Multiple studies have proven that higher degrees of IOP fluctuation are an independent risk factor for glaucoma progression, and many glaucoma experts agree that the consideration of IOP variability should be a piece of the puzzle when managing glaucoma patients.[24] The iCare Home tonometer provides an opportunity to accomplish this feat without having the patient spend 12 hours in the exam room.

Peristat online perimetry, available since 2002, is a free and portable way to screen for field loss outside of the office. The test is available at www.keepyoursight.org and requires nothing more than a computer with a 17" or larger screen.[25] The test takes less than five minutes, and results have been shown to correlate well with those of the gold standard 24-2 Humphrey field test.[26,27]

The Melbourne Rapid Fields (MRF, M&S Technologies) perimetry test has also been shown to correlate well with traditional Humphrey

results.[28] This program can be used on a tablet or computer screen at home as a web-based exam for glaucoma patients who are concerned about coming into the office during the ongoing COVID-19 crisis. Also, many virtual reality programs offer at-home visual field screening.

In addition to acting as a screening tool for undiagnosed glaucoma patients, the use of these portable and at-home perimetry tests may provide information to us as providers that can help to supplement results from more traditional testing methods in the office.[25] They can also act an opportunity for the patient to "practice," and thereby improve accuracy of field testing in the office at future visits. We learned from the OHTS study years ago that the best accuracy of field test results comes after three or more tests.[29]

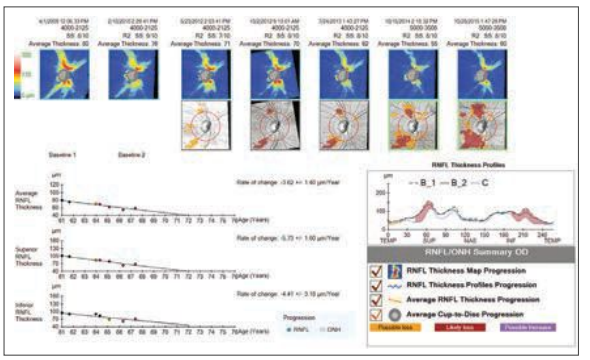
6 *OCT allows earlier detection of progression.* OCT has been available for nearly 20 years now, with the newer generation (spectral domain) models becoming widely available in the past 10 years. While this technology is amazingly helpful in diagnosing early glaucoma in a typical suspect with apparent optic nerve cupping on exam, we've now had the technology for long enough to be able to use it to detect progression of glaucoma as well.

One of the most important measures to look for when trying to detect progression of glaucoma on OCT is repeatable, significant RNFL loss, at both the nerve head and the macular ganglion cell complex (GCC). But what constitutes "significant"? Most experts agree that normal aging accounts for less than one micron per year of average RNFL loss on OCT.[30,31] The machine itself has a test-retest variability of about five microns; in light of that, experts agree that about 10 microns (two standard deviations of the machines inter-test variability) of repeatable change on a reliable test would constitute progression.[30] A reliable test has a signal strength of 7/10 or better, which is easier to achieve on a dilated pupil, and is most accurate when performed in the same state each time (dilated vs. undilated).

OCT is generally recommended once per year on a glaucoma suspect or mild glaucoma patient who has not shown progression, but is valuable to do more often when progression is suspected, to either confirm past change or look for more.[30]

Progression confirmed on OCT alone or with a concurrent new field defect should emphasize the need for additional treatment measures and lower target IOP. In advanced glaucoma, clinicians need to beware of the "floor effect," which occurs when OCT technology ceases to detect further change in RNFL thickness at the nerve head, which occurs when readings approach 40-50 microns.[30,31] In this case, RNFL OCT at the nerve is no longer helpful. Macular thickness, however, is still valuable, as it will continue to show decline in late-stage disease.[30] Visual fields are crucial in advanced disease as well, as vision loss can and will continue to occur with progression, despite RNFL thickness readings becoming stagnant.

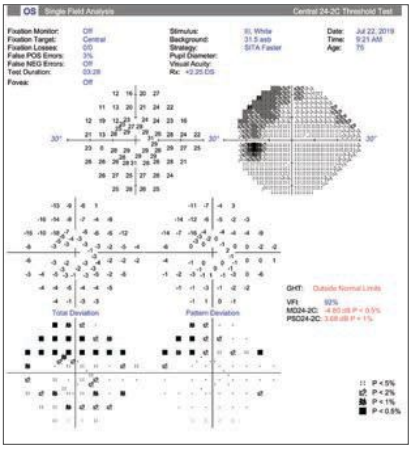
Obtaining an OCT of both the RNFL and GCC at the macula is crucial early on in the diagnosis of glaucoma. In addition to abnormal scans being predictive of future visual field loss and progression, these early tests can be used for comparison to future scans for many years to come in the attempt to catch progression early and to modify therapy as needed.[32]



Progression analysis showing significant RNFL loss to the superior and inferior temporal regions over a six-year time period. Despite aggressive treatment approaches, this patient continued to progress with IOP in the low teens.

7 *Better visual field testing protocols yield new clues.* In recent years, there have been several important advances in visual field testing for glaucoma. One big change has been increased use of the 10-2 visual field strategy for detecting central defects. One study found central field defects were missed in nearly 40% of glaucoma suspects and 35% of presumed OHTN patients on 24-2 SITA Standard (SS) testing but revealed with the 10-2 strategy.[32]

This is an important finding, as central field loss leads to decline in vision-related quality of life, decreased central acuity and is predictive of risk for future field progression, especially in patients with normal tension glaucoma.[33] Current thinking suggests 10-2 testing should be considered at baseline for all glaucoma suspects and those with diagnosed glaucoma, along with 24-2, to improve detection of small, central defects. The 10-2 pattern is also indicated in cases where OCT macular scan (GCA, GCC) shows loss or thinning.[34]



The central 24-2C test pattern incorporates the new SITA-Faster testing strategy along with 10 extra test locations to the traditional 24-2 grid pattern. This has the potential to replace the 10-2 test for central field testing.

Another new advance is development of the SITA Faster test strategy for the Humphrey 24-2. SITA Fast has been around for many years, as long as SITA Standard (SS); they were both developed in 1990s to replace older, slower full threshold test modalities.[35] They were found to save time and be more accurate. SITA Faster, which has a duration 30% shorter than SITA Fast and 53% shorter than SITA Standard, has been available since 2019.[35] So far, it has proved to be nearly identical to SITA Fast in accuracy and comparability to SS; this is a good thing, as prior studies found that there was no significant difference in the ability to detect glaucomatous field progression between the two test strategies, and only a slight increase in precision of defects with SS.[36]

One downside to the faster test has been a higher false positive rate, which can lead to unreliable results.[37] The advantage lies in having a shorter option for patients who have tended to tire easily or fall asleep on past tests, or those who may have trouble sitting for longer periods due to physical limitations. Another advantage is the ability to perform more frequent VF tests, which will help provide better progression analysis. Most of us have many patients that traditionally "hate" perimetry, and a shorter test duration could certainly attempt to change that mindset. It also helps us as providers to keep things moving in a busy clinical setting without sacrificing quality patient care.

Lastly, in the spirit of combining both the need for central testing and the benefit of increased speed, Zeiss now offers a software package that includes the "SITA Faster 24-2C." The 24-2C test pattern combines all 24-2 points plus 10 points from the 10-2 strategy centrally, and theoretically could provide the information from the two separate tests into one. This may be an excellent clinical choice moving forward to save time but provide important information regarding peripheral and central visual function in glaucoma patients.

In any given patient, glaucoma usually progresses slowly, giving us ample time to consider our options. But the research supporting our care protocols moves quickly, and it's incumbent on all of us to keep up with the advances to ensure we do the best job possible in limiting glaucoma's impact in every affected patient we see. ■

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26th Annual Glaucoma Report

A PRACTICAL APPROACH TO ANGLE-CLOSURE

Learn to triage these patients and how to intervene appropriately with in-office treatments or swift referrals as needed. **By Michael Cymbor, OD, and Nicole Stout, OD**

When a patient complains of recent eye injection, deep eye pain and nausea, and has an intraocular pressure (IOP) of 52mm Hg, our first thought is angle-closure crisis, and every optometrist takes an "all hands on deck" approach until the crisis is under control. However, the steps necessary in the more common scenario of the asymptomatic patient presenting with an IOP of 18mm Hg with narrow angles are much less clear.

This article reviews the medical and surgical treatments of angle-closure over the continuum of the disease. Angle-closure is not a single diagnosis but rather a spectrum.[1] The urgency and treatment should reflect the location on this spectrum.

Angle-closure by the Numbers

Angle-closure glaucoma (ACG) affects approximately 23 million people, and the number is expected to increase to 32 million by 2040.[2] ACG is responsible for nearly half of all blindness caused by glaucoma worldwide.[3]

When it comes to angle assessment in the management of glaucoma, several older terms such as occludable, sub-acute, latent or intermittent, may not be helpful given there is a lack of consensus on their meaning.

Angle-closure disease may be primary, secondary or, more likely, a combination of both. It may occur suddenly as an angle-closure crisis, or be chronic, progressing slowly over the course of years. Although a traditional approach views angle-closure as a single disease entity,

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Estimated Time to Complete Activity: 2 hours

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

- Describe clinical factors that define chronic, intermittent and acute angle-closure.
- Explain the differences between primary and secondary angle-closure.
- Discuss how the various classifications affect the long-term outcomes.
- Manage angle-closure patients.
- Determine when to refer patients for surgical management.

Target Audience: This activity is intended for optometrists engaged in the care of patients with angle-closure.

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, the Accreditation Council for Pharmacy Education, and the American Nurses Credentialing Center, to provide continuing education for the healthcare team. Postgraduate Institute for Medicine is accredited by COPE to provide continuing education to optometrists.

Faculty/Editorial Board: Michael Cymbor, OD, Nittany Eye Associates, and Nicole Stout, OD, Northeastern State University Oklahoma College of Optometry.

Credit Statement: This course is COPE approved for 2 hours of CE credit. Course ID is **68529-GL**. Check with your local state licensing board to see if this counts toward your CE requirement for relicensure.

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angle-closure is a heterogenous disease involving different mechanisms that should be identified by the clinician.

Case Example

A 63-year-old white male was referred to a glaucoma specialist by his local optometrist due to increasing IOP and worsening glaucoma. The patient's mother, father and brother all have glaucoma. He reported taking 0.2% brimonidine BID and 0.5% timolol qAM, both OU, for several years.

Best-corrected visual acuity was 20/30 OD and 20/25 OS. He had an afferent pupillary defect OD. His IOP was 23.7mm Hg OD and 21.8mm Hg OS. His corneal hysteresis was 7.3 OD and 7.8 OS. Central corneal thickness was 523µm OD and 530µm OS. Visual field testing revealed a severely reduced field OD>OS with a mean defect of 22.6 OD and 14.8 OS.

Optical coherence tomography (OCT) revealed severely reduced retinal nerve fiber layer and ganglion cell complex OD and moderately reduced OS. OCT angiography showed reduced vessel density OD>OS.

The cup-to-disc ratio was graded at 0.8/0.8 OD and 0.65/0.65 OS. Gonioscopy showed minimal trabecular meshwork (TM) visible with grade 2 pigment OU in all quadrants (*Figure 1*). Anterior segment OCT (AS-OCT) confirmed narrow angles (*Figure 2*).

The patient was diagnosed with severe angle-closure glaucoma OD and moderate ACG OS, staged based on the visual field defect. We performed a bilateral YAG laser peripheral iridotomy (LPI). This had little effect on IOPs or angle opening. We proceeded with cataract surgery with the hope of also performing goniosynecialysis and Kahook Dual Blade goniotomy (New World Medical).

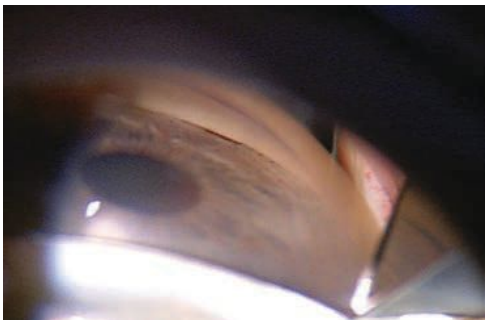

Fig 1. This patient's initial gonioscopy shows a narrow angle with the TM barely visible.

Primary vs. Secondary

Primary angle-closure (PAC) covers a broad spectrum of angle disease. The common feature to all primary angle-closure is the presence of narrow drainage angles characterized by the apposition of the TM and the peripheral iris. The currently accepted classification system in primary angle-closure disease is primary angle-closure suspect (PACS), PAC and primary angle-closure glaucoma (PACG).[4] PACS includes patients who have greater than 180 degrees of iridotrabecular contact with a normal IOP and no optic nerve damage. PAC has greater than 180 degrees of iridotrabecular contact with peripheral anterior synechiae (PAS) or elevated IOP but no optic neuropathy. PACG has everything contained with PAC along with glaucomatous optic neuropathy or the presence of glaucomatous visual field defects.[5]

Secondary ACG occurs as a result of an underlying pathological process. It can be classified as resulting from an anterior "pulling" mechanism by which the peripheral iris is pulled into the angle, occluding the TM, such as:[1-7]

- Neovascular membrane forming in the anterior chamber angle secondary to retinal ischemia, which can occur in conditions such as proliferative diabetic retinopathy, central retinal vein occlusion, central retinal artery occlusion and ocular ischemic syndrome;
- PAS secondary to inflammation that can occur following anterior segment surgery or in chronic uveitis;
- Endothelial membrane obstructing the angle in iridocorneal endothelial (ICE) syndrome or posterior polymorphous corneal dystrophy; or
- Epithelial membrane from epithelial downgrowth following ocular trauma.

Conversely, it may also occur through a posterior "pushing" mechanism where the iris or ciliary body is pushed forward to occlude the angle, such as:[1-7]

- Absolute pupillary block occurring when 360 degrees of posterior synechiae cause iris bombe (a form of secondary pupillary block). This occurs as a result of inflammatory conditions, such as uveitis, that cause the iris to fibrose to the anterior surface of the lens, impeding the normal flow of aqueous;
- Lens-induced angle-closure through subluxation, anterior lens displacement, malpositioning of an intraocular lens or phacomorphic glaucoma (all forms of secondary pupillary block);
- Aphakic pupillary block, which occurs as a result of anterior vitreous displacement and adhesion between the vitreous humor and the iris (a form of secondary pupillary block);
- Ciliary body cysts or tumors, which can cause anterior displacement of the peripheral iris;
- Posterior segment space-occupying lesions, such as tumors, silicone oil or a gas bubble, that cause anterior displacement of the lens-iris diaphragm;
- Choroidal effusion, which most commonly occurs as a complication following glaucoma surgery, but may also be secondary to other intraocular surgeries, inflammatory or infectious diseases, trauma, neoplasms, drug reactions (topiramate and

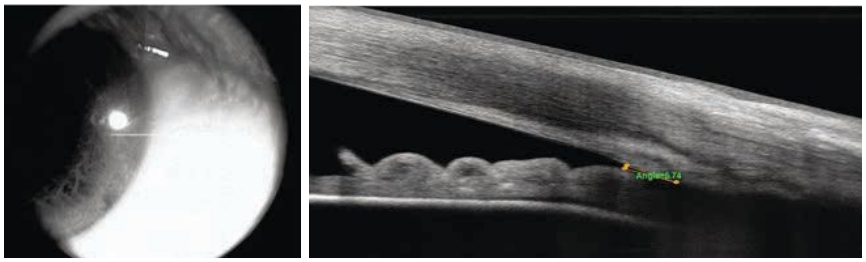

Fig 2. The patient's initial OCT shows a narrow angle.

sulfonamide-induced angle-closure), venous congestion or idiopathic uveal effusion; or

• Ciliary block (also known as aqueous misdirection), which causes shallowing of the anterior chamber as a result of aqueous humor being misdirected into the vitreous body displacing the lens-iris diaphragm forward. This condition can occur following ocular surgery.

Secondary angle-closure can involve an aspect of secondary pupillary block or can occur without pupillary block.[6-11]

However, most cases of angle-closure are due to pupillary block, which occurs when movement of the aqueous from the posterior to anterior chamber is halted, creating a pressure gradient that leads to forward bowing of the peripheral iris, resulting in sudden obstruction of the TM. Of all acute angle-closure patients in the United States, 90% present with pupillary block.[12]

Diagnosing Angle Issues

Four factors help clinicians diagnose potential angle issues: symptoms, signs, risk factors and angle assessment. A careful history and clinical exam are necessary to make the proper diagnosis.

Symptoms of a primary or secondary angle-closure crisis include eye redness, reduced vision, halos, ocular or periocular pain, nausea and vomiting.[3] While symptoms are common in an angle-closure crisis, most cases of chronic angle-closure are asymptomatic.[13] Ocular signs include elevated IOP, conjunctival injection with ciliary flush, corneal edema and a mid-dilated pupil.[3]

Demographic risk factors include advancing age, female gender and Asian ancestry.[14,15] Asian populations typically have thicker irises with a more anterior lens position.[16] Ocular risk factors include hyperopia, shallow anterior chamber, small anterior chamber volume and area, thicker peripheral iris and a higher insertion, increased lens vault and an anterior ciliary body position.[17,18]

Gonioscopy is still the standard when evaluating angle structures, and clinicians must be intimately familiar with angle assessment. Unfortunately, angle assessment may be among the most underused aspects of glaucoma management. One study found that 40% of diagnosed open-angle glaucoma patients actually had angle-closure.[19] Other studies show that gonioscopy/angle assessment is performed less than 50% of the time in glaucoma patients and suspects.[20,21]

When the angle is open, the most posterior angle structure visible is the ciliary body (CB), found between the iris root and the scleral spur. It is usually brown but may appear as light gray. The second most posterior structure is the scleral spur and can vary in color from white to gray. It is found in the posterior margin of the scleral sulcus, between the CB and the TM. The scleral spur is comprised of collagen tissue and serves as the anchor for the ciliary muscle.

The TM is next, found between the scleral spur and Schwalbe's line. It can be subdivided into anterior and posterior TM. It is typically light gray in younger patients and becomes more pigmented in older individuals. The anterior third of the TM is nonfunctional, while the posterior two-thirds filters aqueous into Schlemm's canal. Schwalbe's line is the most anterior angle structure and represents the end of a clear cornea. While there are three main angle classification systems—Scheie, Shaffer and Spaeth—the universally accepted classification system is to simply describe the most posterior structure seen by quadrants.[22-24]

Gonioscopy is critical for identifying some causes of secondary angle-closure. Indentation gonioscopy can help clinicians differentiate between iridocorneal apposition and peripheral anterior synechia. This technique is performed using a small-diameter gonioscopy lens to apply pressure to the central cornea, displacing the aqueous humor towards the angle, which separates the iris from the cornea and allows for better visualization of the angle structures. Angle structure visibility with indentation suggests iridocorneal apposition, while synechial angle-closure should not improve angle structure visibility upon indentation.

While gonioscopy remains the standard, technologies such as AS-OCT, ultrasound biomicroscopy (UBM) and Scheimpflug imaging are playing a more prominent role as more doctors gain access. Furthermore, angle-closure diagnosis rates increase when objective analysis is included.[25] AS-OCT acquires a high-resolution cross-sectional image of the anterior chamber. It often shows the angle narrower than gonioscopy, particularly in the superior and inferior quadrants.[25] This may be due to OCT's ability to measure angles in scotopic conditions. One disadvantage of AS-OCT is that current

devices only sample a small section of the angle at one time.

UBM is also an excellent tool for imaging the anterior segment and can be helpful in identifying the underlying pathology; however, it is not readily available in most private practices.[6,7,26] UBM has the advantage of being able to image behind the iris, including the lens and the CB, but is costly because it is typically a stand-alone instrument.

Scheimpflug imaging may also play a role in angle imaging. Scheimpflug imaging can sample a much larger portion of the angle, but the resolution is less than either AS-OCT or UBM.[27]

While each technology has advantages, objective angle analysis complements gonioscopy.

Acute Treatment Approaches

Treatment of acute angle-closure crisis is typically prompt medical stabilization followed by laser and/or surgical stabilization.

Medical stabilization. This may include treatment with topical alpha agonists, beta blockers, carbonic anhydrase inhibitors and rho-kinase (ROCK) inhibitors. Medical treatment may also include topical steroids to relieve inflammation. Oral treatment may include carbonic anhydrase inhibitors. This approach should be avoided in topiramate- or sulfonamide-induced angle-closure; instead, the causative medication should be discontinued promptly.[7,10]

Oral or intravenous hyperosmotic medications may be used when rapid IOP lowering is not achieved with the above-mentioned treatments. Compression gonioscopy performed with a small-diameter lens may be necessary to break recent iridotrabecular adhesion. In absolute pupillary block, clinicians should use a strong cycloplegic agent and 10% phenylephrine ophthalmic solution to try and break the posterior synechia, in addition to pharmacological interventions that attempt to lower IOP and control inflammation.

In practical terms, optometric medical stabilization means achieving a significant in-office IOP decrease until an LPI can be performed the same day. Medical stabilization should be tailored to how quickly the LPI can be performed. Once an angle-closure crisis is identified, the optometrist should immediately investigate LPI options and have a clear idea of when it can be performed. If the LPI can be performed immediately on-site or at a referral destination close by, medical stabilization might mean putting in a round of pressure-lowering drops and/or a dose of acetazolamide prior to the LPI. If it cannot be performed until a few hours later, clinicians should put more emphasis on medical stabilization as to not subject the patient to a prolonged elevated IOP.

Laser treatment. Once medical stabilization is achieved and the iris can be visualized, the next step would historically be LPI. If the optometrist practices in a state that permits optometric LPI, the optometrist would then perform an emergent LPI, which is generally effective at relieving pupillary block.

If LPI does not open the angle and decrease IOP, plateau iris syndrome (PIS) should be suspected. PIS is when a large or anteriorly positioned CB pushes the peripheral iris forward, potentially closing the angle. This may be present in up to one-third of angle-closure cases.[28] Plateau iris can occur with or without pupillary block. Compression gonioscopy is critical in the diagnosis and will show a marked peripheral iris roll. This occurs because the iris follows the anatomy of the lens from central to peripheral and rises after the level of the equatorial lens up to the anteriorly placed or enlarged CB. UBM may be helpful to visualize the anteriorly positioned ciliary processes.

Argon laser peripheral iridoplasty may help open the angle.[29] This technique applies laser to the peripheral iris, reducing its thickness and pulling it away from the TM. Laser iridoplasty may also reverse recent PAS.[30] Prompt lens extraction surgery may also be considered. Single-pass four-throw pupilloplasty, which reconstructs the pupil, can be an option in persistent cases.[31]

Most secondary angle-closure glaucomas with a pupillary block component will require an LPI. Approximately 25% of patients with pupillary block will continue to show iridotrabecular contact even after LPI.[32] Factors that may adversely affect LPI success include eyes with greater than 180 degrees of PAS, higher baseline IOP and narrower angles as determined by UBM and AS-OCT.[33] Even if LPI is initially successful, it should not be viewed as a long-term cure. The natural lens will continue to grow, narrowing the anterior chamber over time and increasing lens vault.[34]

Once LPI stabilization of both the angle and IOP occurs, the clinician faces several options. The patient may only require observation with the initiation of topical medication or adjustment of current therapy upon IOP increase.

Provided that the TM is visible to at least 180 degrees, selective laser trabeculoplasty (SLT) may help stabilize IOP. SLT may be limited in angles with 180 degrees or more of PAS and if the TM experiences significant IOP-induced trauma during acute PAC. There appears to be no difference in SLT outcomes between patients with PAC and PACG.[35] Transscleral (delivery through the pars plana) and endoscopic cyclophotocoagulation are also options, as they reduce CB aqueous formation and shrink the CB.[36]

Surgical treatment. Even though LPI is the most historically common choice of treatment after medical stabilization, moving directly to lens extraction may be the better option.

The EAGLE study compared LPI with clear lens extraction by looking at patients over 50 years old with mild to moderate PACG with a presenting IOP above 30mm Hg.[37] The study found a reduction in the need for further medications or glaucoma surgeries in the clear lens extraction group along with a better quality of life and better cost-effectiveness. A recent study suggests that lens extraction should be performed early as a way to prevent PACG.[4] In the case of phacomorphic glaucoma, cataract surgery should be performed as the definitive treatment.[10]

Phacoemulsification with intraocular lens implantation may relieve iridotrabecular contact, lowering IOP. In some cases, goniosynecialysis may be needed to break contact. This involves mechanically disrupting PAS by gently pushing on the peripheral iris to break the attachment between the iris and the TM.

If successful, a variety of TM-targeting minimally invasive glaucoma surgeries (MIGS) may then be employed, such as Kahook Dual Blade goniotomy, Trabectome (Microsurgical Technolo) and iStent (Glaukos). Trabeculectomies and tubes are also an option for more advanced cases. As with our patient, Kahook Dual Blade and goniosynecialysis combined with phacoemulsification can provide reductions in both IOP and the need for IOP-lowering medications.[38]

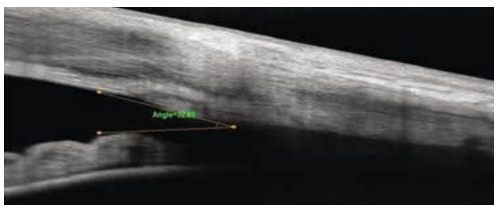

Fig. 4. This is the patient's corresponding AS-OCT after successful treatment.

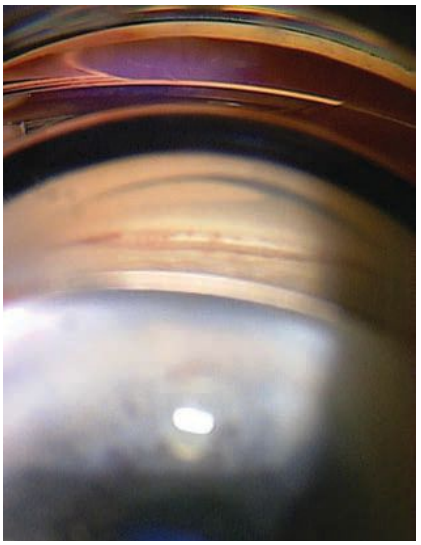

Fig. 3. This image depicts the patient's angle after goniosynechialysis, cataract surgery and Kahook Dual Blade goniotomy.

If phacoemulsification, and possibly goniosynecialysis, does not relieve iridotrabecular contact, the optometrist may need to refer the patient for a more aggressive MIGS such as the Xen gel stent (Allergan). A trabeculectomy or a tube procedure may also be needed but both have more postoperative complications for angle-closure than primary open-angle glaucoma patients.[39]

Secondary angle-closure considerations. Causes of secondary ACG due to posterior "pushing" mechanisms that do not involve pupillary block are often a result of the peripheral iris being displaced forward by the lens or CB. In these cases, the use of a cycloplegic agent to induce posterior rotation of the CB is often indicated, in addition to topical IOP-lowering drops and topical steroids.[7]

Many of these conditions require a referral to a glaucoma, retina or ocular oncology specialist to manage the underlying cause. Although pilocarpine can be used in primary phakic pupillary block glaucoma to pull the peripheral iris away from the TM, it can cause contraction of the ciliary muscle, resulting in anterior lens movement and paradoxical worsening of the angle-closure in cases of secondary ACG.[40]

Secondary angle-closure glaucoma resulting from anterior "pulling" mechanisms also often requires a referral for surgical intervention, such as in the case of secondary ACG caused by significant PAS where goniosynecialysis could be performed. This procedure is more likely to be successful if the synechia are relatively new.[7,10]

In neovascular glaucoma, after attempting to get the IOP and inflammation under control with pharmacological therapies, the patient should be referred to a retina specialist for treatment of the underlying retinal ischemia with panretinal photocoagulation and/or anti-vascular endothelial growth factor agents. These patients will often also require a referral to a glaucoma specialist for more invasive glaucoma surgeries such as a tube procedure.[7,8,10]

After the acutely elevated IOP is lowered and the underlying cause of the primary or secondary ACG has been treated, clinicians should monitor these patients regularly with IOP checks, optic nerve head assessments, OCTs, angle assessments and visual fields to monitor for further glaucomatous progression and to detect if additional intervention becomes necessary.

Caring for the Chronic Patient

While an acute angle-closure crisis is a clinical emergency requiring immediate care, chronic angle-closure may be more insidious and progress slowly. It remains a clinical challenge to determine the ideal time to intervene. For instance, questions persist regarding whether LPI should be recommended for all PACS patients to prevent PAC and/or PACG.

The recent ZAP trial showed a statistically significant but clinically small decrease in the risk of PAC conversion and recommended against the widespread use of prophylactic LPIs in their study population.[41] Further analysis of the ZAP trial found that 44 PACS patients needed treatment to prevent one new PAC case over six years.[42]

LPI is mostly benign, usually opens the angle to some extent and potentially prevents an angle-closure crisis. Nevertheless, side effects may occur, including dysphotopsia and accelerated cataract formation.[43,44]

In our clinic, we typically follow most asymptomatic PACS patients every six to 12 months. We monitor for changes in the angle, optic nerve and visual field. While we approach each patient individually, we generally perform LPI if the patient mentions symptoms suggestive of closure, has a family history of angle-closure or if they show progression of angle narrowing.

Eyes that develop PAC or PACG should be treated.[33] The treatment of chronic angle-closure is similar to the treatment for acute angle-closure: stabilize the IOP medically or with SLT, evaluate the angle, perform LPI when appropriate and consider cataract or clear lens extraction (PACG cases) with or without MIGS. The clinician should escalate therapy when progression is identified. Chronic angle-closure treatment may follow a course of years, rather than days or months. Patients with PAC or PACG who are followed closely and treated more aggressively than primary open-angle glaucoma patients generally have favorable long-term outcomes.[45]

Our Patient

Fortunately, phacoemulsification combined with goniosynecialysis opened our patient's angle enough to proceed with Kahook Dual Blade goniotomy (*Figure 3*). These three procedures stabilized aqueous outflow and IOP. One year later, his IOP is 11.4mm Hg OD and 10.4mm Hg OS on no medications. His fields and optic nerve OCTs are stable along with his AS-OCTs (*Figure 4*).

As optometrists continue to play a more significant role in all aspects of glaucoma management, it is critical that we better appreciate the importance of angle assessment, use all of our angle diagnostic options, refer patients when appropriate and monitor and manage these patients over the course of their lives. ■

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OSC QUIZ

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1. Angle-closure disease may present as:
a. Primary.
b. Secondary.
c. A combination of both primary and secondary.
d. All of the above.

2. Primary angle-closure glaucoma includes everything, *except*?
a. Greater than 180 degrees of iridotrabecular contact.
b. Elevated intraocular pressure.
c. Reduced levels of nitric oxide.
d. Optic neuropathy.

3. Which of the following is a risk factor in angle-closure disease?
a. Myopia.
b. Hyperopia.
c. Lacquer cracks.
d. Staphyloma.

4. All of the following are advantages of anterior segment OCT, *except*:
a. Resolution.
b. Objective assessment.
c. Sampling a large angle area.
d. Scotopic imaging.

5. Most cases of acute angle-closure are due to:
a. Pupillary block.
b. Hyperopia.
c. Underdevelopment of trabecular meshwork.
d. Overdevelopment of the ciliary body.

6. Which of the following medications is a possible medical treatment for angle-closure?
a. Beta blockers.
b. Rock inhibitors.
c. Oral hyperosmotic.
d. All of the above.

7. What is compression gonioscopy used for?
a. Diagnosing plateau iris.
b. Identifying posterior synechiae.
c. Breaking recent iridotrabecular contact.
d. a and c.

8. Under which of the following situations is LPI most successful?
a. 270 degrees of peripheral anterior synechiae.
b. Narrower angles.
c. Lower baseline IOP.
d. Ciliary body swelling.

9. Which of the following is *not* a symptom of acute angle-closure crisis?
a. Reduced vision.
b. Diplopia.
c. Ocular/periocular pain.
d. Nausea/vomiting.

10. Plateau iris may be present in up to _____ of angle-closure cases.
a. One-half.
b. One-third.
c. One-fourth.
d. One-fifth.

11. Which of the following procedures can break iridotrabecular contact?
a. Kahook Dual Blade.
b. Goniosynecialysis.
c. Cataract surgery.
d. b and c.

12. Which trial recommends the use of LPI in most cases of primary angle-closure suspects?
a. Eagle.
b. ZAP.
c. OHTS.
d. None of the above.

13. Which of the following examination techniques can be used to differentiate between iridocorneal apposition and peripheral anterior synechia?
a. AS-OCT.
b. Dilated fundus examination.
c. Gonioscopy.
d. UBM.

14. What is the most posterior angle structure visible on gonioscopy when the angle is wide open?
a. Posterior trabecular meshwork.
b. Scleral spur.
c. Ciliary body.
d. Schwalbe's line.

15. Which of the following causes of secondary angle-closure glaucoma uses an anterior pulling mechanism?
a. Aphakic pupillary block.
b. ICE syndrome.
c. Choroidal effusion.
d. Ciliary block.

16. In which of the following is the use of carbonic anhydrase inhibitors contraindicated?
a. Primary pupillary block glaucoma.
b. Neovascular glaucoma.
c. Ciliary block glaucoma.
d. Topiramate induced angle-closure glaucoma.

17. Which of the following involves a secondary pupillary block component?
a. Ciliary body swelling following panretinal photocoagulation.
b. Phacomorphic glaucoma.
c. Glaucoma secondary posterior polymorphous corneal dystrophy.
d. Choroidal effusion.

18. Which of the following would be least used in the treatment of secondary pupillary block from peripheral anterior synechiae?
a. Timolol 0.5% ophthalmic solution.
b. Cyclopentolate 1% ophthalmic solution.
c. Pilocarpine 2% ophthalmic solution.
d. Phenylephrine 10% ophthalmic solution.

19. All of the following tests would be useful in the diagnosis of angle-closure glaucoma, *except*:
a. UBM.
b. Anterior segment OCT.
c. Pachymetry.
d. Gonioscopy.

20. Which of the following causes of secondary angle-closure glaucoma involve a posterior pushing mechanism?
a. Ciliary body tumors.
b. Peripheral anterior synechia.
c. Neovascular glaucoma.
d. Glaucoma secondary to epithelial downgrowth following a penetrating trauma.

Examination Answer Sheet

A Practical Approach to Angle-closure

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Answers to CE exam:

1. Ⓐ Ⓑ Ⓒ Ⓓ
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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. Describe clinical factors that define chronic, intermittent and acute angle-closure. ① ② ③ ④ ⑤
22. Explain the differences between primary and secondary angle-closure. ① ② ③ ④ ⑤
23. Discuss how the various classifications affect the long-term outcomes. ① ② ③ ④ ⑤
24. Manage angle-closure patients. ① ② ③ ④ ⑤
25. Determine when to refer patients for surgical management. ① ② ③ ④ ⑤

26. 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.

27. 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): ☐

28. 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 pharmaceutical therapy ⓒ Choice of treatment/management approach ⓓ Change in current practice for referral ⓔ Change in non-pharmaceutical therapy ⓕ Change in differential diagnosis ⓖ Change in diagnostic testing ⓗ Other, please specify: _____

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

ⓐ Very confident ⓑ Somewhat confident ⓒ Unsure ⓓ Not confident

30. Which of the following do you anticipate will be the primary barrier to implementing these changes?
ⓐ Formulary restrictions
ⓑ Time constraints
ⓒ System constraints
ⓓ Insurance/financial issues
ⓔ Lack of interprofessional team support
ⓕ Treatment related adverse events
ⓖ Patient adherence/compliance
ⓗ Other, please specify:

31. Additional comments on this course:

_____

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Rate the quality of the material provided:
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32. The content was evidence-based. ① ② ③ ④ ⑤
33. The content was balanced and free of bias. ① ② ③ ④ ⑤
34. The presentation was clear and effective. ① ② ③ ④ ⑤

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

Signature _____ Date _____

Lesson 119917 RO-OSC-0720

Riboflavin vs. Rose Bengal

When considering photodynamic therapy, make sure to evaluate the efficacy of each photosensitizer. **Edited by Joseph P. Shovlin, OD**

Q I've recently seen some discussion on treating fungal keratitis with rose bengal and photodynamic therapy (PDT). How does this combination's efficacy compare with riboflavin and corneal crosslinking (CXL)? Are there any indications for one procedure over the other?

A "Most optometrists associate PDT with treating wet macular degeneration," says Brian Chou, OD, of San Diego. He notes that this light-activated treatment's capabilities don't stop there. Upon activation, a photosensitizer releases reactive oxygen to targeted cells and tissue to help manage a wide range of conditions, from acne to cancer.

The most recognized form of PDT in eye care is currently CXL for keratoconus and post-LASIK ectasia.[1] Riboflavin and ultraviolet (UV) light increase and strengthen molecular bonding between corneal collagen fibrils to prevent progressive ectasia. Other eye-related applications of PDT include corneal neovascularization, microbial keratitis and certain choroidal diseases.[2-4]

Fungal Keratitis Management

Of the total microbial keratitis cases in the United States, 6% to 20% are fungal.[5] The preferred topical treatments are natamycin 5% for *Fusarium* (filamentous) and amphotericin B 0.15% for *Candida* (yeast) and *Aspergillus* (filamentous).[6] Due to poor penetrance, deep stromal infections may also require repeated debridement, systemic antimycotics or both, advises Dr. Chou. Even so, he adds that treatment is limited, and resolution often takes months.

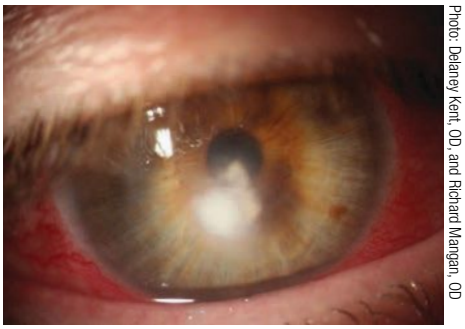

Photo: Delaney Kent, OD, and Richard Mangan, OD

PDT with rose bengal can be a suitable option for fungal keratitis treatment.

PDT is a controversial treatment for fungal keratitis, according to Dr. Chou. Several studies have proposed using rose bengal or riboflavin as the PDT photosensitizer. A team of researchers found that riboflavin and UVA irradiation reduced *Fusarium* colony-forming units *in vitro* and improved the clinical appearance of *Fusarium* keratitis in the *in vivo* mouse model.[7]

However, Dr. Chou says there is greater evidence that the combination of riboflavin and UVA does not effectively inhibit fungal proliferation. A 2017 randomized clinical trial that looked into CXL treatment with riboflavin for deep stromal fungal keratitis was aborted because the clinical group experienced more perforations than the controls.[8] Furthermore, recent results of an *in vivo* clinical trial of 403 patients with filamentous fungal keratitis published in *Ophthalmology* showed no benefit of adjuvant CXL.[9]

PDT with rose bengal may offer greater promise, Dr. Chou suggests. *In vitro*, rose bengal and green light (518nm) effectively inhibited fungal isolates (*Fusarium*, *Aspergillus* and *Candida*), whereas riboflavin with UVA (375nm) permitted unrestricted growth.[10] A case report of a 56-year-old rigid gas permeable contact lens wearer with culture-positive *Fusarium* keratitis described a worsening presentation with hourly natamycin 5%, intrastromal amphotericin B injection and oral fluconazole.[11] On day 44, she had rose bengal PDT, and within four days, she had experienced significant improvement.[11]

Keep rose bengal PDT on your radar, and be on the lookout for more to come on its viability as a treatment option. ■

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Navigating Retinal Necrosis

This rare, acute disorder can be tied to a number of systemic diseases.

By Rami Aboumourad, OD, and Richard Mangan, OD

A 19-year-old Caucasian male presented with symptoms of progressive, deteriorating vision and a red painful left eye for six days. He had been seen at a local Urgent Care facility, where he was diagnosed with ocular allergies, and then by a local eye care provider who referred the patient to our tertiary care center.

The patient had a medical history of seizures and was developmentally delayed secondary to neonatal herpes simplex virus type 2 (HSV-2) encephalitis. Review of systems revealed symptoms consistent with an upper respiratory infection for seven days. All other medical, ocular and family histories were unremarkable, and the patient was otherwise systemically healthy.

Upon exam, unaided visual acuity was 20/400 in both eyes with pinhole improvement to 20/40 OD and 20/80 OS. There was relative afferent pupillary defect in the left eye; extraocular motility, confrontation visual fields and intraocular pressures were all otherwise unremarkable.

The slit lamp exam showed bilateral palpebral conjunctival follicles but no palpable preauricular lymph nodes. The ocular exam of the right eye was otherwise unremarkable. The left eye had significant diffuse episcleral injection with inferior corneal stellate keratic precipitates (*Figure 1*). There were 2+ anterior chamber cell and flare (SUN classification) and moderate vitreous cell mildly obscuring the view of the fundus.

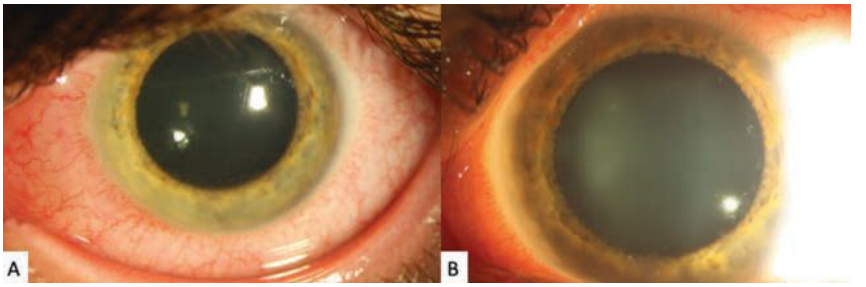

Fig. 1. These gross external slit lamp photos of the patient's left eye shows significant diffuse episcleral injection. A faint posterior corneal opacities can be see, although the detail is poor.

The exam did uncover grade 3 optic disc edema (using the modified Frisén scale). The retinal vasculature was tortuous, and focal retinal whitening and hemorrhaging was seen in the superonasal periphery. (*Figures 2 and 3*).

Differential Diagnosis

A number of disease entities could underly our patient's presentation. Conditions that can present with panuveitis could include sarcoidosis, tuberculosis, syphilis, Behçet disease, Vogt-Koyanagi-Harada syndrome and toxoplasmosis. One must also consider the viral entities that cause a necrotizing retinitis, such as varicella zoster virus (VZV), herpes simplex virus type 1 or 2 (HSV-1 or HSV-2), cytomegalovirus (CMV) and Epstein-Barr virus (EBV).

Analysis of blood serum can evaluate for syphilis, tuberculosis and toxoplasmosis. Aqueous and vitreous humor can be analyzed by polymerase chain reaction (PCR) to detect for the presence of VZV, HSV-1, HSV-2, CMV and EBV. When other diagnostic modalities have failed, send vitreous humor to pathology for histological evaluation. Computed tomography (CT) of the chest is essential to evaluate for sarcoidosis and tuberculosis.

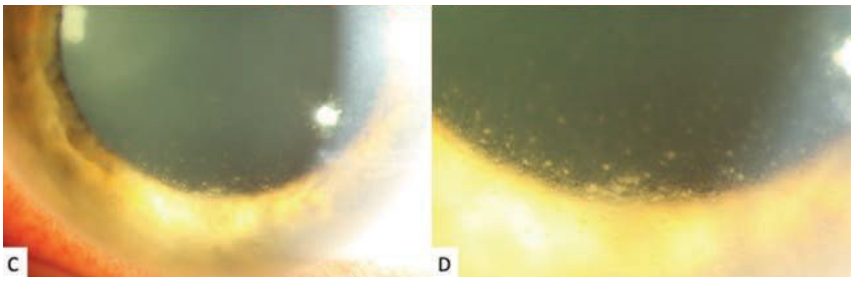

Fig. 1c and 1d. High-magnification slit lamp photographs of the left eye showing inferior stellate keratic precipitates on the lower half of the cornea.

Diagnosis

We saw reason to highly suspect acute retinal necrosis due to the clinical presentation and known history of neonatal HSV-2 encephalitis. A diagnostic anterior chamber paracentesis was performed on the left eye, and the aqueous humor was sent for PCR analysis for VZV, HSV-1, HSV-2 and CMV; only HSV-2 was detected. Serological studies revealed normal blood composition and were negative for syphilis and toxoplasma titers. A negative CT ruled out sarcoidosis and tuberculosis.

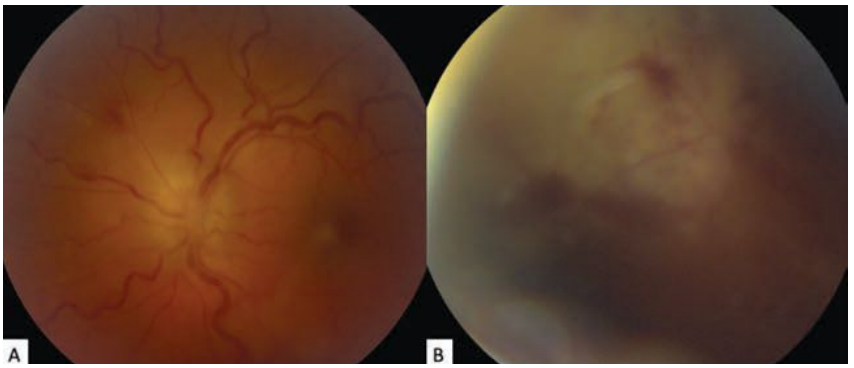

Fig. 2. (a) The patient's fundus photo shows their left eye's grade 3 optic disc edema, using the modified Frisén scale. Vascular tortuosity, faint retinal whitening and hemorrhaging is also visible superonasally. (b) This photo of the superonasal peripheral fundus shows focal retinal whitening with hemorrhage obscured by vitreous haze.

Discussion

While rare, acute retinal necrosis (ARN) is a potentially visually devastating condition of immunocompetent patients.[1,2] Although initially unilateral, the fellow eye can be involved within three to four weeks if untreated but can also occur decades after the initial presentation.[3,4]

Infectious etiology is due to the *Herpesviridae* family, most commonly by VZV, followed by HSV-1 and HSV-2; average age at onset is 52.4 years for VZV, 44.3 years for HSV-1, and 24.3 years for HSV-2.[5,6] Researchers have published no incidence data for ARN in the United States, but studies from the United Kingdom show a minimum annual incidence of 0.63 cases per million with a slight male predilection.[7,8] Researchers suggested ARN has outbreak seasonality, with higher incidence in the first half of the year (winter and spring) and peak incidence in February.[9] Prompt recognition and treatment is absolutely critical to optimize the visual prognosis in these patients.

ARN diagnosis is largely clinical. The American Uveitis Society (AUS) defines the diagnostic criteria as acute panuveitis with focal or multifocal peripheral retinal necrosis, occlusive retinal vasculitis (predominantly arterioles) and rapid (circumferential) disease progression in the absence of antiviral therapy.[1,3] Supporting clinical findings may include optic nerve involvement, scleritis and pain.[1] Diagnostic aqueous or vitreous humor PCR can confirm ARN and isolate an etiological organism; samples from aqueous or vitreous humor are thought to be equivocal, but some studies suggest vitreous humor may have greater yield.[8,10-13]

Therapeautics

Goals of therapy are to halt progression in the affected eye and prevent involvement of the fellow eye.[3,14-16] The mainstay of treatment involves systemic antiviral therapy with intravenous (IV) acyclovir for five to 10 days, followed by transition to an oral antiviral agent for six to eight weeks.[3,4,10,12,14-17] Oral antiviral options include acyclovir, valacyclovir, famciclovir and valganciclovir; except for acyclovir, all are pro-drugs.

Of the oral antiviral agents, valacyclovir has the greatest bioavailability and penetration of acyclovir into the vitreous cavity.[10,15,18] Moreover, high-dose oral valacyclovir (2g by mouth three times daily) can achieve vitreous concentrations comparable with IV acyclovir with a similar side effect profile.[10,16,19]

Moorfields Eye Hospital compared high-dose oral valacyclovir with IV acyclovir and demonstrated that they were clinically equivalent in best-corrected visual acuity, risk to developing a retinal detachment and safety.[20] Oral valganciclovir can achieve concentrations comparable with IV ganciclovir, but it's reserved mainly for CMV retinitis treatment.[14] Intravitreal ganciclovir or foscarnet are generally second-line options for aggressive or refractory cases not responding to systemic therapy alone; however, combined intravitreal and systemic antiviral therapy may be better than systemic therapy alone (lower incidence of retinal detachment and severe visual loss of 20/200 or worse and higher incidence of better final visual acuity).[10,14,17]

Given the robust inflammatory response seen in the immunocompetent patients of ARN, corticosteroid therapy is often necessary

to minimize damage to ocular structures.[14,15] Oral administration is best and should be administered 24 to 48 hours after initiating systemic antiviral therapy.[3,12,14] Platelet hyperaggregation has been observed in ARN and can be addressed with corticosteroids or anticoagulants such as aspirin.[14]

Complications of ARN can include retinal detachment, optic atrophy, vascular occlusion and involvement of the fellow eye.[2,3,12] Research shows a high propensity for retinas to detach, both from the atrophic nature of the necrotic retina as well as secondary to a tractional component from downstream vitreous contraction.[3,4,10]

Some have favored prophylactic laser photocoagulation to strengthen chorioretinal adhesions as a barricade posterior to areas of retinitis.[4,14] Although variable success has been reported, an obvious limitation to this option is vitreous inflammation and poor visibility impeding the ability to apply adequate laser.[3,4,12] For this reason, less severe cases are likely to have a more favorable response and outcome; conversely more severe cases are the ones that are more prone to retinal detachment.[3,4,12] Nevertheless, there is likely an indication to apply laser barricade as soon as the view allows.[3,4]

Although implementing early vitrectomy may reduce the rate of retinal detachment, one study showed final visual outcomes were equivalent with those who did not undergo early vitrectomy, likely owing to the multifactorial nature of vision loss in this population and significant role that optic atrophy can play.[12]

Herpes Association

Multiple case reports have hypothesized that ARN can be a complication of either active or prior herpetic encephalitis.[21,22] Given the rarity and mortality of herpetic encephalitis, very little data exists, so it is poorly described and understood.[21,22]

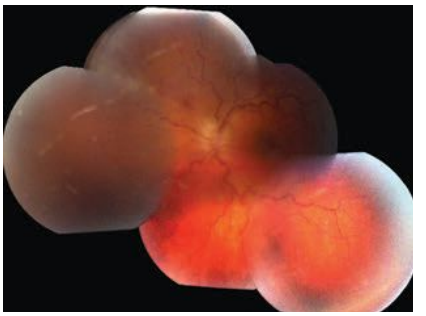

Fig. 3. These montage fundus photos portray the patient's mild vitreous haze, optic disc edema, vascular tortuosity and the posterior aspect of active retinitis superonasally.

It would appear that anybody who has suffered herpetic encephalitis may be at an increased risk of herpetic eye disease given that the eye is an extension of the central nervous system. Moreover, it may be worthwhile to consider and discuss lifelong prophylaxis with these patients who have had herpetic encephalitis with or without ocular involvement, as well as those who have had unilateral ocular involvement in efforts to spare the fellow eye.[21]

Following Up

Our patient's immediate management included in-patient admission for IV acyclovir with transition to oral valacyclovir, atropine eye drops and co-administration of prednisolone eye drops and oral prednisone after regression of retinitis was demonstrated at 36-hour follow-up. The patient received repeat intravitreal ganciclovir injections, prophylactic laser barricade, and was tapered off the steroids as the uveitis began to subside. Despite aggressive therapy, the patient was poorly compliant to maintenance dosing of oral valacyclovir and eventually reactivated with subsequent rhegmatogenous retinal detachment. Best-corrected acuity in the left eye was 20/250 at most recent follow-up (nine years since initial presentation). ■

Dr. Aboumourad practices at the Bascom Palmer Eye Institute in Miami.

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Edited By Carlo J. Pelino, OD, and Joseph J. Pizzimenti, OD

When Facial Paralysis Strikes

Diagnosing and managing Bell's palsy requires optometrists to rule out any underlying condition that could be triggering a patient's signs and symptoms.

By Sean Gretz, OD

A 65-year-old African American male presented to the primary care clinic complaining of irritation and foreign body sensation in his right eye. He had excessive tearing and couldn't close his right eye. His symptoms began two weeks prior, following a chemotherapy session.

The patient is currently being treated for multiple myeloma and hypertension. He recovered from hepatitis and prostate cancer 15 years ago, which was treated with radiation. He also reported multiple spinal fractures and open-heart surgery over 20 years ago.

The patient denied diplopia, headache and loss of vision as well as any neurological symptoms of unilateral weakness or slurred speech. His entering blood pressure was high at 160/100mm Hg, but he denied chest pain, headache, shortness of breath and dizziness.

Case Findings

The patient's entering best-corrected visual acuities were 20/30 OD and 20/20 OS. Pupil and motility testing revealed no abnormalities. His confrontation visual fields were unremarkable OU. Slit lamp examination revealed a quiet anterior chamber OU, conjunctival injection with lagophthalmos OD and significant superficial keratitis OD. His intraocular pressures were measured at 15mm Hg OU. Dilated fundus examination revealed arterial attenuation but no other notable pathology. The patient displayed a right-sided facial palsy involving the upper and lower face (*Figures 1-3*).

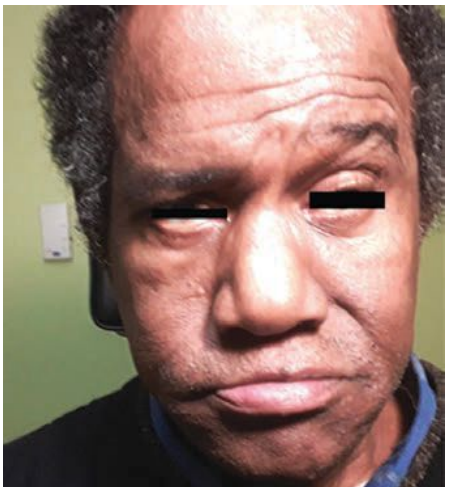

Fig. 1. The patient suffers from paralysis of the right side of the face in primary gaze.

Since motility and cover testing did not reveal any significant deviation or underaction, we determined that cranial nerves III, IV and VI were unaffected. The patient's corneal sensation was intact, as were the maxillary and mandibular branches of the trigeminal nerve. We assessed the patient for balance and gait issues to rule out cranial nerve VIII involvement. He denied any dampening of sound or hyperacusis, which would indicate involvement of the stapedius muscle—a common occurrence in Bell's palsy. Our assessment of cranial nerves IX through XII was unremarkable as well.

We performed Humphrey visual field testing to rule out field loss and cerebral involvement (*Figure 4*). Our results were moderately reliable and did not indicate cerebral involvement or neurological field defect. Assessment of the optic nerve found well-perfused tissue with distinct margins and no evidence of optic atrophy, edema or excavation. OCT showed no significant retinal nerve fiber layer (RNFL) dropout or signs of optic neuropathy OD or OS (*Figure 5*).

We promptly referred the patient for a gadolinium-enhanced MRI of the parotid gland, temporal bone and brain. The MRI did not reveal metastasis, neoplasm or tissue enhancement. Enhancement of the cranial nerve VI nucleus in Bell's palsy is reported in 57% to 100% of patients. A lack of enhancement

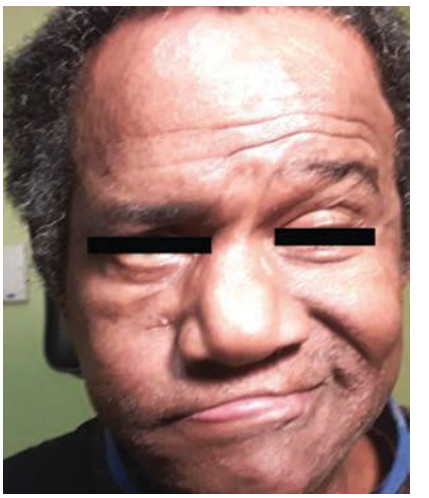

Fig. 2. Weakness of the frontalis muscle, orbicularis oculi and lower cheek are notable in stasis and while frowning.

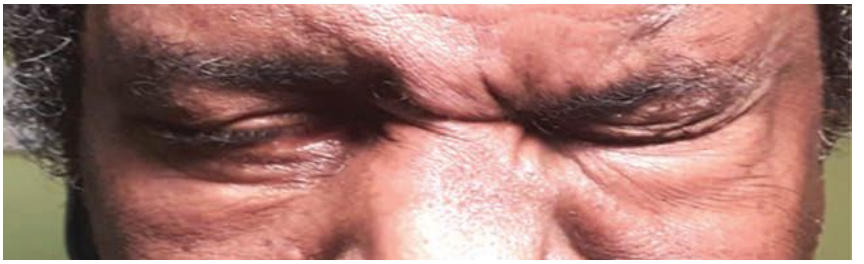

Fig. 3. The patient is unable to forcefully close his right eye due to orbicularis weakness.

is considered a good prognostic sign.

We ordered serologic testing, including CBC with differential, ESR, Lyme anti-body, Epstein-Barr titer, FTA-ABS, RPR and ANA, in coordination with the patient's oncology team. There was no indication of an inflammatory or infectious cause of the patient's condition.

Follow-up

After two weeks of treatment with topical lubricants, the patient returned for a follow-up exam. He reported good ocular comfort with topical lubrication, and his corneal epithelium had significantly improved. His visual acuity had improved to 20/25+1 OD, and we observed a mild improvement in his orbicularis oculi function. The patient could now completely close his right eye with minimal effort (*Figure 6*).

He came in again after six weeks, during which time his muscle function had continued to improve with no residual weakness or synkinesis.

Discussion

Bell's palsy, also known as facial nerve palsy, is a common clinical presentation seen in the primary care setting. It is defined as an acute, ipsilateral facial nerve (cranial nerve VII) paralysis of unknown etiology that results in weakness of the platysma and muscles of facial expression.[1,2] Bell's palsy is the most common disorder that affects the facial nerve and is responsible for about 80% of all facial mononeuropathies.[3] Its annual incidence is 15 to 30 per 100,000 people, with equal numbers of men and women affected.[2,3]

Bell's palsy is a diagnosis of exclusion; therefore, a thorough medical history and review of systems are paramount in assessing the risk of a systemic cause.[1-3] Conditions that may mimic Bell's palsy include CNS neoplasms, stroke, HIV, multiple sclerosis, Guillain-Barré syndrome, Ramsay Hunt syndrome, Melkersson-Rosenthal syndrome, Lyme disease, otitis media, cholesteatoma, sarcoidosis, trauma to the facial nerve, autoimmune diseases—such as Sjögren's syndrome—and metabolic disorders, including diabetes.[3]

Idiopathic facial palsy is believed to have an inflammatory pathophysiology. Herpes simplex virus (HSV) activation has been implicated, though the evidence is not entirely conclusive.[3,4] HSV-1 genomes were identified in the facial nerve endoneurial fluid and auricular muscles of 11 of 14 patients undergoing decompression surgery for Bell's palsy but in no controls.[3-5]

Management is geared toward reducing facial nerve inflammation and preventing corneal complications that stem from paresis of the facial muscles and depends on the underlying etiology. When Bell's palsy presents acutely, stroke or cerebrovascular incident must be ruled out as the cause of the facial weakness.[2,3,5] Signs of a stroke include slurred speech, unilateral weakness, vision loss, dizziness and disorientation. Immediate referral to the emergency room is warranted if any of these signs are noted.

Corticosteroids are currently the drug of choice when medical therapy is needed.[3,6] Early treatment with oral glucocorticoids within 72

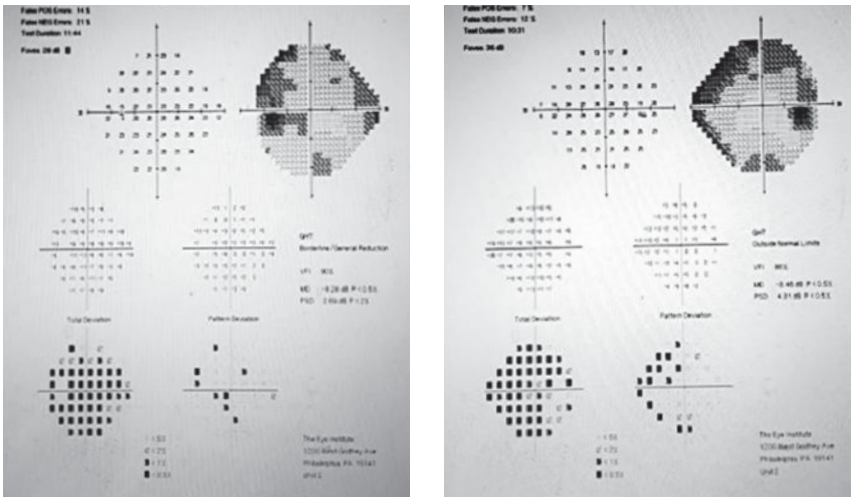

Fig. 4. Humphrey visual field testing of the left eye (left) and right eye (right) shows some points of loss inferior to fixation OD and superior nasal OS.

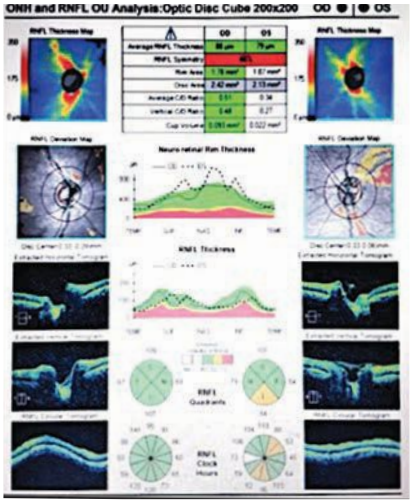

Fig. 5. OCT imaging shows no evidence of RNFL dropout OD and borderline thin RNFL superior temporal to the disc OS.

hours of onset has been shown to expedite the resolution of paralysis with limited residual symptoms.[6] The suggested regimen is 60mg to 80mg of prednisone per day for one week, after which point it should be tapered off by 10mg per day.[3,6,7]

Our patient in this case was initially seen after the 72-hour window, so we did not prescribe an oral medication. Oral antivirals have been widely prescribed as monotherapy or in combination with steroids; however, their effectiveness for Bell's palsy is widely debated.[3,4,8]

The primary objective in cases of Bell's palsy is to maintain corneal integrity. Topical lubrication is the first-line treatment and can be prescribed QID or even as often as Q1H depending on the severity of the condition. If corneal integrity is highly compromised, moisture goggles, amniotic membrane therapy or tarsorrhaphy may be indicated. The literature shows no consensus for the benefit of, or indication for, surgery in the treatment of Bell's palsy.[3,6]

Patients who do not fully recover their facial function can have varying degrees of facial weakness, hypertonia and synkinesis, which can all be managed with physical therapy.[3,4,6] Synkinesis results from post-paralytic re-innervation of different muscles by axons from the same motor neuron. An example found in (aberrant) Bell's palsy regeneration is eyelid closure when a patient smiles. Botulinum toxin injections may benefit patients with synkinesis, facial spasm or hyperlacrimation.[3,6] Weight insertion into the upper eyelid or tarsorrhaphy can improve eyelid closure. Cosmetic and functional improvement may be possible with facial reanimation surgery. Most surgeons will not perform reanimation surgery unless no improvement has been noted for at least nine months.

Patients with Bell's palsy will have a favorable prognosis if some recovery is seen within the first 21 days of onset and should have some notable recovery by four months after the onset of symptoms.[3,6] If no improvement is noted by then, repeat imaging and additional work-ups may be indicated. An MRI delineates the soft tissue structures and is the best way to evaluate the intraparotid facial nerve for inflammation, edema or neoplasm.

When a patient presents with acute onset facial nerve palsy, a thorough history and physical examination should be performed. The clinician must selectively test the involved muscles of the face and order additional neurological and serological testing as necessary to further assess for pathology.

Although our suspicion for Bell's palsy was high at the onset, this patient had several underlying conditions, including metastasis, stroke and infection, that complicated the case. This highlights the importance of fully evaluating a high-risk patient who presents with neurological abnormalities in a primary care setting.

Dr. Gretz provides comprehensive eye care with a focus on ocular disease and emergency medicine at Simon Eye Associates and is a member of the Delaware Optometric Association and the American Optometric Association. He graduated from the Pennsylvania College of Optometry at Salus University in 2018, where he completed a residency in primary care and ocular disease.

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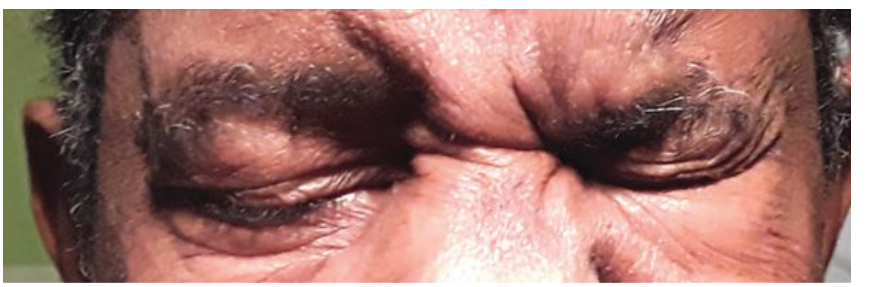

Fig. 6. The patient's orbicularis function visibly improved by his six-week follow-up.

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Meetings + Conferences

NOTE: Information is subject to change due to the pandemic. Please contact meeting organizers to confirm events and dates.

August

■ **5.** *NJAO Annual Summer Seminar*. Jumping Brook Country Club, Neptune, NJ. Host: New Jersey Academy of Optometry. CE hours: 6. For more information, email Dennis Lyons at dhl2020@aol.com or go to www.aaopt.org/membership/us-and-international-chapters/njchapter.

■ **8-9.** *Glaucoma in the Gorge*. Best Western Conference Center, Hood River, OR. Host: Ocular Therapeutics Continuing Education. Key faculty: Tony Litwak, Jim Thimons. CE hours: 10. For more information, email Tony Litwak at info@otce.net or go to www.otce.net.

■ **14-15.** *Envision Virtual Conference East 2020*. Host: Envision University. CE hours: Total: 32, maximum per OD: 11. For more information, email Michael Epp at michael.epp@envisionus.com, call 316-440-1515 or go to www.envisionconference.org.

■ **21-23.** *UAB School of Optometry Fall Continuing Education Weekend (Virtual)*. Host: UAB School of Optometry. CE hours: 18. For more information, email Kathryn Trammell at ktram@uab.edu, call 205-934-5701 or go to uab.edu/optometry/ce.

■ **28-30.** *Northern Escape 2020.* Delta Hotels by Marriott Quebec, Quebec City. Host: Optometric Education Consultants. CE hours: 15. For more information, email Vanessa McDonald at optoec@gmail.com, call 954-612-4142 or go to www.optometricedu.com/home.

■ **30.** *NECO Ocular Surface Symposium with Dry Eye Coach*. New England College of Optometry (NECO), Boston. Host: NECO. Key faculty: Whitney Hauser, Walt Whitley, Scott Schachter. CE hours: 8. For more information, email Morris Berman at bermanm@neco.edu or call 617-266-2030.

September

■ **5-9.** *VT1/Visual Dysfunctions (Virtual).* Host: Optometric Extension Program Foundation. Key faculty: John Abbondanza. CE hours: 35. For more information, email Karen Ruder at karen.ruder@oep.org, call 410-561-3791 or go to www.oepf.org.

■ **6-10.** *Tropical CE Sonoma 2020*. Hyatt Regency Sonoma, Sonoma, CA. Host: Tropical CE. Key faculty: John Mc Greal, Jr., Jill Autry. CE hours: 14. For more information, email Stuart Autry at sautry@tropicalce.com or go to www.tropicalce.com.

■ **7-8.** *Primary Eye Care Update.* NSU Event Center, Tahlequah, OK. Host: Oklahoma College of Optometry. CE hours: 10. For more information, email Callie McAtee at mcateec@nsuok.edu, call 918-316-3602 or go to optometry.nsuok.edu/continuingeducation/scheduleofevents/primaryeyecareupdate.aspx.

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Diagnostic **Quiz**

He Kept His Eye on the Ball

A patient develops worsening near vision three years after a blunt trauma.

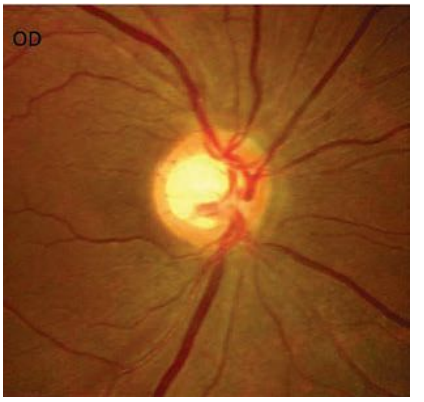
By Andrew S. Gurwood, OD

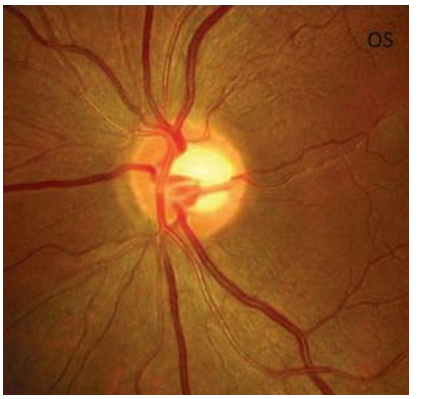
History

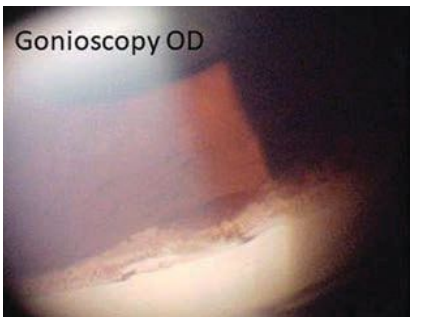
A 44-year-old male presented to the office for a routine eye exam. He reported that his vision was fine at distance but that he now needed reading glasses. He denied previous surgery or any history of glaucoma, but he did say he incurred a blunt trauma—hit in the face with a football—to his right eye three years earlier. He denied systemic diseases and allergies of any kind.

Diagnostic Data

His best-corrected entering visual acuities were 20/20 OD and 20/20 OS at distance and near. Refraction uncovered mild hyperopia with presbyopia measuring +0.50/+1.75 OU. His external examination was unremarkable with no evidence of afferent pupillary defect. His biomicroscopic examination was essentially normal with some pigmentation granules observed on the endothelium, OD. Goldmann applanation tonometry measured 30mm Hg OD and 21mm Hg OS. The pertinent anterior and posterior segment findings are demonstrated in the photographs.







After a little roughhousing injured his eye, a patient's near vision got progressively worse for three years. Using this history, fundus photos and a gonioscopy exam, can you help identify why he suddenly needs reading glasses?

Your Diagnosis

Does the case presented require any additional tests, history or information? What would be your diagnosis? What is the patient's likely prognosis? To find out, visit www.reviewofoptometry.com. ■

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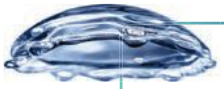

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