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Geographic Atrophy: Diagnosis, Imaging, and Collaboration

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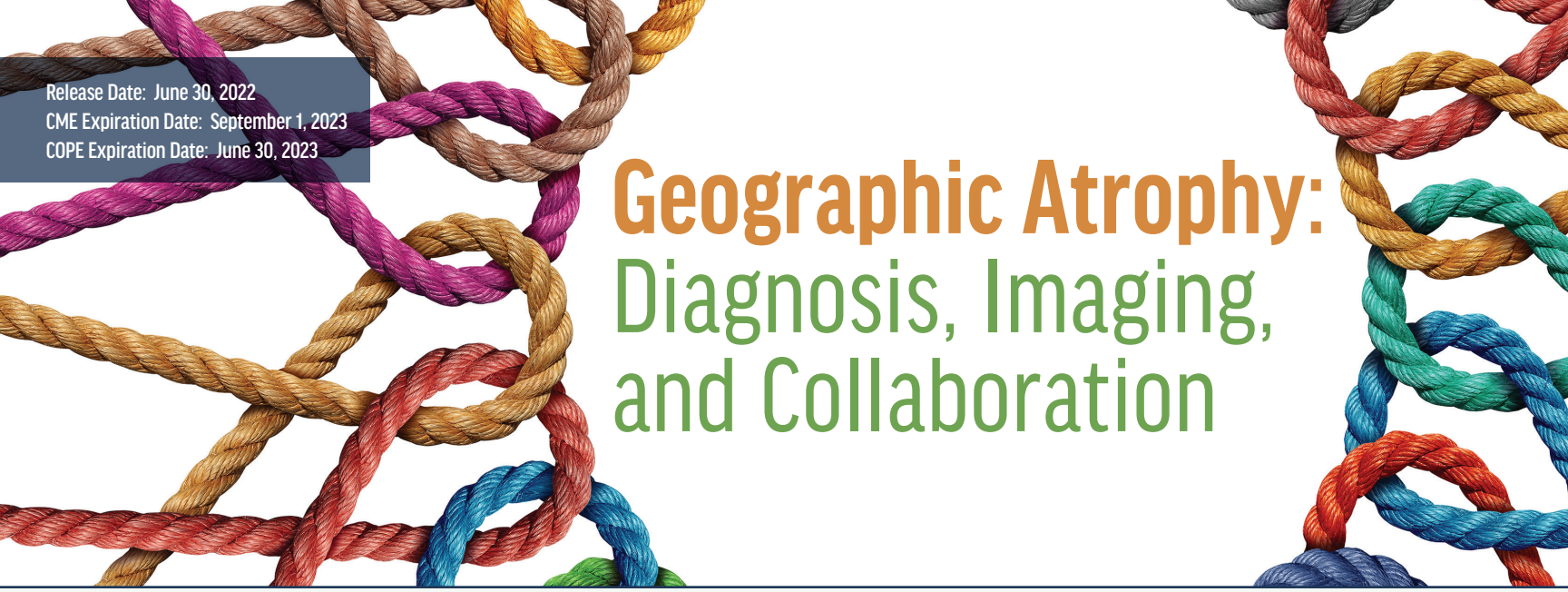

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Geographic Atrophy: Diagnosis, Imaging, and Collaboration

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Content Source

This continuing education (CE/CME) activity captures content from a roundtable discussion.

Activity Description

This supplement summarizes a discussion on geographic atrophy, including prevalence, burden, diagnosis, and imaging.

Target Audience

This certified CE/CME activity is designed for optometrists.

Learning Objectives

Upon completion of this activity, the participant should be able to:

- **Summarize** the prevalence of AMD and GA and **define** the burden of illness linked specifically to GA
- **Describe** GA disease detection and factors influencing progression

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PRETEST QUESTIONS

Please complete prior to accessing the material and submit with Posttest/Activity Evaluation/Satisfaction Measures for credit.

1. Please rate your confidence in your ability to describe geographic atrophy (GA) disease detection and factors influencing progression (based on a scale of 1 to 5, with 1 being not at all confident and 5 being extremely confident).

A. 1
B. 2
C. 3
D. 4
E. 5

2. What was the global prevalence of age-related macular degeneration (AMD) in 2016?

A. ~130 million patients
B. ~170 million patients
C. ~230 million patients
D. ~270 million patients

3. A 66-year-old African-American man presents to your office for routine eye examination. He has a family history of AMD. His social history is positive for tobacco and alcohol use. He has a history of diabetes, hypertension, and obesity. All of the following are risk factors for AMD and GA in this patient EXCEPT:

A. Family history of AMD
B. African-American race
C. Smoking history
D. History of obesity

4. All of the following are good tests to assess GA progression, EXCEPT:

A. Contrast sensitivity measurement
B. Dark adaptation
C. Reading speed
D. Best corrected visual acuity

5. A 77-year-old patient with a history of AMD and GA in his right eye presents to your office for evaluation. He notes that his vision seems to have gotten progressively worse over time, however on examination today his visual acuity measurement is stable from a year prior. Which of the following tests might be more useful in determining this patient's current visual function and predict potential future vision loss?

A. Low luminance visual acuity
B. Fluorescein angiography of the right eye
C. Corneal pachymetry
D. Corneal hysteresis

6. A 69-year-old woman with a history of AMD and GA in both eyes presents to your office for evaluation. Of the following, what imaging modality would be the best to quantify and track her GA, as well as the status of her AMD?

A. Color fundus photography
B. Spectral domain optical coherence tomography (SD-OCT)
C. B-scan ultrasonography
D. Corneal topography

7. A 75-year-old new patient presents to your clinic for evaluation. She has a history of bilateral visual loss. On examination, you note bilateral presence of hypopigmented deposits throughout the macula. Your differential diagnosis for this patient includes both AMD and Stargardt disease. What imaging modality might help you differentiate between these two disease entities?

A. SD-OCT
B. B-scan ultrasonography
C. Color fundus photography
D. Fundus autofluorescence

8. Which of the following fundus autofluorescence GA patterns is indicative of higher likelihood of progression?

A. Small lesion
B. Unifocal lesion
C. Diffuse-trickling pattern lesions
D. Banded lesions

9. A 75-year-old Caucasian man presents to your office for evaluation. On exam, you note bilateral medium-sized drusen and some GA. You obtain an SD-OCT that shows evidence of drusen as well as subretinal drusenoid deposits. You also note bilateral cataract. Which of the following features of this patient's exam predict highest risk for GA progression?

A. Medium-sized drusen
B. Geographic atrophy
C. Subretinal drusenoid deposits
D. Cataracts

10. A 98-year-old patient with AMD and GA presents to your office for evaluation. On exam, you note bilateral small drusen in both eyes. On SD-OCT, you note hyporeflective drusen cores, intraretinal hyperreflective foci, subretinal drusenoid deposits, and a small extrafoveal pigment epithelial detachment. All of the following put this patient at increased risk for progression to late AMD and atrophy, EXCEPT:

A. Hyporeflective drusen cores
B. Intraretinal hyperreflective foci
C. Subretinal drusenoid deposits
D. Pigment epithelial detachment

11. Which of the following factors have been linked with a high rate of GA progression?

A. Unifocal lesions
B. Unilateral disease
C. Multifocal lesions
D. Presence of cataracts

GEOGRAPHIC ATROPHY: DIAGNOSIS, IMAGING, AND COLLABORATION

We may be sitting on the cusp of a new era of eye care. Two drugs—pegcetacoplan and avacincaptad pegol—are in phase 3 studies for the treatment of geographic atrophy (GA), a progressive disease resulting in central vision loss, for which no drug has been approved. If the FDA were to approve either or both of these drugs for the treatment of GA, patients could be eligible for a treatment that would slow the progression of their disease.

Optometrists and ophthalmologists have become collaborative partners in the treatment of wet age-related macular degeneration (AMD). Current referral patterns serve as a model for collaboration that may be implemented if a drug for GA is approved. Given the increasing likelihood of an intravitreal treatment for GA and the diverse practice patterns in our respective offices, a panel of optometrists and ophthalmologists was convened to discuss the contemporary dynamics surrounding diagnosis, treatment, patient management, and referral.

—SriniVas Sadda, MD, and Yasha Modi, MD
Program Co-Chairs

EPIDEMIOLOGY AND SOCIETAL BURDEN OF AMD AND GA

Dr. Modi: The global prevalence of AMD is staggering. An estimated 170 million patients had AMD in 2016,[1] and it is expected to increase to 288 million patients by 2040.[2] In the United States, approximately 11 million patients had AMD in 2016,[1] and that total is expected to double by 2050.[3]

AMD can be classified as early, intermediate, or advanced; advanced disease is subtyped as neovascular (or wet) AMD or as advanced atrophic with GA being the end stage. In 2014, approximately 5 million patients around the world had GA in at least one eye.[2,4] In the United States in 2020, an estimated 3 million patients had GA.[5]

Age is a leading risk factor for developing GA. Epidemiology studies observed that among patients 43 to 53, almost no patients have GA. However, disease prevalence increases to 1.3% in patients aged 59 to 69, 3.2% in patients aged at least 75, 6.7% in those aged at least 80, and nearly 12% in patients older than 85.[6-8]

Dr. Sadda: There are a handful of other risk factors for AMD and GA that have been identified, including family history of AMD, white race, smoking, obesity, hypertension, and genotype.[9-11] Current smoker status is a risk factor for advanced AMD development,[12] and smoking pack years can be used to determine risk for developing visual loss due to AMD.[13] The presence of the alleles CFH and ARMS2 are linked with developing early AMD, and the presence of at least one of those risk alleles is linked with advanced AMD development.[12] The presence of the CFH variant Y402H is linked with such high risk of GA development that it has been found to be a risk factor independent of smoking history.[13]

Dr. Modi: Information about disease prevalence and risk factors offers a broad view of GA. When we zoom in, we better understand how GA affects our patients on a day-to-day basis.

Q | Dr. Rodman, as someone who practices in a high-volume eye clinic with a variety of presenting pathologies, how have you observed GA affecting the lives of your patients?

Julie Rodman, OD, MSc: Patients whose vision has been severely affected by GA progression are robbed of their independence. When patients can no longer drive or safely navigate public spaces, quality of life is drastically curtailed. Extensive research has proven that the ability to perform a range of tasks—from computer use and reading to meal preparation and self-care—are negatively associated with AMD.[14]

Anecdotally, I have observed increased anxiety and depression in patients who are experiencing vision loss due to GA. The literature supports that observation. Dawson et al found that depression rates were significantly higher in patients with AMD than in those without it.[15] The fact that no safe and effective therapy has been approved for the treatment of GA may play a role in patient mindset. Patients with wet AMD whose disease has responded to therapy have been classified as "cautiously optimistic" about their future, whereas patients with GA experienced "profound loss."[16] We cannot forget about the deleterious mental health consequences of vision loss among our patients with GA.

Jessica Steen, OD: I've observed similar dynamics. In formal sense, we can assess patients' abilities and limitations via use of the National Eye Institute Visual Function Questionnaire 25 (VFQ-25). In a clinical environment, however, such tools may be impractical. Rather, a conversation about a patient's vision-related challenges and goals may better provide the clarity and specificity eye care

providers need to properly assess real-world visual function.

Embarrassment, social isolation, and low self-esteem are real consequences of GA. Because optometrists are often the first clinicians to encounter patients with GA, we are compelled to listen to our patients' mental health concerns and connect patients to counseling services through their primary care physician or community-based programs. Now that there are treatments for GA on the horizon, we need to start thinking about the changes in referral practice patterns to retinal specialists in order to ensure prompt treatment, once available.

Steven G. Ferrucci, OD: Optometrists who will begin referring patients to ophthalmologists after a therapy is approved should begin tracking the progression of patients' GA now so that a robust patient history can be provided upon referral. However, this in itself presents a challenge.

One of the biggest obstacles that eye care providers face is characterizing and tracking vision loss due to GA progression. For many conditions, best corrected visual acuity (BCVA) is an effective metric for visual function. This is not the case for GA, which manifests as a single or as a series of scotomas on the retina and results in reduced vision at various patches in the visual field. Functionally, this means that reading may be difficult for patients, that contrast sensitivity may worsen, and that dark adaptation may take longer. Those quality of vision issues cannot be measured by BCVA and a standard chair examination.

Dr. Rodman: Conventionally, a patient with 20/30 BCVA is considered to have good vision. However, objective measurements of visual acuity do not always relate to functional abilities. For example, a patient who is unable to read quickly, perform activities of daily living, or function safely in dark rooms is not experiencing the quality of life we'd expect from a patient who has 20/30 vision.

Dr. Sadda: One potential area of concern I have with GA patients is driving safety. Even when the fovea is not involved and the visual acuity is good, some lesion configurations will be problematic.

Dr. Ferrucci: I have a patient with a large GA lesion that spares the macula. (*See Case 1 on page 11 for further details.*) If he were to have his visual acuity assessed by his local department of motor vehicles, he would likely pass. However, given the location and size of his scotomas, I am certain that he is unable to safely drive a car.

Rishi P. Singh, MD: Driving is one the most important activities of daily living concerning those with GA. A majority (52%) of GA patients who still carry a driver's license do not feel confident in their ability to drive during daytime hours; that number rises to 88% when asked about nighttime driving.[17] In the United Kingdom, approximately two-thirds of patients were ineligible to drive within a median 1.6 years of GA diagnosis.[18]

Dr. Modi: Being legally eligible to drive but functionally unable to do so illustrates a major disconnect between the metrics used to assess GA and the reality of functional vision.

Q | Dr. Steen mentioned the impracticality of the VFQ-25 in a clinical setting. Are there other assessments that might be more conducive to real-world practice? And are eye care providers using them?

Dr. Ferrucci: Tests evaluating contrast sensitivity, dark adaptation, or reading speed have been used to assess GA progression; indeed, reading speed has even been shown to correlate with GA lesion size.[19] Still, few eye care providers employ them in examinations of patients with GA. These tests either require specialized equipment or interrupt workflow to the point that they become untenable for a typical optometric practice. That said, I think low luminance visual acuity (LLVA) evaluations could possibly be adopted by some optometric clinics as an effective tool for GA assessment.

Dr. Modi: I agree that LLVA might be a tool that more clinics—both optometric and ophthalmic—could effectively employ. LLVA requires placing a neutral density filter over the patient's eye and asking them to read a typical Snellen chart. Among patients with early GA who present with otherwise good BCVA and functional vision, diminished LLVA performance may predict subsequent vision loss.[20] That could be especially important when it comes to determining which patients are most at-risk for progression—and which patients should be seen with the utmost priority by providers.

Dr. Sadda: In 2016, my colleagues and I published a paper that, in part, argued that LLVA should be included as an outcome in clinical trials evaluating patients with GA.[21] In particular, we found the sensitivity of LLVA to be preferred to that of BCVA assessments.

CLINICAL PRESENTATION OF GA IN THE OPTOMETRIC CLINIC

Dr. Sadda: In current clinical practice, many patients with GA are not under the care of retina specialists. Rahimy et al reported in 2020 that 26% of real-world patients with GA were under the care of an optometrist or general ophthalmologist.[22] I'd like to learn more about the severity of AMD most commonly encountered by the optometrists on this panel.

Q | What, in your estimation, is the breakdown of AMD presentations in your respective optometric clinics?

Dr. Steen: A majority of the AMD I see is intermediate in nature. Patients with intermediate AMD typically stay within our orbit of care, and we are not currently referring to retina specialists for collaborative management. We continue to monitor these patients for biomarkers associated with high risk for disease progression and tailor our follow-up schedules according to overall risk and, of course, refer them to a retina specialist as soon as evidence of wet AMD is detected.

Dr. Ferrucci: I estimate that half of the AMD patients I see at the Sepulveda VA Medical Center have intermediate AMD or advanced AMD. This may be due to the older population we specialize in treating. When we observe advanced AMD, we refer patients to the hospital's Retina Service for treatment and continue to monitor them during follow-up appointments.

The other half of the AMD patients who rotate through my clinic have early AMD, and I encourage them to return for routine follow-up appointments so that I can monitor any changes to their anatomy or visual function. I also advise that they come to our office if they detect any subjective changes to their vision.

Dr. Rodman: My clinic, too, refers patients with exudative disease to a retina specialist. As of now, however, many optometrists do not refer patients with GA to a retina specialist. Until there is a safe and effective treatment, I think this will remain the practice pattern.

Dr. Steen: That said, optometrists may refer GA patients to a low-vision specialist in hopes of mitigating the downward spiral of quality of life we alluded to in the opening portion of our conversation. Unfortunately, many optometrists feel that a low-vision specialist referral is the best we can offer at the moment.

Dr. Ferrucci: I refer GA patients with significant deterioration to their anatomy or visual function to our in-house retina specialty team, fully aware that there is no therapy available just yet.

Dr. Modi: If a treatment is eventually approved, I anticipate that optometrists will start referring GA patients to retina clinics for evaluation. Even if GA patients arrive in retina clinics before a GA therapy is actually in our hands, retina specialists will be able to enter those patients into their patient management systems, establish relationships with them, and promptly alert them when a treatment is available.

Dr. Singh: Referring early GA patients is so important in the waiting period between phase 3 clinical trial data and FDA approval, which is the in-between zone we currently inhabit. Because the drugs that are under investigation for GA that are farthest along arrest GA progression rather than reverse it, we might be able to maximize the therapeutic effect for patients if we administer therapy before their vision deteriorates. Although we still need more data to say it with certainty, it seems that we may not be able to do much for patients with advanced GA. The earlier the referral, the better.

EVALUATION OF IMAGING MODALITIES FOR CHARACTERIZATION OF GA

Dr. Modi: Eye care providers have struggled to find a functional vision test with adequate sensitivity that allows clinical workflow to remain intact and provides enough data so that clinicians can track disease progression. However, imaging modalities have been found to be an effective tool for characterizing the anatomy of patients with AMD and GA—including among those who present with very good BCVA.

Q | **Which imaging modalities are being used by the panel to evaluate patients with intermediate AMD and GA?**

Dr. Rodman: Color fundus photography (CFP) can be used to provide a baseline image of a GA patient. CFP is widely available to eye care providers, and has been used in a number of studies to classify dry AMD lesion types[23] and track the natural history of GA.[24,25] For these reasons, optometrists who don't have access to higher technology should consider using CFP to track GA lesions.

Personally, I find spectral-domain optical coherence tomography (SD-OCT) to be one of the best imaging modalities for evaluating GA patients. Objectively, the body of evidence characterizing the value of SD-OCT imaging is large[26-29]; specifically, it helps to identify anatomical abnormalities in the outer retina and choroid and thus allow us to grade and classify lesions appropriately.

I also find OCT angiography (OCTA) to be an effective tool, even if the modality does take a little bit longer to acquire an image. Among these OCT modalities, primary eye care providers are more likely to have access to SD-OCT, and I expect that many of them have experience with interpreting SD-OCT imaging reports.

Dr. Modi: What are the specific brands that everyone uses for imaging?

Dr. Rodman: At my clinic, we employ the Optovue (Visionix) SD-OCT platform with an outstanding angiography platform.

Dr. Steen: My work at Nova Southeastern University gives me access to a variety of technologies. My colleagues and I use devices from three manufacturers: the Cirrus line of OCT platforms (Zeiss), the Optovue SD-OCT platform, both with OCTA capabilities, and the Spectralis family of OCT platforms (Heidelberg).

Dr. Ferrucci: I have access to a Spectralis and an Optovue, the latter of which has OCTA capabilities. In my estimation, about two-thirds of optometrists have access to an SD-OCT platform, most of whom would use the Optovue. A few optometrists may still be employing a time-domain OCT, but I think that they're outliers.

Dr. Modi: What about the retina specialists on the panel?

Dr. Sadda: I agree that a baseline CFP image is a great place to start with a patient who has intermediate AMD or GA, and acquisition of fundus autofluorescence (FAF) at baseline if available may also be useful in tracking progression over time. I use widefield imaging platforms, which are easy to use. I find them particularly useful in patients with disease that may extend to the periphery. When it comes to intermediate AMD patients, interpretation of SD-OCT images is a highly effective means of stratifying patients into risk groups, which in turn dictates our directed follow-up scheduling.

Among routine GA patients, I have not found a clinical use for

OCTA quite yet, although I find it valuable at the research level. That said, it is a useful tool for patients who may have progressed to neovascular AMD.

Dr. Singh: SD-OCT is my go-to modality for evaluating patients with intermediate AMD. I pay special attention to both the macula and the extrafoveal region, the latter of which is easy to overlook or deemphasize during examination. Doing so puts us at risk of missing a diagnosis of extrafoveal GA. FAF, too, is an effective means of detecting extrafoveal lesions.

I document in the patient's health record where lesions are located, and submit the proper ICD-10 codes for foveal or extrafoveal GA. Although we may not be able to administer therapy just yet, documenting the location of lesions in patients with GA may be key if the approved indication for a GA therapy is, in part, based on lesion location. I would advise that clinicians who see patients with GA lesions begin changing their documentation patterns now, thereby developing a routine for establishing patient history in anticipation of an approved therapy.

I find OCTA to be an attractive modality for GA evaluation, but we should note that a significant number of artifacts appear on imaging reports. Correcting for these artifacts requires use of additional imaging software, which is itself cumbersome and likely to slow a clinic's workflow.

Dr. Sadda: In a sense, I think of CFP as the most traditional (and highly researched) modality, SD-OCT and FAF as the modalities most popular in the present, and OCTA as the modality that will be most leveraged in the future. Dr. Rodman, you have extensively used OCTA imaging. How exactly are you using it for intermediate AMD and GA patients?

Dr. Rodman: I utilize software that is built into Optovue technology (Angioanalytics) that provides a quantitative analysis of flow area within an exudative lesion. This technology allows for early detection of lesions as well as tracking of lesions pre- and post-treatment. I am aware that many of my colleagues do not have access to OCTA platforms—but still, I find this way of tracking anatomic changes to be highly effective.

ASSESSING RISK OF INTERMEDIATE AMD AND GA PROGRESSION

Dr. Modi: Changes in choriocapillaris vessel density on OCTA may potentially play a prognostic role in AMD, but, for the moment, clinicians rely on biomarkers that are more easily observed with less advanced equipment. Drusen size and volume has been used as a biomarker, as so has the presence or absence of pigment demonstrated clinically or on fundus examination. This has been well studied.[30-33]

Dr. Singh: I continue to rely on the simplified scale established by the Age-Related Eye Disease Study, or AREDS.[34] In that system, drusen and pigmentation are weighed alongside factors such as bilaterality to determine a 5-year risk progression. (*To explore risks associated with bilateral disease, see Case 2 on page 11.*)

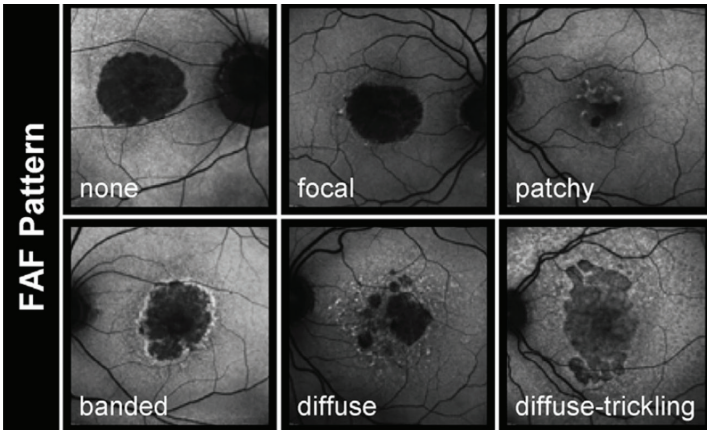

Figure 1. Lesion patterns observed on FAF may be key to determining which patients are at the highest risk of progression. Patients with banded and diffuse-trickling lesions are among the highest risk for progression.

Q | **Dr. Modi: What role does FAF play when it comes to classifying risk of progression?**

Dr. Sadda: FAF has numerous uses in characterizing nonexudative disease in this patient population. In patients with intermediate AMD, FAF imaging findings can confirm an AMD diagnosis and rule out a masquerading condition such as Stargardt disease. In fact, Stargardt disease is so easily misdiagnosed as AMD that some patients with Stargardt disease have been enrolled in AMD clinical trials. FAF imaging increases my confidence that anatomic manifestations of disease are, in fact, due to AMD.

Autofluorescent patterns along the border of GA lesions, as well as the shape and pattern of a lesion, allow clinicians to categorize lesions (Figure 1). Because patients with particular GA lesion patterns are more likely to progress than others, acquiring baseline FAF in new GA patients is helpful for determining their risk of progression. Small and unifocal lesions are less likely to progress than large and multifocal lesions.[35] Lesions with a hyperautofluorescent band at the lesion margin, which are categorized as banded lesions, are at higher risk for more rapid progression.[36] Other high-risk classifications include diffuse and diffuse-trickling lesions: Diffuse lesions will show FAF spots outside of the GA lesion area itself and will spread toward the posterior pole[35] whereas diffuse-trickling lesions will present as gray (ie, not black[37]) hypoautofluorescence in the diffuse pattern. Diffuse-trickling patterns are associated with especially rapid progression of GA.[38]

FAF images showing hypofluorescent lesions indicative of atrophy are intuitive to patients, and they serve as a useful educational tool during a consultation. They may also serve a critical function in the near future: if a therapy is approved for GA, we might begin a roll out of therapy by prioritizing patients whose extrafoveal lesions are at the highest risk for progression but whose lesions have not yet affected the central visual field.

Dr. Modi: Given the value of FAF, why haven't more clinicians adopted it for use in GA patients?

Dr. Sadda: FAF imaging requires a bright flash that might be uncomfortable for some patients. Even I don't use it in a majority of my patients as a monitoring tool. Doctors' and patients' preference for SD-OCT is rooted in its convenience for both parties and its ability to reliably image GA patients.

Dr. Rodman: When patients sit for SD-OCT imaging, near-infrared reflectance (NIR) imaging can be simultaneously acquired on many platforms. NIR allows clinicians to observe reticular pseudodrusen (RPD), also called subretinal drusenoid deposits, which present as hyperreflective foci.[39] It may be difficult for some eye care providers to distinguish RPD from drusen. Doing so, however, is key to assessing risk of progression: patients with RPD may be nearly 5 times more likely to develop GA progression compared with patients who do not have RPD.[40] FAF is also able to detect RPD, but, as we've discussed, many clinicians' do not have access to FAF thus limiting the diagnostic value of this technology.

Dr. Sadda: In a natural history of drusen study, we identified that intermediate AMD eyes with heterogeneous internal reflectivity within drusen (also termed hyporeflective drusen cores or hDC) or intraretinal hyperreflective foci (HRF) were associated with a higher risk for progression to late AMD.[41] A 2018 study by Tan et al found that these areas of hDC corresponded to calcific nodules within the drusen.[42] Histopathologic correlation studies have shown that HRF can correspond to RPE cells that have migrated intraretinally.[43] We have observed that HRF and hDC, along with RPD[37,39,43,44] (also known as subretinal drusenoid deposits on OCT) and a high central drusen volume, are important risk factors for progression to late AMD and atrophy.[45,46] In clinical practice, I use these OCT-based risk factors to risk stratify my patients, and identify patients who need more frequent follow-up.

Dr. Steen: Other important factors have been linked with a high rate of GA progression. The presence of multifocal lesions,[47] noncentral lesions,[48,49] and bilateral disease[48] all place a patient at risk for rapid progression. Patients with noncentral GA lesions are also at high risk for center involvement, with 57% of such patients experiencing lesions in the center within 4 years.[48]

Dr. Modi: Nomenclature surrounding GA and AMD has evolved over the past several years. Dr. Sadda and colleagues were integral in establishing the standard language used in today's practice.

Q | Dr. Sadda, could you describe the work of the Classification of Atrophy Meeting (CAM)?

Dr. Sadda: The CAM began their work by examining trends in imaging AMD. We focused our energy on SD-OCT, as it was a useful modality for this condition that was most accessible and widely used. After a literature review, we determined that there was no accepted definition of atrophy observed on OCT.

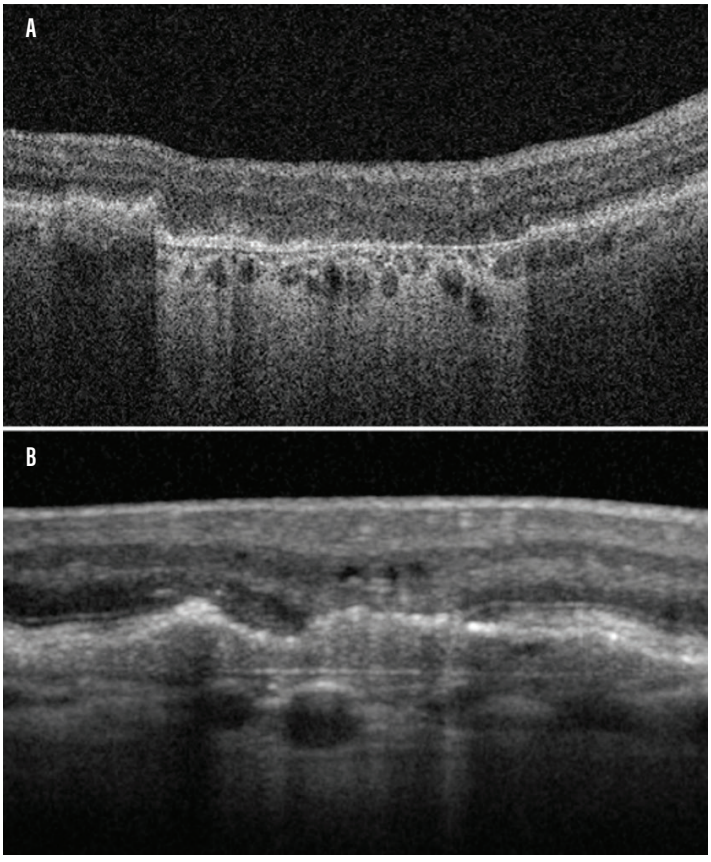

Figure 2. OCT images depicting cRORA (A) and iRORA (B). In figure A, four criteria are met: a region of hypertransmission at least 250 µm, a zone of attenuation or disruption of the RPE that is at least 250 µm, signs of overlying photoreceptor degeneration, and absence of an RPE tear.

There were a series of terms—sometimes slightly different from one another, sometimes synonymous—that were used to describe atrophy in AMD. The CAM standardized that language. For example, hypertransmission depicted on SD-OCT, which indicates increased signal penetration to the choroid, is sometimes called sub-RPE illumination. Others called that phenomenon hypertransmission defect to emphasize that the hypertransmission was due to a defect of the overlying RPE and photoreceptors. Regardless of what it is called, detecting this pattern on SD-OCT images allows clinicians to quickly identify AMD patients who may have already have atrophy or at high-risk to imminently develop atrophy.

Hypertransmission on SD-OCT can sometimes occur due to loss of pigment in the RPE rather than due to frank loss of cells. The CAM believed that a large enough overlying disturbance of the RPE and the photoreceptors would clearly indicate that a hypertransmission signal depicted atrophy. We determined in order to be measured reproducibly, that this area of hypertransmission and RPE defect would need to be approximately 250 µm in diameter, which is twice the size of the smallest measurement of a large drusen and was double the width of a vein near the

optic nerve (which could be used as an easy reference point during examination). To make the diagnosis of atrophy, overlying photoreceptor loss would need to be evident, and there could be no signs of an RPE tear. We termed anatomy that fit these four criteria would qualify as complete RPE and outer retinal atrophy, or cRORA[50] (Figure 2):

- a region of hypertransmission at least 250 µm
- a zone of attenuation or disruption of the RPE that is at least 250 µm
- signs of overlying photoreceptor degeneration
- absence of an RPE tear

Patients who do not quite fit those criteria but still demonstrate evidence of anatomic changes have, per the CAM, incomplete RPE and outer retinal atrophy, or iRORA.[51] Sometimes clinicians call this nascent GA.

Dr. Steen: Understanding the definitions and qualifications for cRORA and iRORA will be particularly important when a therapy is available for the treatment of GA. Depending on the specific indications for treatment, clinical criteria will need to be applied to determine which patients may be eligible for treatment. Educating the optometric community on these terms will be key to establishing proper and effective referral practices.

Dr. Ferrucci: There are many eye care providers who are unfamiliar with the useful nomenclature established by the CAM.

Q | Which biomarkers should a clinician first identify when trying to determine if a patient is presenting with iRORA or cRORA on SD-OCT?

Dr. Sadda: When you see bright areas on an en face OCT image that you identify as sub-RPE illumination, the next best step is to read OCT B-scans to determine if this is due to loss of photoreceptors and RPE. After that, estimate if the lesion is greater than 250 µm in diameter. After assessing the integrity of the RPE and ruling out an RPE tear, you can diagnose iRORA or cRORA.

Practically speaking, the areas of cRORA are absolute scotomas where the patient cannot see, and areas of iRORA will have some evidence of partial photoreceptor and RPE preservation. You may find that your diagnosis aligns with the patient's reported visual function.

Dr. Singh: In addition to classifying lesions as iRORA or cRORA, determining lesion location (ie, foveal or extrafoveal) will be of paramount importance if a drug that arrests or slows the development of GA lesions is approved. If we treat foveal lesions, we will be trying to prevent a scotoma from growing any larger than it already is, but our patients will remain with whatever vision loss they presented with. If we treat extrafoveal lesions, then we may be able to prevent or slow the progression of a lesion that would otherwise encroach on the fovea if left untreated. The more valuable proposition will be to treat extrafoveal lesions and prevent vision loss. This is especially true if phase 3 or postmarketing data show that treatments are more effective at slowing the growth of extrafoveal lesions compared with foveal lesions.

Still, regardless of the location of GA lesions, I anticipate that the optometrists and general ophthalmologists who refer to retina clinics will begin referring all GA patients for further evaluation. Doing so may shorten the period between a therapy coming to market and the patient receiving treatment.

Dr. Modi: Collaboration between optometrists and ophthalmologists will be foundational to success if a drug is approved for the treatment of GA. Although it may lead to crowded retina clinics, retina specialists will rely on our optometric colleagues to send patients with any evidence of GA to our clinics for evaluation and, if appropriate, treatment. The deeper the documented history and longitudinal imaging that optometrists can provide, the better we can determine rate of change of the GA and determine the best course of action.

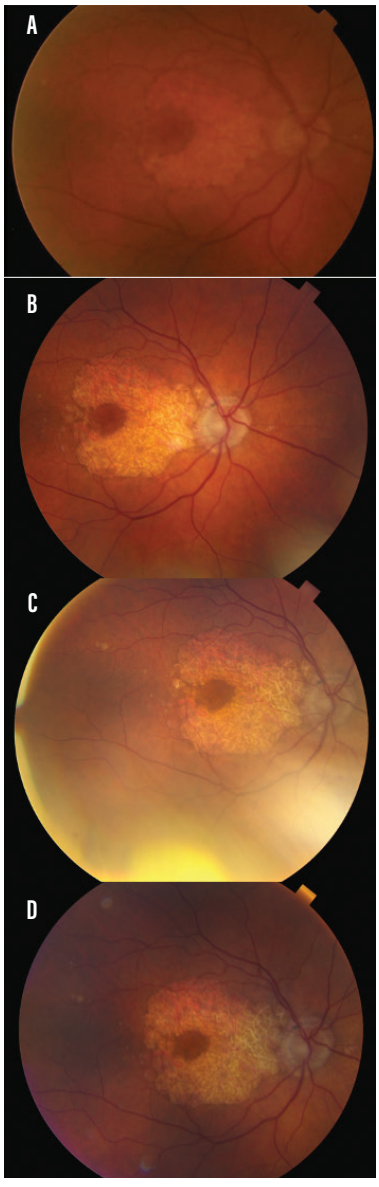

Figure 3. An 88-year-old man who presented to the clinic approximately 7 years ago has demonstrated progression of a GA lesion that spares the macula OD. The above images are from 2015 (A), 2018 (B), 2019 (C), and 2021 (D). BCVA was 20/25 OD at his most recent evaluation.

Dr. Ferrucci: Encouraging optometrists who currently manage GA patients to enroll patients in clinical trials for pipeline therapies would be an excellent way to elevate awareness around the current GA treatment paradigms. Optometrists who practice in collaborative settings have an advantage in this regard.

Dr. Sadda: We're hopeful that the treatments on the horizon, such as pegcetacoplan or avacincaptad pegol, will be approved by the FDA as soon as it is determined that it is safe and effective. In preparation for that possibility, it is incumbent upon all eye care providers to evolve our practice patterns regarding GA. For retina specialists, that will mean improving our communication

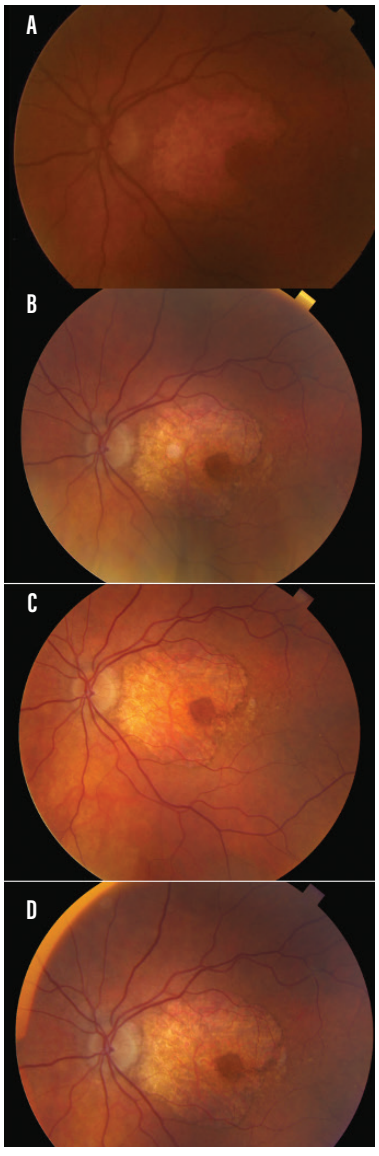

Figure 4. The patient's left eye also demonstrated growth of a macula-sparing GA lesion in 2015 (A), 2018 (B), 2019 (C), and 2021 (D). BCVA was 20/30 OS at his most recent evaluation.

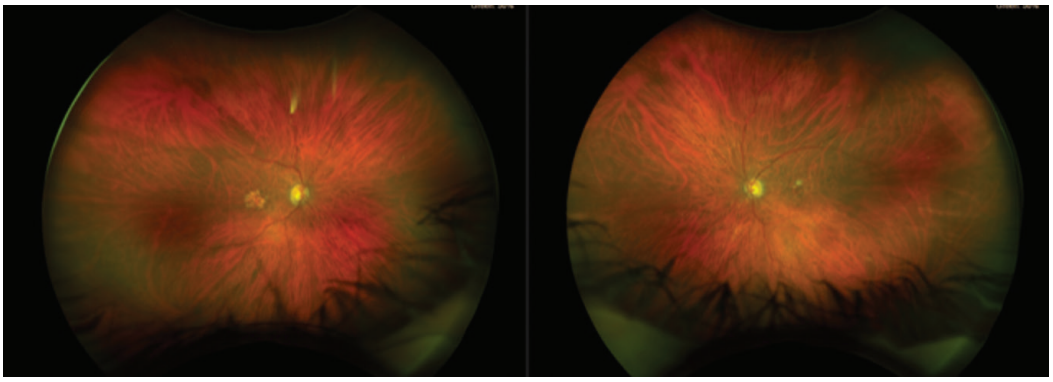

Figure 5. CFP shows evidence of foveal-involving GA OD and intermediate AMD OS. Due to the risk of progression OS, the prognosis for this patient is a likely conversion to bilateral GA.

with our optometric referral partners. Engaging with primary eye care providers now and explaining how the retina clinic's operations will change in the event of a drug approval will yield significant dividends for GA patients and retina clinics alike.

CASE 1: LESIONS SPARING THE MACULA IN A PATIENT DURING A 6-YEAR PERIOD

Dr. Ferrucci: An 88-year-old man presented to the clinic in 2015 with evidence of macula-sparing GA in his right eye (Figure 3A). His GA lesions grew significantly when he was imaged again in 2018 (Figure 3B) and 2019 (Figure 3C). In 2021, the patient's GA lesions continued to spare the macula despite continued growth (Figure 3D). Development of a macula-sparing GA lesion of similar size and shape was observed in his left eye at the same timepoints (Figure 4). His BCVA was 20/25 OD and 20/30 OS.

Dr. Sadda: Is this patient still driving or reading?

Dr. Ferrucci: This patient is unable to drive. He reports trouble reading, and says that he often loses his place. That tracks with the location of his lesions.

Dr. Steen: We often hear from patients that quality-of-life issues are affected by their deteriorating vision, and this case illustrates how quickly a lesion can grow. Unfortunately, I expect macular involvement somewhat soon in this patient.

Dr. Ferrucci: I agree that he is at high risk of losing significant visual function soon. I asked that this patient return for follow-up every 4 to 6 months so that I can continue to closely document his progression. When a treatment for GA is finally approved, he will be promptly referred to the retina service within my hospital system for evaluation and treatment.

CASE 2: UNILATERAL GA WITH CONTRALATERAL INTERMEDIATE AMD

Dr. Rodman: A patient presented to the clinic with complaints of visual disruption. Examination (Figure 5) revealed foveal-involving GA OD; drusen indicated the presence of intermediate AMD in the fellow eye, but evidence of GA was not observed. This case prompts us to think about the importance of fellow eye status in establishing a prognosis. Dr. Singh, how heavily do you weigh the anatomy of a fellow eye without GA when examining a patient with unilateral GA?

Dr. Singh: Bilateral GA places a patient at risk of rapid progression,[48] so if I see evidence of fellow eye involvement, I may further probe for evidence of both GA and wet AMD. In this instance, CFP has clearly illustrated a GA lesions OD. I would order OCT or FAF testing for this patient before definitively saying they do not have contralateral GA.

Dr. Modi: Screening the fellow eye for disease, even during injection-only visits for patients with wet AMD, is a best practice. The costs of not initiating timely therapy in an eye with new exudative disease is too high, and even a quick examination without imaging is preferred to nothing at all.

Dr. Sadda: Patients such as the one in this case could benefit when a drug is approved for the treatment of GA. The two drugs furthest along in the pipeline aim to slow the progression of GA rather than reverse it. Treatment for the right eye with GA could

be considered, but perhaps even more critically the left eye could be monitored carefully for conversion to GA and then be treated as soon as GA was detected—this may allow functional vision to be attained by this patient. Patients like this are perfect for referral to a retina specialist who can hopefully begin documenting this patient's disease progression and promptly treat them when a drug enters the market. ■

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GEOGRAPHIC ATROPHY: DIAGNOSIS, IMAGING, AND COLLABORATION

Release Date: June 30, 2022
CME Expiration Date: September 1, 2023
COPE Expiration Date: June 30, 2023

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DEMOGRAPHIC INFORMATION

Profession
___ MD/DO
___ OD
___ NP
___ Nurse/APN
___ PA
___ Other

Years in Practice
___ >20
___ 11-20
___ 6-10
___ 1-5
___ <1

Patients Seen Per Week
(with the disease targeted
in this educational activity)
___ 0
___ 1-15
___ 16-30
___ 31-50
____ >50

Region
___ Midwest
___ Northeast
___ Northwest
___ Southeast
___ Southwest

LEARNING OBJECTIVES

Did the program meet the following educational objectives?	**Agree**	**Neutral**	**Disagree**
Summarize the prevalence of AMD and GA and **define** the burden of illness linked specifically to GA	_____	_____	_____
Describe GA disease detection and factors influencing progression	_____	_____	_____

POSTTEST QUESTIONS

Please complete at the conclusion of the program.

1. Based on this activity, please rate your confidence in your ability to describe geographic atrophy (GA) disease detection and factors influencing progression (based on a scale of 1 to 5, with 1 being not at all confident and 5 being extremely confident).

A. 1
B. 2
C. 3
D. 4
E. 5

2. What was the global prevalence of age-related macular degeneration (AMD) in 2016?

A. ~130 million patients
B. ~170 million patients
C. ~230 million patients
D. ~270 million patients

3. A 66-year-old African-American man presents to your office for routine eye examination. He has a family history of AMD. His social history is positive for tobacco and alcohol use. He has a history of diabetes, hypertension, and obesity. All of the following are risk factors for AMD and GA in this patient EXCEPT:

A. Family history of AMD
B. African-American race
C. Smoking history
D. History of obesity

4. All of the following are good tests to assess GA progression, EXCEPT:

A. Contrast sensitivity measurement
B. Dark adaptation
C. Reading speed
D. Best corrected visual acuity

5. A 77-year-old patient with a history of AMD and GA in his right eye presents to your office for evaluation. He notes that his vision seems to have gotten progressively worse over time, however on examination today his visual acuity measurement is stable from a year prior. Which of the following tests might be more useful in determining this patient's current visual function and predict potential future vision loss?

A. Low luminance visual acuity
B. Fluorescein angiography of the right eye
C. Corneal pachymetry
D. Corneal hysteresis

6. A 69-year-old woman with a history of AMD and GA in both eyes presents to your office for evaluation. Of the following, what imaging modality would be the best to quantify and track her GA, as well as the status of her AMD?

A. Color fundus photography
B. Spectral domain optical coherence tomography (SD-OCT)
C. B-scan ultrasonography
D. Corneal topography

7. A 75-year-old new patient presents to your clinic for evaluation. She has a history of bilateral visual loss. On examination, you note bilateral presence of hypopigmented deposits throughout the macula. Your differential diagnosis for this patient includes both AMD and Stargardt disease. What imaging modality might help you differentiate between these two disease entities?

A. SD-OCT
B. B-scan ultrasonography
C. Color fundus photography
D. Fundus autofluorescence

8. Which of the following fundus autofluorescence GA patterns is indicative of higher likelihood of progression?

A. Small lesion
B. Unifocal lesion
C. Diffuse-trickling pattern lesions
D. Banded lesions

9. A 75-year-old Caucasian man presents to your office for evaluation. On exam, you note bilateral medium-sized drusen and some GA. You obtain an SD-OCT that shows evidence of drusen as well as subretinal drusenoid deposits. You also note bilateral cataract. Which of the following features of this patient's exam predict highest risk for GA progression?

A. Medium-sized drusen
B. Geographic atrophy
C. Subretinal drusenoid deposits
D. Cataracts

10. A 98-year-old patient with AMD and GA presents to your office for evaluation. On exam, you note bilateral small drusen in both eyes. On SD-OCT, you note hyporeflective drusen cores, intraretinal hyperreflective foci, subretinal drusenoid deposits, and a small extrafoveal pigment epithelial detachment. All of the following put this patient at increased risk for progression to late AMD and atrophy, EXCEPT:

A. Hyporeflective drusen cores
B. Intraretinal hyperreflective foci
C. Subretinal drusenoid deposits
D. Pigment epithelial detachment

11. Which of the following factors have been linked with a high rate of GA progression?

A. Unifocal lesions
B. Unilateral disease
C. Multifocal lesions
D. Presence of cataracts

ACTIVITY EVALUATION

Your responses to the questions below will help us evaluate this activity. They will provide us with evidence that improvements were made in patient care as a result of this activity.

Rate your knowledge/skill level prior to participating in this course: 5 = High, 1 = Low_____

Rate your knowledge/skill level after participating in this course: 5 = High, 1 = Low_____

This activity improved my competence in managing patients with this disease/condition/symptom. _____ Yes _____No

Probability of changing practice behavior based on this activity: _____High _____ Low _____No change needed

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

Change in pharmaceutical therapy _____
Change in nonpharmaceutical therapy _____
Change in diagnostic testing _____
Choice of treatment/management approach _____
Change in current practice for referral _____
Change in differential diagnosis _____
My practice has been reinforced _____
I do not plan to implement any new changes in practice _____

Please identify any barriers to change (check all that apply):

_____ Cost
_____ Lack of consensus or professional guidelines
_____ Lack of administrative support
_____ Lack of experience
_____ Lack of time to assess/counsel patients
_____ Lack of opportunity (patients)
_____ Reimbursement/insurance issues
_____ Lack of resources (equipment)
_____ Patient compliance issues
_____ No barriers
_____ Other. Please specify: _____

The design of the program was effective for the content conveyed	___ Yes	___ No
The content supported the identified learning objectives	___ Yes	___ No
The content was free of commercial bias	___ Yes	___ No
The content was relative to your practice	___ Yes	___ No
The faculty was effective	___ Yes	___ No
You were satisfied overall with the activity	___ Yes	___ No
You would recommend this program to your colleagues	___ Yes	___ No

Please check the Core Competencies (as defined by the Accreditation Council for Graduate Medical Education) that were enhanced through your participation in this activity:

_____ Patient Care
_____ Practice-Based Learning and Improvement
_____ Professionalism
_____ Medical Knowledge
_____ Interpersonal and Communication Skills
_____ System-Based Practice

Additional comments:

_____

_____ I certify that I have participated in this entire activity.

This information will help evaluate this activity; may we contact you by email in 3 months to inquire if you have made changes to your practice based on this activity? If so, please provide your email address below.

_____

_____

MODERNOPTOMETRY