SHARPEN YOUR AMD DIAGNOSTIC SKILLS

Know how to spot early AMD, and have a plan of action.

BY DAMON DIERKER, OD, FAAO

Age-related macular degeneration (AMD) is the leading cause of adult blindness in the Western world.[1] An estimated 14 million Americans have AMD,[2] and this number is projected to sharply increase in the coming decades due to the aging baby boomer population.[1] Although there is no cure for AMD, early detection, patient education, and the use of risk reduction strategies, as well as early intervention with anti-VEGF drugs once neovascular disease is confirmed, can lead to better outcomes.

A 2017 study found that optometrists and ophthalmologists miss signs of early to moderate AMD at least 25% of the time,[3] indicating an unmet need in clinician education. This article provides a brief overview of signs and symptoms to be on the lookout for, as well as what to do when you detect them.

RISK FACTORS AND EARLY SYMPTOMS

Age and family history are the most important risk factors for AMD. Approximately one of three people older than 75 years has signs of AMD.[2] And individuals who have a parent or sibling with AMD have an approximately threefold increased risk of developing the disease.[4] Among modifiable risk factors, smoking is the most critical. A history of smoking increases the risk of AMD development and progression, the odds increasing with the amount an individual has smoked.[5] Other risk factors include hypertension, cardiovascular disease, obesity, and low macular pigment density.[6,7]

We think of classic AMD symptoms as metamorphopsia and central vision

AMD CLASSIFICATION

EARLY AMD: medium-sized (63-125 µm) drusen and no AMD pigment abnormalities

INTERMEDIATE AMD: any large drusen (>125 µm in size) and/or any AMD pigment abnormalities

ADVANCED AMD: any geographic atrophy or neovascular disease

loss, but these are actually symptoms of *advanced disease*. In early AMD, the most common symptom is poor night vision.[8] Even in early stages, AMD causes profound defects in rod-mediated dark adaptation.[9] Although night vision problems can be related to normal aging and cataracts, be suspicious of AMD in patients who have poor night vision not explained by clinical examination.

MAKING THE DIAGNOSIS

All patients with AMD need a plan of action. We can provide the best advice to patients with AMD if we stratify them into categories based on structure and function.

Fundus photography remains the gold standard for diagnosing and staging nonexudative AMD. In 2013, the Beckman Initiative for Macular Research published a classification system to assist eye care providers in identifying AMD-related structural changes (see *AMD Classification*).[10]

OCT is also valuable in identifying AMD at early stages. Drusen, a hallmark of AMD, are identified on OCT as discrete elevations of the retinal pigment epithelium (RPE) layer at the level of Bruch membrane (Figure). Drusen that form above the RPE are known as reticular pseudodrusen and are a more ominous sign. Patients with this type of drusen are much more likely to progress to advanced disease.[11]

Until recently, we have not had an adequate tool for measuring AMD function in early disease. VA is preserved until advanced stages of AMD, and contrast sensitivity and color vision testing have limited diagnostic accuracy.[12] As the disease progresses, dark adaptation can worsen, even though fundus findings and acuity may remain unchanged.[13] Researchers have shown that impaired dark adaptation precedes the development of visible drusen by at least 3 years.[14] Dark adaptation testing (AdaptDx, MacuLogix) is a tool we can use to detect AMD *before* it can be seen clinically.

A new diagnosis, *subclinical* AMD, has been proposed, and I believe that identifying patients at this stage can improve outcomes further. The AdaptDx rapid diagnostic test can be used to confirm AMD in just a few minutes with 90% sensitivity and specificity.[15] An extended test protocol allows practitioners to monitor patients for disease progression, akin to monitoring glaucoma with visual field testing.

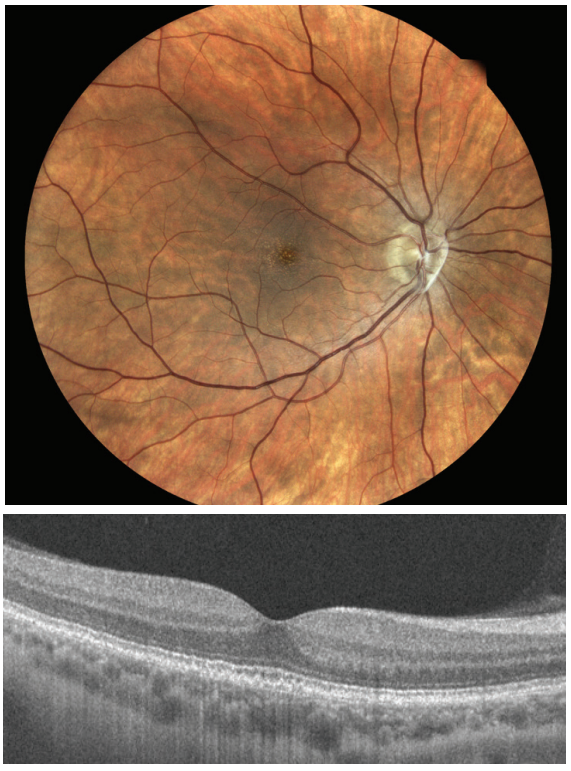

Figure. OCT findings show numerous medium-sized drusen (>63 µm to ≤125 µm) in a patient with early AMD. No pigmentary abnormalities are present.

PLAN OF ACTION

Once we have identified a patient with AMD, we need to educate that patient appropriately and provide an evidence-based plan for reducing the risk of progression.

Early and Subclinical AMD

First, discuss your findings with the patient in direct, easy-to-understand terms. No one wants to hear that he or she has AMD, but patients will be grateful when they understand that their prognosis is better with early detection.

Address smoking as appropriate. I generally refer patients to their primary care physicians for specific smoking cessation strategies. It's also important to discuss lifestyle management, including weight control and regular exercise. Emphasize the importance of diet and nutrition. I review the need to incorporate plenty of fruits and vegetables into the diet every day. Green leafy vegetables (eg, spinach, kale, collard greens) are especially important.

I encourage omega-3 fatty acid supplements if patients are not eating several servings of healthy fish (salmon, tuna, etc.) on a weekly basis. An eye-specific supplement containing the macular carotenoids (lutein, zeaxanthin, and meso-zeaxanthin) should be considered for everyone. There are no known contraindications to carotenoid formulations. These supplements have antioxidant and anti-inflammatory properties, and patients with early AMD can actually improve their contrast sensitivity and visual performance with sustained use.[16]

Don't forget to provide patients with written instructions detailing your recommendations and emphasize the need to contact your office urgently if they detect any vision changes. Schedule a follow-up visit in 6 to 12 months to monitor for progression. I typically rely on dark adaptation testing, OCT, and fundus photography as indicated for following patients with early disease.

AT A GLANCE

- What we think of as classic AMD symptoms, metamorphopsia and central vision loss, are actually symptoms of *advanced* disease.
- Suspect AMD in patients who have poor night vision not explained by clinical examination.
- Drusen, a hallmark of AMD, are identified on OCT as discrete elevations of the retinal pigment epithelium layer at the level of Bruch membrane.
- Impaired dark adaptation precedes the development of visible drusen by at least 3 years.

Intermediate AMD

All recommendations above for early and subclinical disease also apply to patients with intermediate AMD. A few important additions should be considered. For one, monitor patients more frequently, generally every 4 to 6 months. Also obtain OCT (and OCT angiography, if this modality is available) at every visit, regardless of changes in symptoms or VA. Patients with intermediate AMD have up to a 50% risk of progressing to advanced disease over the next 5 years.[17]

Consider recommending an AREDS2-based supplement, but be cautious with the amount of zinc it contains. A large number of products using the AREDS2 name are available, and the amount of zinc they contain varies widely. Although this topic is controversial, I order genetic testing (Macula Risk, ArcticDx) for patients with intermediate AMD to determine whether zinc is appropriate. An alternative approach is to consider a supplement containing 25 mg of zinc instead of 80 mg, as AREDS2 showed no significant additional risk reduction in patients taking the higher dose.[18]

Consider prescribing a home monitoring system (ForeseeHome, Notal Vision). It takes our patients roughly 3 minutes per eye to perform, and possible disease progression triggers a ForeseeHome alert to my office so we can bring the patient in for reevaluation. In the HOME study by the AREDS2 Group, patients using ForeseeHome had a better chance of having good VA at the time of progression to neovascular AMD compared to those using an Amsler grid.[19]

Advanced Disease

Patients with geographic atrophy should be monitored in a similar manner to those with intermediate disease. There is no FDA-approved treatment for geographic atrophy at this time.

Any suspicion of neovascular disease necessitates referral to an ophthalmologist, preferably a retina specialist, within a few days to a week for consideration of treatment with anti-VEGF therapy.

ODs CAN HELP PREVENT BLINDNESS

Be on the lookout for AMD in all of your patients over age 50 years, particularly if they complain of poor night vision. Order appropriate tests to aid in making a confident diagnosis. Have a direct conversation with your patients and develop a plan of action to slow down the disease. Early detection can help reduce the risk of blindness for millions of Americans with AMD. ■

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