EYEING UP EVAPORATION: GETTING AT THE HEART OF DRY EYE DISEASE


CECELIA KOETTING, OD, FAAO, DIPABO, AND MILE BRUJIC, OD, FAAO

Dry eye disease (DED) is a disorder affecting an estimated 38 million people in the United States.[1] Despite its commonality, the majority of cases are undiagnosed. Current data indicate approximately 18 million people are diagnosed with DED,[1] and of those, only 1.2 million are treated with prescription medication.[2] DED can cause fluctuations in vision, pain, and discomfort, and may lead to significant patient impairment impacting everything from mood to work and leisure activities. In severe cases requiring surgery, its disabling effects are comparable with that of hospital dialysis and severe angina.[3] Cecelia Koetting, OD, FAAO, DipABO, and Mile Brujic, OD, FAAO, hosted a recent webinar discussing approaches to identifying and diagnosing DED.

APPROACHES TO DED DIAGNOSIS

There is a huge gap between the number of patients diagnosed with DED and the number of patients who receive treatment for it.[1,2] Fortunately, there are several methods and tools to help in the diagnosis of DED that are easily implemented and do not require an investment in costly equipment. "I take a two-fold approach toward diagnosis that assimilates subjective information with what I see objectively. Then, I make the best clinical judgement on what is contributing the most to the disease and map a path forward," said Dr. Brujic.

Validated questionnaires, such as the Ocular Surface Disease Index (OSDI)[4] and the Standard Patient Evaluation of Eye Dryness (SPEED),[5] can provide subjective information, particularly for symptomatic patients. Simple and quick verbal questionnaires may also be used. "I have tried both the OSDI and SPEED questionnaires, and I have developed a hybrid questionnaire. All new patients complete it with their intake forms. It gets patients to think about things that they otherwise would not have mentioned and has helped me identify many DED patients," said Dr. Koetting.

In addition to these subjective tests, multiple objective tests for DED are available to clinicians. "My patients get a fluorescein and/or lissamine green strip, which will stain the ocular surface and, less obviously, the conjunctiva of DED patients," said Dr. Brujic (Figure 1). "If you look through a Wratten #12 yellow filter, it will really help the fluorescein staining to jump out. Additionally, we can use tear osmolarity tests to objectively measure tear film homeostasis and MMP-9 tests to look for inflammation."

EVALUATING MEIBOMIAN GLAND FUNCTION

Evaporative tear loss, which often overlaps with aqueous deficiency, is the underlying etiology in nearly 90% of DED cases.[7] Meibomian gland dysfunction (MGD) is a principal contributor to evaporative DED.[8] Without adequate or quality meibum, the aqueous-protecting lipid layer is compromised, which leads to a cycle of rapid tear film disruption and evaporation.[9]

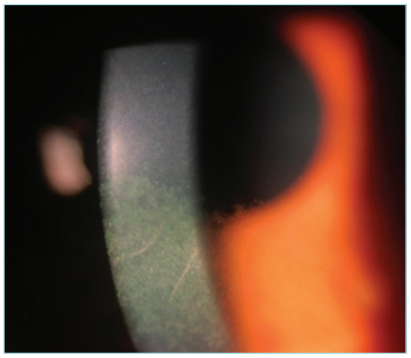

Image courtesy of Christopher Starr, MD

Figure 1. Lissamine green staining indicative of DED.

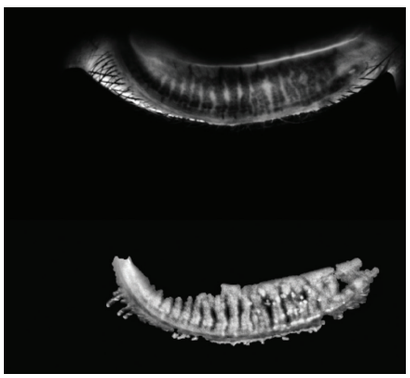

Image courtesy of Cecilia Koetting, OD, FAAO, DIPAO

Figure 2. This patient's meibography of the lower lid shows mild atrophy with mild overfilling of the glands and loss of gland structure.

Meibography technology can help identify patients with meibomian gland atrophy, damage, or inspissation (Figure 2). The technology uses black-and-white infrared images to determine a meiboscore between zero and 3. The higher the score, the more gland loss and architectural changes are present.[8] Although meibography is a powerful diagnostic

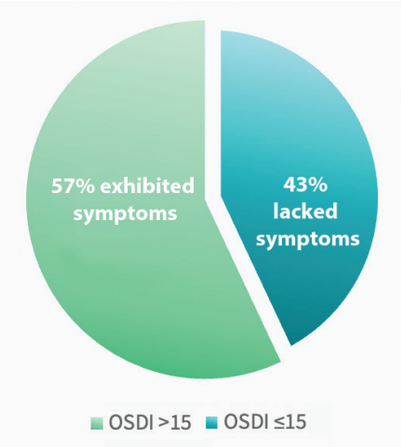

Figure 3. Clinical signs and symptoms of DED do not always correlate, making diagnosis difficult. In a retrospective analysis of 11 sites across the United States and European Union, Sullivan et al[10] found that, of 263 patients who showed clinical signs of DED, only 57% (green wedge) also had an OSDI score of > 15 and reported symptoms that were consistent with a DED diagnosis.

tool, there are additional methods we can employ to identify MGD.

Assessing meibomian gland function is vital in determining DED etiology and developing the best treatment path. This can be done easily with just a slit lamp. "We cannot assume that if meibomian gland structure is good that the function is good. Look, lift, push, and pull. Have your patient look down and examine the eyelids, the lash margins, and the biofilm for signs of demodex, blepharitis, and any other abnormalities. Check lid tonicity by pulling the lower and the upper lids away from the eye to see how elastic they are. Use a cotton swab, your finger, or a spatula device to push on the glands to grade how easily the meibum comes out, as well as the quality of it. I like to take pictures with my phone to show my patients what is going on, and some doctors even have cameras mounted on their slit lamps. If we can catch clogging and dysfunction early, we can avoid atrophy later," said Dr. Koetting.

There is often a mismatch in symptom severity versus the clinical assessment of DED[10] (Figure 3), which can make diagnosis challenging. Some of the reasons for this mismatch may be: (1) DED is multifactorial; (2) it can be caused or exacerbated by a wide range of ocular surface diseases; and (3) the pathophysiology is individual and therefore does not follow a consistent course.

Likewise, there are many factors that can contribute to discordance between signs and symptoms. "A large cohort study conducted with 648 patients in the Netherlands[11] sought to identify predictors of discordance between symptoms and signs and found that several factors, such as chronic pain, atopic disease, and depression, are associated with more severe symptoms than would be suggested by clinical signs. In particular, patients with lower self-perceived overall health report more severe symptoms versus signs," said Dr. Brujic.

ADDRESSING EVAPORATION: THE FINAL COMMON PATHWAY IN DED

Although there are multiple options to treat aqueous deficiency and the inflammatory components of DED, as of this writing, there are currently no prescription pharmaceutical eye drops that directly target the evaporative component. Some existing prescription pharmaceutical products aimed at the inflammatory cycle in DED can be associated with high rates of dissatisfaction and discontinuation.[12] Preservative-free artificial tears are a first-line therapy; however, residence time is short, and drops with high viscosity may cause blurred vision and reduced contrast sensitivity.[13] Device-based treatments such as the Systane iLux2 (Alcon), LipiFlow (Johnson & Johnson), TearCare (SightSciences, Inc.), and intense pulsed light (IPL), as well as interventional home therapies, including nutraceuticals, lid cleansers, and warm compresses, can effectively help to disrupt the cycle of DED. "We want to keep it simple and specific, because we know that the more we give a patient to do, the less likely they are to be compliant. Until we have medication that can treat the root cause of evaporative DED, we need to work with the tools we currently have available," said Dr. Koetting. ■

"WE CANNOT ASSUME THAT IF MEIBOMIAN GLAND STRUCTURE IS GOOD THAT THE FUNCTION IS GOOD." —DR. KOETTING

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