Discovering the Root Cause of Dry Eye Symptoms

In-office technologies aid clinicians' diagnosis and treatment of DED.

BY HOWARD BARNEBEY, MD

Many conditions have been lumped together as dry eye disease (DED), including dysfunctional tear syndrome, ocular surface disease (OSD), and meibomian gland dysfunction. Some of these terms suggest the etiology of DED, whereas others imply that multiple factors contribute to patients' symptoms. Only 12% to 15% of patients exhibit true DED, meaning tear production is inadequate.[1]

Concomitant diseases may also contribute to DED symptoms. Ophthalmologists are just beginning to understand the relationship between OSD and glaucoma. For example, glaucoma treatments may worsen OSD, but this theory has not received adequate attention. On the contrary, outcomes of refractive procedures such as LASIK, cataract surgery, or IOL implantation to give patients better vision and quality of life, depend on the health of the ocular surface, so studies advise ophthalmologists to examine and treat the ocular surface prior to rather than after refractive surgical procedures. Although most ophthalmologists focus on "the big three"—retinal disease, diabetes, and macular degeneration—there are many patients who do not fall into those categories but who experience severe discomfort from OSD. Until recently, the necessary tools to address DED

Have you experienced these symptoms last week?	All the time	Most of the time	Half of the time	Sometimes	Not at all
Eyes feel burning	4	3	2	1	0
Eyes feel dry	4	3	2	1	0
Eyes scratchy/gritty	4	3	2	1	0
Eyes feel sore	4	3	2	1	0
Eyes tear	4	3	2	1	0

How severe are your symptoms?	Miserable	Bothersome	Uncomfortable	Mild	Not bothersome
Burning	4	3	2	1	0
Dry	4	3	2	1	0
Scratchy/gritty	4	3	2	1	0
Sore	4	3	2	1	0
Tearing	4	3	2	1	0

Figure. Dry eye questionnaire.

> "Most OSD therapy centers on the functionality of the meibomian glands, so this is where the process begins."

patients' needs were unavailable. With the development of new diagnostic technologies, ophthalmologists can now determine the root cause of DED symptoms and tailor treatment plans accordingly.

EVALUATING SIGNS AND SYMPTOMS

Two factors prompt my DED workup. First, I use modified versions of the Ocular Surface Disease Index (OSDI) questionnaire and the Standard Patient Evaluation of Eye Dryness (SPEED) questionnaire to determine patients' symptomatology (Figure). Second, I perform a routine slit-lamp examination. I always check the eyelids and the tear film for staining of the cornea or conjunctival irritation. If either of the questionnaires or the slit-lamp examination reveals an abnormality, I initiate a DED evaluation.

I divide dry eye evaluations into three categories: expedient, intermediate, and comprehensive examinations. In light of the many potential causes of DED symptoms, I choose to perform a comprehensive evaluation. First, I image the eye with an ocular surface interferometer (LipiView; TearScience), a device that does not disrupt the natural homeostasis of tears in the eyelids. It measures the thickness of the oil layer in the tear film in nanometers, and it measures the ratio of partial to complete blinks and the quality of the blink. Armed with this information, I educate the patient on the results of his or her test.

Next, I perform meibography by visualizing the glands with an ocular coherence tomographer (Spectralis OCT; Heidelberg Engineering) to evaluate the meibomian glands' structure. After the image is taken, the patient is directed to the examination room where I review his or her medications, complaints, and vision. I then evaluate tear breakup time by placing 25 to 30 mL of fluorescein on the tear film. I instill a significant amount of additional fluorescein in the eye to test for staining of the cornea as well as lissamine green to test for staining of the conjunctiva. While waiting for the eye to absorb the liquid, I evaluate meibomian gland secretions using a handheld instrument (Meibomian Gland Evaluator; TearScience) that simulates the pressure on the eyelid gland that is comparable to a blink. I can visualize the secretions and determine whether they are clear, turbid, or nonexistent. I have also begun using an in-office test (InflammaDry; Rapid Pathogen Screening) that detects matrix metalloproteinase 9 in tears. This inflammatory marker is consistently elevated in DED.[2] I look for neovascularization and examine the conjunctival insertion along the posterior aspect of the eyelid to determine if it is linear or irregular.

I then remove all of the liquid instilled in the patient's eye and conduct a Schirmer test. Despite its reputation for inconsistent and variable results, in my experience, if it is performed with anesthesia and in less than 5 minutes, a Schirmer test can be a solid indicator of whether or not the tear glands produce enough tears to keep the eye adequately moist.

ESTABLISHING A TREATMENT PLAN

If the ocular surface interferometer determines that a patient's lipid layer is thin and the meibomian gland image shows signs of gland dropout, then I recommend thermal pulsation treatment (LipiFlow; TearScience) to open up the glands and restore the lipid layer of the tear film.

If the patient does not have a complete blink, I recommend blinking exercises, which provide a tremendous amount of symptom relief. Research has shown that blink exercises are important to help with reducing evaporating rates.[3] In my experience, however, patients often have difficulty adhering to these exercises, so I review their current symptoms with my examination findings and discuss the importance of persistence with the dry eye program and the long-term goal of comfort and ocular health. For those individuals who do not make many tears, I recommend cyclosporine ophthalmic emulsion 0.05% (Restasis; Allergan) to restore the aqueous layer and loteprednol etabonate ophthalmic suspension 0.5% (Lotemax; Bausch + Lomb) to reduce inflammation.

CONCLUSION

Ophthalmologists now have some tools in their armamentarium that allow for more accurate diagnoses to tailor treatment options to the specific causes of DED. Most OSD therapy centers on the functionality of the meibomian glands, so this is where the process begins. ■

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