Dry eye is not just an irritating condition; it is a disease of the ocular surface—this is an important lesson for clinicians to remember. Dry eye disease (DED) is a multifactorial, progressive, chronic disease that results in discomfort, visual disturbance, and tear film instability with potential damage to the ocular surface, accompanied by increased tear film osmolarity. DED causes considerable morbidity and has a significant economic impact in both direct and indirect costs. Its effect on patients can range from mild and episodic to severe and chronic. Symptoms can include blurred and fluctuating vision, eye discomfort, irritation, foreign body sensation, ocular surface inflammation, redness, excess tearing, and photosensitivity.

Given the potential effects of DED, it is important to assess patients with complaints of dry eye in a systematic and thorough manner (see Steps in Understanding the Condition of a Patient With Complaints of Dry Eye). This article describes my approach to assessing signs and symptoms of DED. The scope of this article is limited to DED encountered in everyday practice; the management of surgery-induced DED-like symptoms is addressed in subsequent articles in this cover focus.

ASSESSING SYMPTOMS

The first step in understanding the condition of a patient with complaints of dry eye is assessing his or her symptoms. I ask patients about general symptoms such as burning or foreign body sensation, and whether they have noticed a rapid rate of eye closure. Another important question is whether the symptoms are more prominent in the morning or during the day. When symptoms are worse in the morning, my diagnosis might center more on blepharitis and meibomian gland disease (MGD); alternatively, if they increase during the day, this points toward tear-deficient DED. Sometimes, however, the situation is not so clear, such as when the patient says that symptoms persist throughout the day, which may mean that both conditions—blepharitis and tear-deficient DED—coexist.

As I mentioned at the outset, we always have to keep in mind that a dry eye is a disease of the ocular surface. Once I understand the patient’s symptoms, I think about how they fit into the picture of ocular surface disease. Does the patient have DED, and, if so, what is the makeup of the disease: Is it due to evaporative causes, tear production inadequacy, inflammation, or some combination of these factors? Information such as this allows us to begin to individualize patient care.

ASSESSING SIGNS

Sometimes the diagnosis of the problem becomes clear simply by looking at the patient’s face. In patients with acne rosacea, for instance, most of the diagnostic work is already done. (Ocular rosacea is often associated with blepharitis.) This is also the time to assess the blink rate and whether the...
Blink is complete or not. These can be important clues to the causes of dry eye.

After taking a good look at the patient’s face, I move to the slit lamp to scan the ocular surface. I start by looking closely at the lid margins, checking for changes that could indicate MGD by gently pressing underneath the lid margin to observe the secretions of the meibomian glands. I also look for signs of inflammation on the lids. The appearance of squamous metaplasia at the lid margin is a marker of a chronic inflammation and one sign of evaporative DED.

Once I have carefully inspected the lid margins, I move to the conjunctiva. Conjunctival hyperemia is one of the best markers to assess inflammation of the ocular surface. Unfortunately, there is no easy way to objectively quantify inflammation. With access to the proper resources, impression cytology and flow cytometry can be used, but this requires specialized laboratory equipment not available in everyday practice. Nonetheless, a subjective impression of the level of conjunctival hyperemia can be an important clue to the presence of inflammation and the nature of an individual’s DED.

It is also important to look for conjunctivochalasis, which can induce chronic inflammation of the ocular surface and affect the distribution of the tear film. Conjunctivochalasis can be responsible for the symptoms that patients complain of—burning, itching, and foreign body sensation—and is therefore another important marker of inflammation.

Next I look at the cornea, focusing on the tear film. The mucin layer of the tear film is thin, but it is important in controlling evaporation of the aqueous. A healthy mucin layer is therefore an indication of equilibrium in the tear film. If the mucin is absent, something is wrong. Filaments of mucin are a biomarker for evaporative DED. The tear meniscus height should also be evaluated to gauge the thickness of the tear film.

Following these inspections, the next stage of the slit-lamp examination is to use vital stains such as fluorescein and lissamine green to check the tear film. Fluorescein is helpful to assess tear breakup time and the stability of the tear film; the distribution of fluorescein staining gives an indication of areas where the tear film is too thin or is incomplete (Figure 1).

Lissamine green staining is valuable because it allows us to see damage at the levels of the cornea and the conjunctiva. In some patients, fluorescein staining shows no damage at the level of the cornea, but symptoms persist. In these cases, lissamine green can often identify damage to the conjunctiva and aid in the diagnosis of dry eye (Figure 2).

I recently presented a paper describing the relationship between inflammation and ophthalmic damage in patients with DED. In our research, we showed that lissamine green staining correlated with the infiltration of inflammatory cells, in particular CD14+ cells, in the conjunctival epithelium, thus providing evidence of the presence of inflammatory factors in the conjunctiva. Lissamine green staining does not take a lot of extra time, but it can provide some important clues for the diagnosis of DED.

OTHER CONSIDERATIONS

The Schirmer test, though widely used, can only be an indicator of the quantity of tears; it cannot be used to make a definitive diagnosis of dry eye. Although it is important to know the quantity of tears the patient is able to produce, this is not the only information needed to make a diagnosis of dry eye. Rather, all the components of the ocular surface must be considered before forming a diagnosis.

Recently introduced so-called objective tests for dry eye, such as the LipiView Ocular Surface Interferometer (TearScience) and the Korb Meibomian Gland Evaluator (TearScience), may provide data for research purposes; however, in my opinion, their usefulness in daily practice is yet to
be seen. Until we know more about the reproducibility of their data, I would not base a diagnosis on their output. (Editor’s Note: For a different viewpoint on objective testing, see Subjective Objectivity, by Walter O. Whitley, OD, MBA, on page 50.)

CONCLUSION

To conclude, I return to the statement I began with: Dry eye is a disease of the ocular surface. When you see a patient with symptoms of dry eye, keep in mind that you are dealing with the whole ocular surface. Look at the lid margins, the cornea, the conjunctiva, the epithelium, and the tear film, and consider them as a functional unit.

As an example, consider a patient with an inferior corneal ulcer: Most ophthalmologists might conceptualize this as a condition of the cornea and approach treatment that way. However, an inferior corneal ulcer is not just a problem with the cornea, it is a problem with the ocular surface (and probably related to pathology in the interior of the lid margin).

Even more common, consider a patient with DED and corneal damage due to blepharitis and MGD, where the problem is not only on the cornea but rather with the entire ocular surface functional unit. If you treat this patient with artificial tears alone, it does not correctly address the underlying issue. You must look at the lid margin for signs of MGD and inflammation and consider whether the inflammation is responsible for the corneal damage. To effectively treat this patient, lid scrubs, antibiotics, and an antiinflammatory treatment such as a topical steroid or cyclosporine may be needed.


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