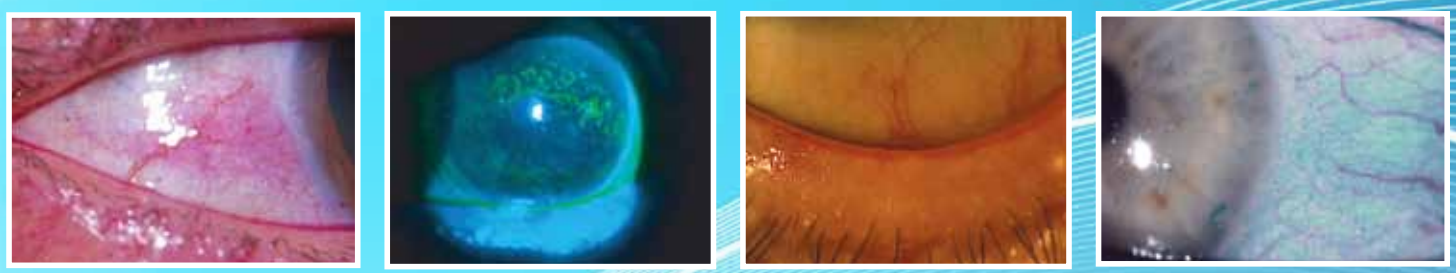
Supplement to
Advanced
OCULAR CARE
CME ACTIVITY
March/April 2012

DIAGNOSIS AND TREATMENT

OF DRY EYE DISEASE AND OCULAR ALLERGY



IMPLEMENTING TREATMENT STRATEGIES TO PROTECT THE OCULAR SURFACE

Cases Highlighting Key Management Pearls

- Cataract Surgery, Sjögren Syndrome
- Perimenopausal Woman With DED
- Contact Lens Intolerance, Stargardt Disease, and Severe OSD

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Diagnosis, Treatment of Dry Eye Disease and Ocular Allergy

Jointly sponsored by the Dulaney Foundation and *Advanced Ocular Care*.

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STATEMENT OF NEED

The incidence of dry eye syndrome was previously estimated to be as high as 14.4%.[1,2] However, new research is showing that it may be even higher.[3] In a prospective, multicenter study of 143 patients undergoing cataract surgery screened for dry eye, the results showed that while 21% of patients had been previously diagnosed with dry eye, more than 40% reported symptoms of dry eye disease.

Additional studies by Sanjay Rao, MD, show that dry eye is not the stable condition it was previously thought to be. Even mild to moderate cases showed significant progression over 12 months when not treated effectively.[4]

Despite the high prevalence of this disorder, many patients are not property diagnosed and treated.[5] One reason is the lack of consensus in defining dry eye and the necessity of a combination of comprehensive patient histories and diagnostic tests to confirm or rule out the condition.[1] Changing population demographics and lifestyle factors indicate that the dry eye syndrome patient population will increase significantly, ensuring that ophthalmologists and comanaging practitioners will see more patients presenting with dry eye symptoms.[5].

One of the challenges involved in recognizing and managing dry eye is that the condition is closely related to ocular allergies, and the two are often confused. Symptoms of dry eye and allergies, including itching, discharge and irritation, overlap extensively.[6] Up to 40% of the population had experienced ocular symptoms at least once in their lifetimes.[7]

The prevalence of dry eye or ocular allergy necessarily translates to a large and growing percentage of cataract and refractive patients who are suffering from ocular surface conditions. This is of particular concern as dry eye affects keratometry readings before cataract surgery, which can lead to placing a toric IOL on the wrong axis, and it can also influence placement of limbal relaxing incisions.[3]

To help patients with dry eye and ocular allergies and optimize surgical outcomes, ophthalmologists and optometrists must comanage care effectively and be well versed on the latest diagnostic techniques and treatment protocols.

1. Preferred Practice Pattern, Dry Eye Syndrome, San Francisco, CA: American Academy of Ophthalmology; September, 2008.
2. Schein OD, Munoz B, Tielsch JM, et al. Prevalence of dry eye among the elderly. *Am J Ophthalmol*. 1997;124:723–728.
3. Trattler W, Reilly C, Goldberg D, et al. Prospective Health Assessment of Cataract Patients' Ocular Surface Study. Poster presented at: Annual meeting of the American Society of Cataract and Refractive Surgery; March 2011; San Diego, CA.
4. Rao SN. Topical cyclosporine 0.05% for the prevention of dry eye disease progression. *J Ocul Pharmacol Ther*. 2010;26(2):157–164.
5. Asbell PA. Increasing importance of dry eye syndrome and the ideal artificial tear: consensus views from a roundtable discussion. *Curr Med Res Opin*. 2006;22(11):2149–2157.
6. Doughty M, Blades K, Ibrahim N. Assessment of the number of eye symptoms and the impact of some confounding variables for office staff in non-air-conditioned building. *Ophthalmic and Physiological Optics*. 2002;22:143–155.
7. Singh K, Axelrod S, Bielory L. The epidemiology of ocular and nasal allergy in the United States, 1988–1994. *J Allergy Clin Immunol*. 2010 ;126(4):778–783.e6.

TARGET AUDIENCE

This certified CME activity is designed for all ophthalmologists and general eye care professionals.

LEARNING OBJECTIVES

Upon completion of this activity, participants should be able to

- distinguish the signs and symptoms of dry eye disease and ocular allergy
- know the role of diagnostic techniques in diagnosing dry eye and ocular allergy
- evaluate therapies to determine effective choices for treating dry eye and ocular allergy

METHOD OF INSTRUCTION

Participants should read the CME activity in its entirety. After reviewing the material, please complete the self-assessment test, which consists of a series of multiple-choice questions. To answer these questions online and receive real-time results, please visit http://www.dulaney-foundation.org and click "Online Courses."

Upon completing the activity and achieving a passing score of over 70% on the self-assessment test, you may print out a CME credit letter awarding *1 AMA PRA Category 1 Credit.*™ The estimated time to complete this activity is 1 hour.

ACCREDITATION AND DESIGNATION

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FACULTY/STAFF DISCLOSURE DECLARATIONS

Dr. Stonecipher has received grant/research support from Alcon Laboratories, Inc.; Allergan, Inc.; and Nidek; and is a consultant to Alcon Laboratories, Inc.; Allergan, Inc.; Ista Pharmaceuticals, Inc.; Merck & Co., Inc.; Nexis Vision; and Nidek. He is a member of the speakers' bureau for Abbott Medical Optics Inc.; Alcon Laboratories, Inc.; Allergan, Inc.; Bausch + Lomb; Endure; Merck & Co., Inc.; Nidek; Oasis Medical; and is a stock or shareholder in Nexis Vision.

Dr. Donnenfeld has received grant/research support from Abbott Medical Optics Inc.; Alcon Laboratories, Inc.; Allergan, Inc.; AqueSys, Inc.; Bausch + Lomb; Elenza; Glaukos Corporation; LacriSciences, LLC; Merck & Co., Inc.; Odyssey Medical; Pfizer Inc.; QLT Inc.; SARcode Bioscience, Inc.; TearLab Corporation; TLC Laser Centers; TrueVision; and WaveTec Vision.

Dr. McDonald has been a consultant to Abbott Medical Optics Inc.; Ace Vision Group; Alcon Laboratories, Inc.; Allergan, Inc.; Bausch + Lomb; Focus Laboratories; Ista Pharmaceuticals, Inc.; Merck & Co., Inc; Nexis Vision; Santen Pharmaceutical Co., Ltd.; TearLab Corporation; and Topcon Medical Systems, Inc..

All others involved in the planning, editing, and peer review of this educational activity have indicated they have no financial relationships to disclose.

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Implementing Treatment Strategies to Protect the Ocular Surface

First, identify the need. Then, treat the disease. Finally, proceed to surgery.

BY KARL G. STONECIPHER, MD

To maximize visual acuity outcomes, it is imperative that eye care specialists take the time to examine patients' ocular surface. Much can be learned from this workup, similar to taking patients' history. Stain patterns—from the lid, a diffuse stain, or a pattern indicative of meibomian gland disease—can provide invaluable information and direct the course of treatment (Figure 1).

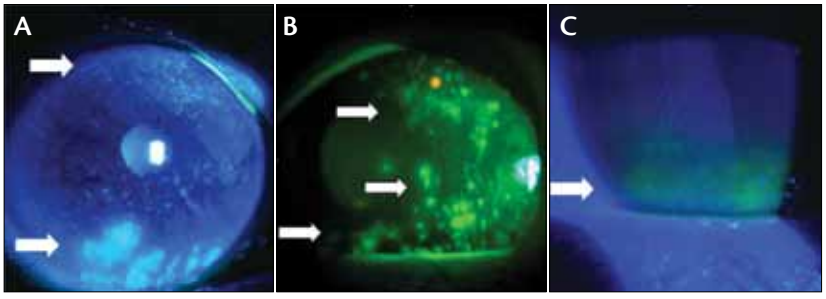

Figure 1. Staining patterns in inflammatory disease: lid pattern staining (A), diffuse staining (B), and meibomian gland disease (C).

DRY EYE DISEASE

Background

Dry eye disease (DED) is a progressive condition as its various pathophysiological causes continually influence and aggravate one another.[1] It develops as a disease much like glaucoma,[2] and with time, patients experience a worsening of the signs and symptoms associated with DED.

Although treatment with artificial tears can provide short-term relief, it will not prevent the disease's progression.[3] In fact, it is my experience that patients on average have already tried at least three preparations of artificial tears before they present to their eye care specialist. Therefore, it is important to appropriately identify patients by the severity of their DED. The best way to do this is by using the consensus guidelines.[4]

Level 1 DED is defined as the patient's having mild to moderate symptoms and no signs or mild to moderate conjunctival signs. In level 2 disease, the patient has moderate to severe symptoms, tear film signs, mild corneal punctate staining, conjunctival staining, and visual signs. Patients with level 3 disease exhibit severe symptoms, marked corneal punctate staining, central corneal staining, and filamentary keratitis. Finally, level 4 DED is associated with severe symptoms and severe corneal staining, erosions, and conjunctival scarring.

Environment

Clinicians should also consider environmental factors in the setting of DED. For example, certain medications can exacerbate the problem, as well as pollutants, heavy visual tasking (computers monitors, televisions, staring at paperwork), and excessive alcohol consumption. The physical environment certainly plays a role; for example, if a patient goes from a humid, seaside environment to one that is dry, he or she may experience symptoms of DED.

Potential Treatment

Consider the Rao study,[3] which looked at topical cyclosporine (Restasis; Allergan, Inc.) for the prevention of the progression of DED (Figure 2). The study compared patients using cyclosporine with those using carboxymethylcellulose sodium 0.5% (Refresh Endura lubricant, now called Optive; Allergan, Inc.). There were 74 patients enrolled from February 2006 to January 2007, and 58 completed 1 year of study (36 in the cyclosporine or treatment group, 22 in the lubricant or control group). When looking at the percentage of patients whose severity of DED increased, 5.5% (2 of 36) of patients using cyclosporine compared with 31.8% (7 of 22) of those using the lubri-

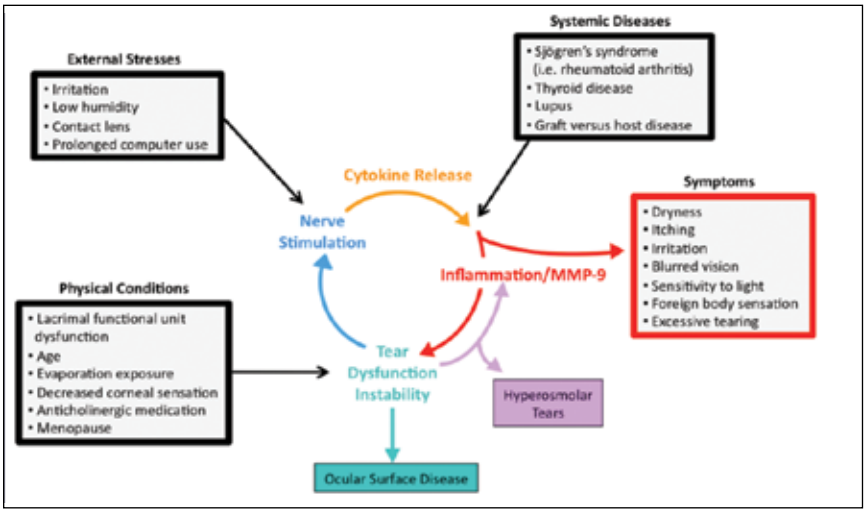

Figure 2. The cycle of inflammation.

cant (control) progressed (P = .007). This is according to the severity level determined by the Delphi consensus guidelines.[5]

Schirmer strip scores showed that, in that control group, there was a progression of effect, whereas in the treatment group, there were improvements in the Schirmer test scores as time went along. There was also a dramatic decrease in the percentage of treated patients who reported a worsening of symptoms versus the percentage in the control population.

SCREENING TOOLS

Ocular Surface Disease Index

A very useful screening tool is the Ocular Surface Disease Index (OSDI). This tried-and-true instrument continues to be used throughout 2 decades of clinical research and practice; it is a validated 12-item questionnaire.[6,7] In my practice, I use this quick screening tool as an intake form. It allows for the quantification of patients' symptoms, and it can reduce the time it takes to perform an examination. I have a technician ask the patient to fill out the OSDI while he or she is the waiting room. Based on the information provided, we can determine if further testing is needed. By the time the patient is in the examination room and I have reviewed the questionnaire, I can ascertain whether the patient has mild, moderate, or severe disease. The OSDI has such a high specificity that, in many cases, I can base my treatment choice on information from this simple screening.

Inflammatory Markers

One of the newer ways to evaluate the health of the ocular surface is to identify the inflammatory markers present in the tear film. Many studies have elucidated the importance of the concentration of tear proteins, lisozymes, and lactoferrins in tears.[8]

An especially useful consideration in terms of inflammatory markers in the tear film is its level of matrix metalloproteinase-9 (MMP-9).[9] These proteolytic enzymes are produced by stressed epithelial cells on the ocular surface. They represent a nonspecific inflammatory marker that has a normal range between 3 and 41 ng/mL. MMP-9 is a more sensitive diagnostic marker than clinical signs that correlate with clinical exam findings. A positive test result would be an MMP-9 level of 40 ng/mL or more, and a negative test result would be MMP-9 less than 40 ng/mL. Increased levels of MMP-9 may contribute to deranged corneal epithelial barrier function, increased corneal desquamation, and corneal surface irregularities. It is a true marker for DED.

InflammaDry (Rapid Pathogen Screening, Inc.; not yet available in the United States) is a test that evaluates a small sample (10 µL) of tear fluid for the presence of MMP-9. The device uses nanogold technology and is built on a platform that involves the direct sampling of a microfiltration immunoassay.

A clinical trial performed by the company (data on file with Rapid Pathogen Screening, Inc.) compared the sensitivity and specificity of the InflammaDry to clinical assessment and the confirmation of the clinical diagnosis of DED. Researchers conducted a prospective, sequential, masked, clinical trial with seven sites and 206 patients, of whom 143 patients were positive for DED and 63 were healthy controls. The criteria evaluated were clinical signs of dry eye, OSDI score greater than 13, Schirmer tear test score less than 10, tear breakup times less than 10, and corneal staining greater than 1. The healthy control had to have an OSDI of less than 7, Schirmer tear test of greater than 10, tear film breakup time of greater than 10, and total corneal staining of none. In the study, interestingly, the sensitivity was 85% and the specificity was 94% and the overall agreement was right at around 87%.

Tear Film Breakup Time and Osmolarity

Tear film instability is a hallmark of DED, and tear breakup time measures the quality of the tear film and

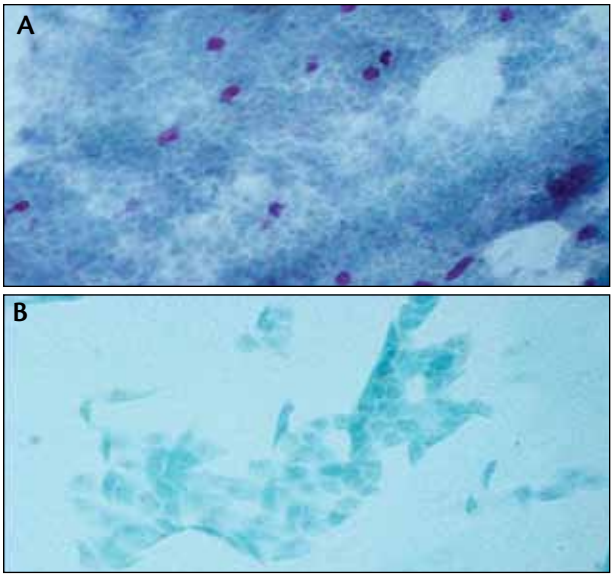

Figure 3. Impression cytology: normal (A) and DED (B).

its ability to resist thin spots (Figure 3). Fluorescein is introduced from a strip, and the yellow filter increases sensitivity. Tear breakup time (TBUT) is calculated from a completed blink to the first dry spot (three repetitions). A TBUT less than 10 seconds is considered abnormal. The sensitivity of this testing is 92% but the specificity drops off to 17%. Corneal staining has a sensitivity of 63% and specificity of 89%. The use of a questionnaire is associated with an 89% sensitivity and 72% specificity overall.[10]

There is some disagreement among specialists with regard to TBUT. For example, when I ask most physicians what they think is an abnormal TBUT they will usually say 10 seconds. Most of the studies of DED define this telltale marker as 7 seconds. Nevertheless, TBUT is a good way to determine the quality of the tear film and how it interacts on the ocular surface in terms of evaporation and other factors.

A new tool available to practitioners is the evaluation of tear film osmolarity, which can show if a patient's disease is mild, moderate, or severe. The TearLab Osmolarity System (TearLab Corporation) provides a measurable item that can be shown to patients and it helps guide treatment. The system's "lab test on a microchip" requires a small sample (50 nL) of the patient's tear film, which is gathered by a special tip and then inserted into the unit for measurement. The TearLab tests showed a sensitivity of 64% and a specificity of 71% in its evaluations.[11] It does not have the same level of sensitivity and specificity as InflammaDry, and is not as sensitive and specific as the OSDI questionnaire. There may be easier tests available, but I believe this testing modality is useful and my colleagues and I have used this in our office.

SURGICAL IMPLICATIONS OF DED

The incidence of DED in the LASIK population has been cited as being 50%, although this varies widely. Most commonly, it occurs within 6 months after surgery and it may last 1 year or more. However, in my experience anywhere from 25% to 33% of patients choose to undergo refractive surgery because they cannot wear their contact lenses or they are coming in with a diagnosis of DED before undergoing surgery.

Studies show that the use of a femtosecond laser reduces the incidence of DED postoperatively, and therefore it is my preferred method.[12-17]

I performed a study that looked at the postoperative day 1 and 3 month uncorrected visual acuity in patients who were treated with topical cyclosporine prior to surgery.[18] These were patients that I deemed as having mild to moderate DED. I found that 80% of my patients obtained a visual acuity of 20/16 and 92% to 93% were 20/20.

In my practice, my colleagues and I get great vision on postoperative day 1 with that "wow factor," but the patients are also able to maintain that level of vision at 3 months. It is important to note that patients with DED have a higher rate of enhancement if the condition is not adequately treated before refractive surgery. ■

1. Nelson JD, Helms H, Fiscella R, et al. A new look at dry eye disease and its treatment. *Adv Ther*. 2000;17(2):84-93.
2. Management and therapy of dry eye disease: Report of the Management and Therapy Subcommittee of the International Dry Eye Workshop (2007). *Ocul Surf*. 2007;5:108-152.
3. Rao SN. Topical cyclosporine 0.05% for the prevention of dry eye disease progression. *J Ocul Pharmacol Ther*. 2010;26(2):157-164.
4. Wilson SE, Stulting RD. Agreement of physician treatment practices with the international task force guidelines for diagnosis and treatment of dry eye disease. *Cornea*. 2007;26(3):284-289.
5. Behrens A, Doyle JJ, Stern L, et al. Dysfunctional tear syndrome: a Delphi approach to treatment recommendations. *Cornea*. 2006;25(8):900-907.
6. Schiffman RM, Christianson MD, Jacobsen G, et al. Reliability and validity of the Ocular Surface Disease Index. *Arch Ophthalmol*. 2000;118(5):615-621.
7. Perry HD, Donnenfeld ED. Dry eye diagnosis and management in 2004. *Curr Opin Ophthalmol*. 2004;15(4):299-304.
8. De Paiva CS, Pflugfelder SC. Diagnostic approaches to lacrimal keratoconjunctivitis. *Dry Eye and Ocular Surface Disorders*. New York, NY: Marcel Dekker; 2004:269-308.
9. Chotikavanich S, de Paiva CS, De Quan L. Production and activity of matrix metalloproteinase-9 on the ocular surface increase in dysfunctional tear syndrome. *Invest Ophthalmol Vis Sci*. 2009;50(7):3203-3209.
10. Versura P, Frigato M, Cellini M, et al. Diagnostic performance of tear function tests in Sjögren's syndrome patients. *Eye* (Lond). 2007;21(2):229-237.
11. TearLab performance testing summary. 510(k) summary; section VI. 2009. http://www.accessdata.fda.gov/cdrh_docs/pdf8/K083184.pdf.
12. Solomon KD, Holzer MP, Sandoval HP, et al. Refractive surgery survey 2001. *J Cataract Refract Surg*. 2002;28(2):346-355.
13. Albietz JM, Lenton LM, McLenmnan SG. The effect of ocular surface management of myopic LASIK outcome. *Adv Exp Med Biol*. 2002;506(Pt A):711-717.
14. Jabbur NS, Sakatani K, O'Brien TP. Survey of complications and recommendations for management in dissatisfied patients seeking a consultation after refractive surgery. *J Cataract Refract Surg*. 2004;30(9):1867-1874.
15. Tanaka M, Takano Y, Dogru M, et al. Effect of preoperative tear function on early functional visual acuity after laser in situ keratomileusis. *J Cataract Refract Surg*. 2004;30(11):2311-2315.
16. Toda I, Asano-Kato N, Komai-Hori Y, et al: Dry eye after laser in situ keratomileusis. *Am J Ophthalmol*. 2001;132(1):1-7.
17. Albietz JM, Lenton LM, McLenmnan SG. Effect of laser in situ keratomileusis for hyperopia on tear film and ocular surface. *J Refract Surg*. 2002;18(2):113-123.
18. Stonecipher KG, Perry HD, Gross RH, Kerney DL. The impact of topical cyclosporine A emulsion 0.05% (tCSA) on the outcomes of patients with keratoconjunctivitis sicca (KCS). *Curr Med Res Opin*. 2005;21(7):1057-1063.

ALLERGIC CONJUNCTIVITIS

According to the Asthma and Allergy Foundation of America, approximately 50 million people in this country have allergies, and the prevalence has been steadily rising since the 1980s.[1] Close to half of those who suffer from allergies have ocular allergy symptoms.[2] There are five main types of allergic eye disease: seasonal allergic conjunctivitis, perennial allergic conjunctivitis, vernal keratoconjunctivitis, atopic keratoconjunctivitis, and giant papillary conjunctivitis. The latter three conditions can be responsible for severe ocular morbidity, however, they will not be discussed herein. Seasonal allergic conjunctivitis is the most common type, it is usually triggered by pollen, and spikes are experienced during the spring and summer months. Perennial allergic conjunctivitis is caused by dust mites, pet dander, and mold, therefore symptoms can occur at any time during the year.

Ocular surface inflammation plays a key role in the pathogenesis and symptomatology of allergic conjunctivitis. Both seasonal and perennial allergic conjunctivitis are type 1 hypersensitivity reactions that involve sensitization of the immune system upon first exposure of the antigen. Upon repeated exposure, the antigen-specific IgE binds to conjunctival mast cells. This triggers their degranulation and then they release intracellularly stored mediators that include histamine, tryptase, chymase, heparin, chondroitin sulfate, prostaglandins, thromboxanes, and leukotrienes.[3] This series of events is considered the acute phase of the allergic response. Later, the release of these mediators combined with a multitude of chemotactic factors causes increased vascular permeability and the attraction and migration of eosinophils, neutrophils, and lymphocytes. What results are the signs and symptoms of allergic conjunctivitis, which, it is important to note, often overlap with other forms of ocular surface disease like dry eye, meibomian gland disease, and infectious conjunctivitis.

The most important clinical signs of allergic conjunctivitis include hyperemia of the conjunctiva and the eyelids; clear, watery, scant discharge; conjunctival chemosis and papillae; and eyelid edema.

The key symptoms of allergic conjunctivitis include itching, tearing, burning, foreign body sensation, and ocular dryness. Itching, of course, is considered the hallmark symptom. In fact, if there is no itching then the diagnosis of allergic conjunctivitis should be reconsidered. Clinicians should educate patients to the fact that vigorous eye rubbing will lead to mast-cell degranulation and make itching worse. The nonocular symptoms of allergic conjunctivitis can include sneezing, rhinorrhea, and nasal congestion. It is also important to note that antihistamine medications can cause or exacerbate dry eyes, and patients should also be told of this connection.

TREATING ALLERGIC CONJUNCTIVITIS

For all of the forms of allergic conjunctivitis, it is important to identify the offending allergen and avoid exposure to it. Of course, pollen is ubiquitous and avoiding it can be close to impossible. Patients can be instructed to wear wraparound protective eyewear and brimmed hats when outdoors, and frequently washing their clothes, hands, and hair can reduce the antigenic load. Another step is the frequent instillation of artificial tears. This works to help reduce the antigenic load by diluting the allergen and irrigating the ocular surface. Use of chilled artificial tears and the application of cold compresses to the eyelids can soothe the irritation and help reduce hyperemia.

The foundation of treating allergic conjunctivitis is to block H1, H2, and, in some cases, H4 histamine receptors, which can be achieved using topical and oral agents. Antihistamines quickly and effectively reduce itching, hyperemia, and edema of the ocular surface. Mast-cell stabilization, sometimes combined with topical antihistamine drops, can also cut down on mast-cell degranulation upon further antigen exposure. In more severe cases, adjunctive anti-inflammatory medications are often indicated and can include topical steroids, topical nonsteroidal anti-inflammatory drugs, and topical cyclosporine A.

1. CDC Fast Facts A-Z. Vital Health Statistics, 2003. http://www.cdc.gov/nchs/fastats.
2. Bielory L. Allergic and immunologic disorders of the eye. Part II: ocular allergy. *J Allergy Clin Immunol.* 2000;106(6):1019-1032.
3. Abelson MB, Smith L, Chapin M. Ocular allergic disease: mechanisms, disease subtypes, treatment. *Ocul Surf.* 2003;1(3):127-149.

DED Cases: Cataract Surgery, Sjögren Syndrome

Two cases illustrate key management pearls.

BY KARL G. STONECIPHER, MD

The case is that of a woman who is scheduled to undergo cataract surgery but otherwise does not complain of symptoms associated with dry eye disease (DED). As part of my preoperative workup, I administered lissamine green and she showed strong positive signs.

What is best course of action for this patient?

- operate
- pretreatment with punctal plugs
- pretreatment with a preparation of artificial tears
- pretreatment with topical cyclosporine

The latter three options from this list are all appropriate. I typically prescribe cyclosporine in this patient population because in my experience, it increases tear production and it allows for better tear flow. I also pretreat such patients with an antibiotic and a steroid for 3 to 4 days, and I also add a nonsteroidal anti-inflammatory drug.

WHEN TO PROCEED TO SURGERY

When the patient no longer shows signs of conjunctival staining, then cataract surgery can be scheduled. It is not advisable to operate on a patient who has evidence of corneal staining. This is particularly true when the patient is interested in a refractive cataract procedure or is expecting to receive a multifocal or an accommodating IOL.

The number one reason that patients are referred to me is because they are unhappy with their vision after receiving multifocal lens implants. The typical patient presents after a successful, routine procedure has been performed by another surgeon. Typically I find that DED was not diagnosed preoperatively and, therefore, these individuals are unhappy with their quality of vision and their ocular surface health is compromised.

CONFIRMING DATA

As an example, consider to the results of the Prospective Health Assessment of Cataract Patient's Ocular Surface Study (PHACO).[1] This study was conducted among patients who presented to my practice with a diagnosis of cataract. It included 102 patients (204 eyes), nearly evenly split between men and women, and the population was 62% white, 11% Hispanic, 4% black, and 4% Asian; the mean age was 71 years. Upon presenting for cataract surgery, 15% had a prior diagnosis of DED.

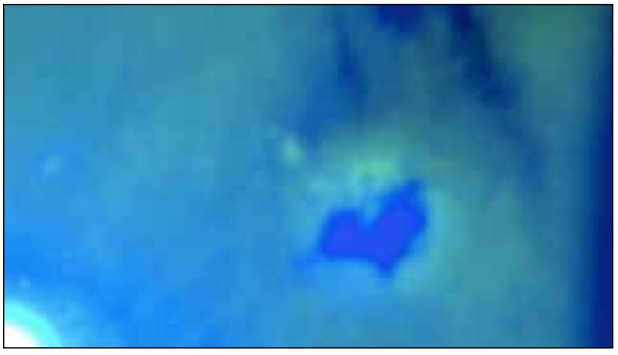

Figure 1. The average tear breakup time (TBUT) was 4.93 seconds with 126 eyes (61.7%) having less than 5 seconds and 169 eyes (82.8%) having 7 seconds or less.

Although many practitioners consider 10 seconds to be an abnormal TBUT, current studies indicate that 7 seconds is a better indication of DED.[2] We were quite amazed that, in our study, the average TBUT was 4.93 seconds, with 126 eyes (61.7%) having less than 5 seconds and 169 eyes (82.8%) having 7 seconds or less (Figure 1).

Take-Home Point

The take-home message is that in a so-called normal cataract surgery population, there are many patients with DED, and it often goes undiagnosed because ophthalmologists are not looking for it.[3] DED causes postoperative visual fluctuation—particularly in patients receiving premium IOLs—and this cannot be compensated for preoperatively. If the ocular surface is not pristine and healthy, the surgeon will have trouble calculating the correct IOL power.

Sjögren Syndrome

The next case is a woman with Sjögren syndrome who is also managed by a rheumatologist and a gastroenterologist. She has had all four punctae closed by thermal cautery, and her eyes are so dry that she cannot even cry.

What should be the next step in this patient?

- treat her with pulsed topical corticosteroids
- treat her with artificial tears
- treat her with topical cyclosporine
- treat her with autologous serum

The goal is to gain some improvement in the quality of the tear film for these types of patients. Many of them will not respond to topical cyclosporine, so that is when other options such as autologous serum, topical corticosteroids, and certain types of tear products should be considered.

BACKGROUND DATA

These individuals typically require aggressive solutions such as punctal occlusion, topical steroids, lubricants, or hydroxypropyl cellulose ophthalmic inserts (Lacrisert; Valeant Ophthalmics). The Delphi panel[4] and the Dry Eye Workshop (DEWS)[5] treatment scales are two ways to consider diagnosis and treatment. The Delphi panel classifies patients without lid margin disease into four levels, and the DEWS panel incorporates the presence of meibomium gland disease into severity levels. I recommend starting at level two with anti-inflammatories, tetracyclines (for meibomianitis and rosacea), punctal plugs, secretogogues, and moisture chamber spectacles.

For the DEWS treatment algorithm, those researchers considered only those therapies with solid evidence in the literature to support efficacy. In Sjögren patients, I would advance to the level 3 and 4 treatments, which include autologous serum, contact lenses, and permanent punctal occlusion. If level 3 treatments are inadequate, I would add systemic anti-inflammatory agents and consider surgery (ie, lid, tarsorrhaphy, mucus membrane, salivary gland, and amniotic membrane transplantation).

I believe systemic supplements, such as those that contain omega-3 fatty acids, are useful in all patients. There are many different brands and formulations of nutriceuticals that may be beneficial. In patients with Sjögren syndrome and level 4 disease as defined by DEWS, a surgeon needs to be particularly careful in how he or she proceeds. For patients' postoperative comfort, there are many ancillary considerations such as the use of a humidifier or goggles to keep the eyes moist.

CONCLUSION

Patients such as the one in this example require a lot of chair time. I find that an educated patient leads to the best outcomes, so in my practice I start with a 90-second screening. I will have the patient complete the Ocular Surface Disease Index, and often I will prescribe cyclosporine at that visit. Next, I ask patients to learn as much as they can about their condition. Treatment modalities and regimens change, and www.webmd.com can be an especially helpful resource for patients. The website www.healthywomen.org is another good tool, because the majority of DED sufferers are women.

Patients on continuous positive airway pressure for sleep apnea often incur DED because they wear masks overnight. Men might be on different types of therapeutic regimens following prostate cancer procedures, and many medications can exacerbate DED. ■

1. Trattler W, Reilly C, Goldberg D, et al. Prospective Health Assessment of Cataract Patients' Ocular Surface Study (PHACO). Poster presented at: The Annual Meeting of the American Society of Cataract and Refractive Surgery; March 2011; San Diego, CA.
2. Abelson MB, Ousler GW 3rd, Nally LA, et al. Alternative reference values for tear-film breakup time in normal and dry eye populations. *Adv Exp Med Biol*. 2002;506(Pt B):1121-1125.
3. Trattler W, Reilly C, Goldberg D, et al. Prospective Health Assessment of Cataract Patients' Ocular Surface Study. Poster presented at: The Annual Meeting of the American Society of Cataract and Refractive Surgery; March 2011; San Diego, CA.
4. Behrens A, Doyle JJ, Stern L, et al. Dysfunctional tear syndrome: a Delphi approach to treatment recommendations. *Cornea*. 2006;25(8):900-907.
5. 2007 Report of the Dry Eye Workshop. *Ocul Surf*. 2007;5(2):65-204.

Case: Perimenopausal Woman With DED

This classic presentation exemplifies the disease.

BY ERIC D. DONNENFELD, MD

Dry eye disease (DED) is one of the most underdiagnosed and prevalent diseases that is seen in eye care practitioners' offices. It is estimated that up to 10% of women will suffer from DED and more than 3% of men.[1]

CASE

This case is that of a 47-year-old woman who is an architect and myopic. Her symptoms include a foreign body sensation, severe burning, visual blurring, and excessive tearing. She has had to reduce her contact

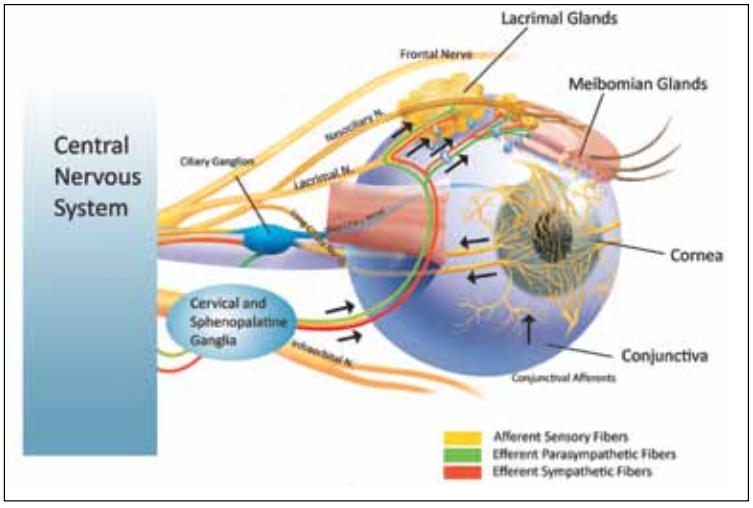

Figure 1. Schematic of the ocular surface. The combination of aging and menopause causes a reduction in corneal sensation and an increase in the inflammatory cells that damage the ocular surface.

lens wear, she frequently uses unpreserved artificial tears, and she has experienced serious limitations on her daily activities.

In a classic presentation, her dry eye started 5 years ago and she has experienced episodic symptoms for 3 years; the symptoms became constant with progressive worsening during the past 2 years. Now, her chronic symptoms are present all day, and her medical history shows irregular menstrual cycles, suggesting that she is going through menopause.

Research has shown that tear breakup time was markedly and statistically significantly improved with cyclosporine, and there was a slight worsening in untreated patients.[2] There was a significant improvement in ocular surface staining at 1 year in patients who received cyclosporine compared with no statistically significant difference in patients who were treated with artificial tears.[2] The severity level of DED also worsened in the patients who received artificial tears versus a 5% worsening among the cyclosporine-treated patients—a statistically significant result. These results show that DED is a progressive disease.[3]

HORMONAL CHANGES

Several studies have found that hormonal changes, mostly associated with menopause, cause a worsening in the symptoms of DED, and that hormonal therapy can actually improve the condition.[4,5] The patient who is at the greatest risk of developing DED is a perimenopausal woman in her 40s and 50s, particularly one undergoing ocular surgery.

Aging and hormones affect the pathogenesis of DED. Aging causes decreased corneal sensory signaling, which provides less information to the brain stem. Hormones have multiple effects, and menopause combines the effects of aging and hormonal changes. In this schematic of the ocular surface (Figure 1), the combination of aging and menopause with reduced androgens causes a reduction in corneal sensation and an increase in inflammatory cells that damage the ocular surface.[6]

VISUAL EXAMINATION

The physical examination of the woman in this case showed 2+ corneal fluorescein staining, 2+ conjunctival lissamine green staining, a decreased tear meniscus, and inspissated meibomian gland secretions. She obviously had disease caused by a combination of mechanisms, as well as aqueous deficiency and evaporative DED (Figure 2). Her tear function on Schirmer testing (with anesthesia) was 5 in the right eye and 4 in the left eye. Her tear breakup time was reduced, with a value of 4 in the right eye and 5 in the left. She had elevated levels of matrix metallopeptidase-9 (MMP-9). The test for MMP-9 is very sensitive, and it detects an enzyme secreted by corneal epithelial cells that have been traumatized by DED.[7]

The Dysfunctional Tear Syndrome, Diagnosis, and Treatment Guide describes severity levels of DED starting with level 1 and going to level 4, based on both signs and symptoms of the disease.

Figure 2. The patient obviously had disease caused by a combination of mechanisms as well as aqueous deficiency and evaporative DED.

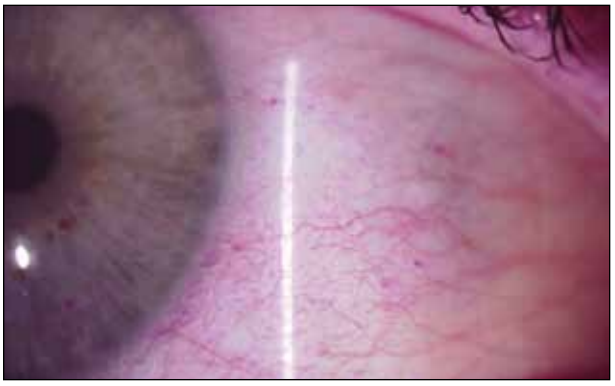

Figure 3. The patient's corneal fluorescein staining reduced to zero, and there was just minimal rose bengal staining of the conjunctiva.

Based on the information presented, what level of DED does the patient described in this case have? Because there is significant corneal and conjunctival staining, I would classify her as having level 2 DED. Her disease is significant, it has progressed, and her condition is in danger of leading to visual debilitation.

TREATMENT

I would start this patient on a regimen of unpreserved artificial tears. I would suggest she administer cyclosporine twice a day and I would consider a corticosteroid for 3 to 4 weeks. I would also prescribe oral fish and flaxseed oil to address the nutritional component to this disease. Combining these treatments gives patients a rapid immunomodulation and controls the disease. This type of patient would normally start responding at about 1 month and would have maximum response to therapy at 3 to 6 months.

For this patient, I did recommend that she discontinue contact lens wear, and I prescribed a unit dose of an unpreserved artificial tear. I added oral fish and flaxseed oil, and at 2 weeks her corneal and conjunctival staining improved to level 1. However, she reported that the foreign body sensation and burning persisted.

At this point, I started her on a topical corticosteroid containing loteprednol four times a day for 1 month in addition to cyclosporine twice a day. Although this combination of a corticosteroid and cyclosporine produces dramatically better results, steroids can only be used for the short term. Cyclosporine is used for long-term maintenance.[8]

OUTCOMES

After 1 month this patient had further improvement in corneal and conjunctival staining, decreased symptoms, and she was considering going back to her contact lenses. If she had not responded, additional therapeutic considerations would include punctal plugs. I am a big believer in punctal plugs, and I always start them after the patient has already received immunomodulation therapy. I do not put plugs in patients with inflammatory DED, however, because it causes the tears to stay in contact with the ocular surface.

As her symptoms improved, the patient in this example had reduced irritation and foreign body sensation, so she decreased her use of artificial tears to three times a day. Her quality of life improved and, remarkably, the Schirmer testing tripled from 4 and 5 to 17 and 15. This is consistent with the FDA trial of cyclosporine, in which 15% of the patients who received the agent had a tripling or more of their Schirmer scores and the tear film became more stable as evidenced by a tear breakup time of 8 to 9 seconds. Her corneal fluorescein staining reduced to zero, and there was just minimal rose bengal staining of the conjunctiva (Figure 3).

SUMMARY

Artificial tears are not a sufficient treatment for chronic DED. They may provide palliative relief, but they do not treat the active component of inflammation associated with DED. Active pharmaceutical therapies are required to halt or reverse the disease's progression. DED is a progressive disease that responds well to immunomodulation, so the sooner that therapy is initiated, the better the patients will respond. Perimenopausal women with DED may benefit from aggressive therapy including anti-inflammatory agents.

CONCLUSION

Consider the entire patient when evaluating him or her for DED. Systemic conditions such as menopause can affect the eye and have an adverse effect on the health of the ocular surface. We as eye care providers must be aware of this, and be ready and willing to treat patients aggressively when they come to us with symptoms that can be debilitating to their quality of life. ■

1. Schaumberg DA. In: Foster CS et al, eds. *Smolin and Thoft's The Cornea Scientific Foundations and Clinical Practice*. Philadelphia, PA: Lippincott Williams & Wilkins; 2004:205-219.
2. Rao SN. Topical cyclosporine 0.05% for the prevention of dry eye disease progression. *J Ocul Pharmacol Ther*. 2010;26(2):157-164.
3. Behrens A, Doyle JJ, Stern L, et al. Dysfunctional tear syndrome: a Delphi approach to treatment recommendations. *Cornea*. 2006;25(8):900-907.
4. Scott G, Yiu SC, Wasilewski D, et al. Combined esterified estrogen and methyltestosterone treatment for dry eye syndrome in postmenopausal women. *Am J Ophthalmol*. 2005;139(6):1109-1110.
5. Taner P, Akarsu C, Atasoy P, et al. The effects of hormone replacement therapy on ocular surface and tear function tests in postmenopausal women. *Ophthalmologica*. 2004;218(4):257-259.
6. Stern ME et al. In: Pflugfelder SC et al, eds. *Dry Eye and Ocular Surface Disorders*. New York, NY: Marcel Dekker; 2004:41-62.
7. Chotiakavanich S, de Paiva CS, Li de Quan, et al. Production and activity of matrix metalloproteinase-9 on the ocular surface increase in dysfunctional tear syndrome. *Invest Ophthalmol Vis Sci*. 2009; 50(7): 3203-3209.
8. Sall K, Stevenson OD, Mundorf TK, et al for the CsA Phase 3 Study Group. Two multicenter, randomized studies of the efficacy and safety of cyclosporine ophthalmic emulsion in moderate to severe dry eye disease. *Ophthalmology*. 2000;107(4):631-639.

Cases: Contact Lens Intolerance, Stargardt Disease, and Severe OSD

BY MARGUERITE B. McDONALD, MD

A 53-year-old marketing executive with a long history of successful contact lens wear in the past presented to me with red eyes, fluctuating vision, and the acute onset of contact lens intolerance. She had a recent hysterectomy for fibroids, but she was not on hormone replacement therapy due to a family history of breast cancer. She had tremendous difficulty removing her lenses, stating that they felt like they were stuck. The amount of time she was able to wear her contact lenses daily was about 2 hours, which is significantly less than the prescribed 16 hours.

This patient had been wearing polymethylmethacrylate contact lenses since she was 15 years old, then switched to gas permeable lenses, and had more recently opted for 2-week disposable soft lenses. She reported her symptomatology as much worse in the evening and exaggerated by cold weather, flu and allergy medications, and alcohol use. She expressed an interest in getting out of spectacles for cosmetic reasons (ie, pertaining to her line of work), but was too myopic for LASIK on previous evaluations by several ophthalmologists because her refractive error was -10.00 D in both eyes.

On initial examination 8 years prior, her BCVA was 20/25 in the right eye and 20/30 in the left. She had nasal and temporal staining of the conjunctiva with rose bengal, and inferocentrally with lissamine green (Figure 1). She had Schirmer scores of only 3 and 2 mm at 5 minutes and a tear breakup time of only 3 and 4 seconds. She also had trace to 1+ conjunctival papillary changes and 2 to 3+ conjunctival erythema, especially nasally and temporally.

What is the most appropriate diagnosis for this patient?

- dry eye disease (DED)
- seasonal allergic conjunctivitis
- contact lens warpage syndrome
- all of the above

This patient has fairly significant DED. I started her on unpreserved tears every 2 hours while awake, cyclosporine emulsion twice a day, a gel at night, unpreserved gel in both eyes, and oral omega-3 nutritional supplements. Her ability to wear soft contact lenses improved from 2 to 4 hours by 6 weeks, after which point she received four intracanalicular punctal plugs. This improved her DED even further, and by 3 months from the time therapy was initiated she was wearing

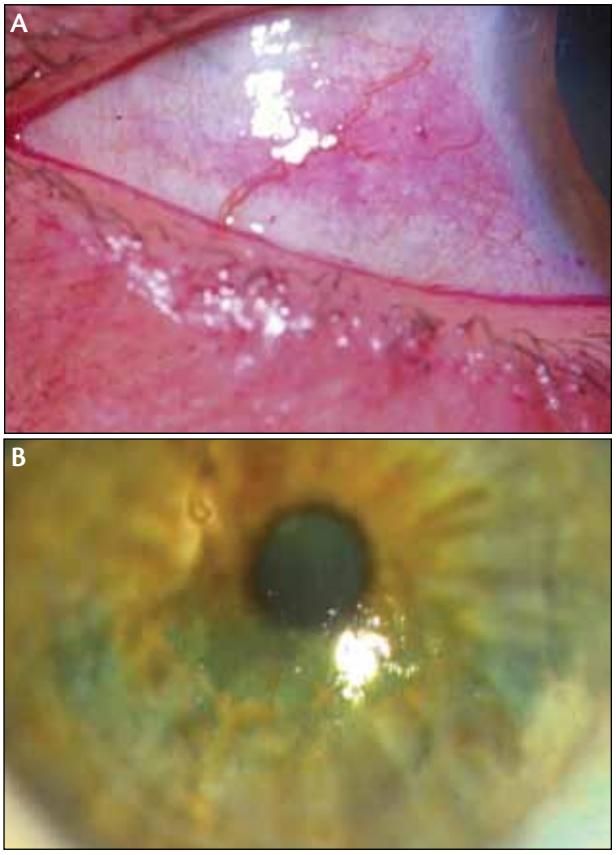

Figure 1. Nasal and temporal staining of the conjunctiva with rose bengal (A), and inferocentrally with lissamine green (B).

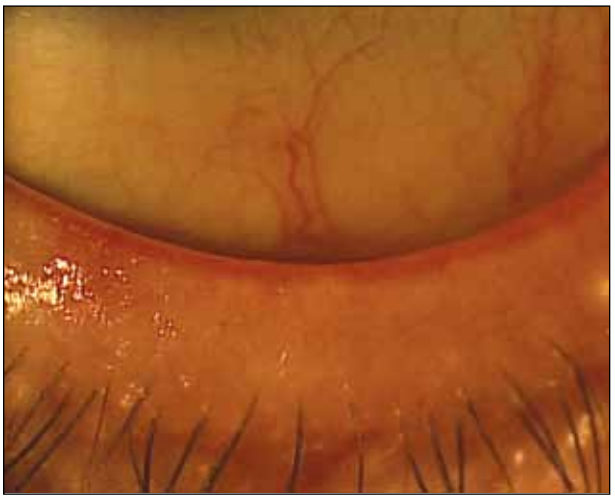

Figure 2. Slit-lamp evaluation revealed 1+ papillary changes in both eyes and 3+ meibomian gland inspissation without any lid scurf.

her lenses for 16 hours every day.

Eight years after starting therapy, she was still able to wear her daily lenses 16 hours a day and reported that they felt "comfortable." Her BCVA was 20/20, Schirmer testing had improved to 8 and 10 mm, and a test for matrix metalloproteinases 9 (MMP-9) (a marker of inflammation), was negative. Additionally, her tear osmolarity testing showed scores of 298 and 306 mOsm/L, which are in the normal range. Had the last two tests existed 8 years prior when therapy was started, it is likely should have tested positive for MMP-9 and tear osmolarity would have measured in the high 300s to 400 mOsm/L.

STARGARDT DISEASE

A 41-year-old woman with known Stargardt disease presented to me complaining of foggy, itchy, and burning eyes. She has a history of seasonal ocular allergies and uses artificial tears and ketotifen antihistamine drops as needed. She is an active individual who tries to work despite of her very decreased vision. As a result of Stargardt disease, she has BCVA of only count fingers at 4 feet and 5 feet, and she had a very decreased tear film bilaterally.

Slit-lamp evaluation revealed 1+ papillary changes in both eyes and 3+ meibomian gland inspissation without any lid scurf (Figure 2). Her tear breakup time was reduced to 4 and 5 seconds, respectively, and her Schirmer scores were also reduced at 5 and 6 mm at 5 minutes. Her tear osmolarity scores were 288 and 282 mOsm/L.

What is the diagnosis?

- DED
- meibomian gland disease (MGD)
- seasonal allergic conjunctivitis
- all of the above

The best choice here is all of the above. There are hallmark signs of DED and MGD, and there is past medical history of seasonal allergic, even though she is not in the middle of a flare with only trace to 1+ papillary changes.

This patient was started on lid soaks and scrubs twice a day with a commercially available, over the counter eye lid scrub pads, as well as azithromycin solution (AzaSite; Merck & Co., Inc.) twice a day (this is an off-label use). I have my patients rub it into their lid margins with their fingers right after they have cleansed their eyelids; this tends to save on the cost of the medicine because only half as much is used. Using azithromycin in this fashion also delivers drug directly to the target tissue with minimal stinging.

This patient was also prescribed artificial tears four times a day and as needed, omega-3 nutritional supplementation by mouth, and ketotifen up to twice a day, as needed, for itching. She returned to my examination room 7 weeks after her first visit with decreased pain, tearing and burning were gone, and her BCVA had improved to 20/200 in both eyes—an important outcome given the restoration of functionality in her life. She had 2 to 3+ meibomian gland inspissation but no scurf. Although there was still a decreased tear lake in both eyes, it was much improved. Tear osmolarity scores were 290 mOsm/L, which is in the normal range.

After this examination, cyclosporine emulsion twice a day was added, as well as loteprednol etabonate (Lotemax; Bausch + Lomb) four times a day for 2 weeks and then twice a day for 2 weeks. I have found that this regimen with a tapering of instillation decreases the stinging that sometimes occurs with the concomitant induction of cyclosporine therapy while also providing an immediate response, because as safe and effective as the agent is, it usually takes about 1 month to start having an effect. Otherwise, patients sometimes become discouraged by week 2 or 3 and stop taking their drops. If this patient's blepharitis continues to respond to therapy, I will likely insert plugs.

SEVERE OCULAR SURFACE DISEASE

This is a case of 61-year-old woman with a very long history of severe DED, MGD, severe seasonal allergic conjunctivitis, trichiasis, and exposure keratitis due to lagopthalmos caused by a blepharoplasty years ago with an aggressive removal of tissue. The patient reports severely red, uncomfortable, dry, burning, and itchy eyes that interfere with her professional and social activities. She is a top executive and she feels that her red, blurry, smeary eyes make people think that she has been up all night drinking or taking drugs.

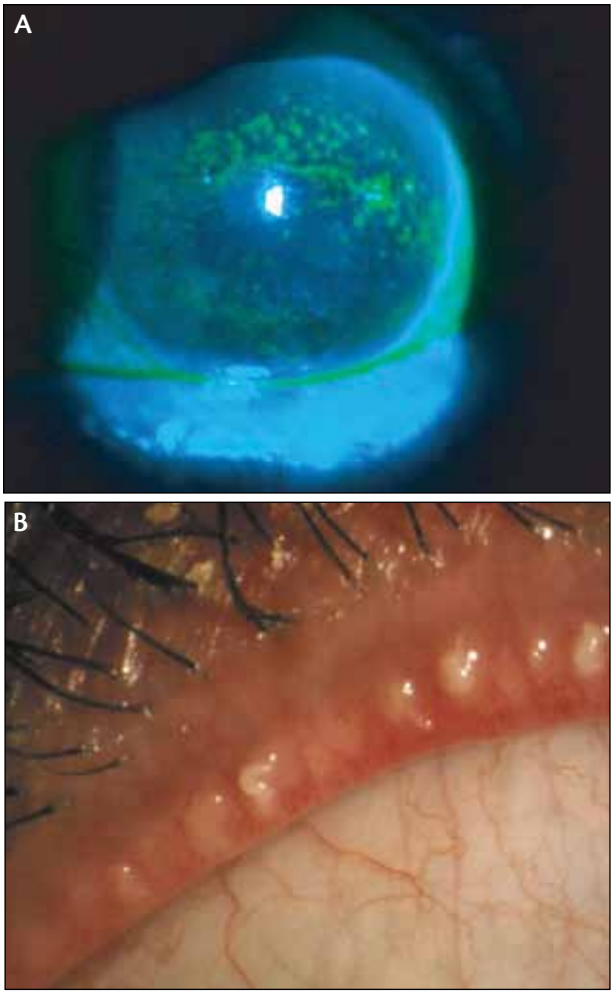

Figure 3. The patient has a very decreased tear lake in both eyes (A) and 3+ meibomian gland ispissation (B).

The patient has been using cyclosporine emulsion twice a day for several years, and she performs soaks and scrubs twice a day with azithromycin solution. She uses unpreserved tears every 2 hours while awake and takes doxycycline 15 mg by mouth daily. In addition, this patient uses liposome spray (Tears Again Liposome Lid Spray; Cynacon/Ocusoft, Inc.) two to four times a day, rubbing it in gently with her fingers if she is not wearing makeup, otherwise she sprays it on in the middle of the day. She uses gland ointment at night and takes omega-3 nutritional supplements. Other medications include epinastine HCl 0.05% twice a day as needed for itching. If she tries to stop any of these things, she says her eyes slam shut and she basically becomes completely functionless. She already has four punctal plugs and intermittently uses desonide cream for extreme flare-ups of MGD and facial or lid allergies. Demodex blepharitis has been ruled out after examination of her lashes under the microscope, so mites are not involved in this.

This patient begs for topical steroids on a routine basis, often calling the office two to three times a week. She is a steroid-responder but refuses latanoprost while on her steroids because of the remote chance of an iris color change, even though the rarity of this happening has been explained. As an alternative, bimatoprost (Lumigan; Allergan, Inc.) is prescribed twice a day while she is on loteprednol etabonate 0.2% and/or another topical steroid or steroid ointment. The patient visits our clinic several times a month for emergency visits, and compliance with her medication protocol is an ongoing issue.

At the time of examination, her UCVA was 20/20 uncorrected and intraocular pressure (IOP) measured 17 and 19 mm Hg. Her IOP usually measures around 14 to 15 mm Hg, but she had just given herself a few days of topical steroid. She reported to our clinical with 3 to 4+ lid erythema and edema in both eyes, 2 to 3+ papillary changes, 3+ meibomian gland inspissation, and a very decreased tear lake in both eyes. There is superficial punctate keratitis 3 to 4+ in both eyes, which is worse inferiorily (Figure 3). At the time of this clinical visit, she did not have any misdirected lashes, she had no trichiatic lashes, and tear osmolarity tests were 294 and 299 mOsm/L.

The question here is what are the treatment options for this patient moving forward? Stopping all treatments "cold turkey" has been attempted, but the patient reported having to sit home with her eyes closed. Which option would you choose?

- Keep the patient on the same regimen she is already on with no steroids except for extreme exacerbations.
- Keep the patient on the same regimen but add low-dose, constant loteprednol etabonate 0.2% (Alrex; Bausch + Lomb) instead of 0.5% once a day with monitoring of the IOP.
- Prescribe stronger or more frequent steroids for extreme exacerbations.
- Add hydroxypropyl cellulose inserts (Lacrisert; Valeant Pharmaceuticals, Inc.) at night in both eyes.

I chose the second route. I kept the patient on the same and rather vigorous regimen and added loteprednol etabonate 0.2% once a day, long term, with IOP monitoring. Ever since, she has been quiet and comfortable for more than 8 weeks. Her IOP has been measured twice, and she has not called the office or made any emergency visits. She still has extreme external disease, but reports being functional. Neither her ocular surface disease nor the treatment interfere with her professional and personal activities. ■

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CME QUESTIONS

1. Which of the following is true of dry eye disease (DED)?

a. it is a progressive condition

b. environmental factors are not important

c. patients with level 1 disease have scarring

d. there are no consensus guidelines to determine the severity of disease

2. Six months' use of a quality artificial tear, prescribed twice daily, can prevent DED.

a. TRUE

b. FALSE

3. What is considered the hallmark symptom of ocular allergy?

a. tearing

b. itching

c. burning

d. foreign body sensation

4. Which of the following is NOT a factor that can worsen DED?

a. menopause

b. aging

c. ocular surgery

d. eye color

5. What tear film breakup time is indicative of DED?

a. 20 seconds

b. 15 seconds

c. 10 seconds

d. 7 seconds

6. Which image shows meibomian gland disease?

a.

b.

c.

d.

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