Surgeons see OSD treatment as integral to practice success

by Eric Donnenfeld, MD

Greater than 25% of cataract patients present with asymptomatic dry eye preoperatively, and 1 in 5 cataract patients present for their preoperative consultation symptomatic of OSD that requires intervention beyond artificial tears, according to the ASCRS Clinical Survey on ocular surface disease (OSD). Respondents say more than half—53%—of their dry eye patients have a mix of meibomian gland dysfunction (MGD) and aqueous deficient dry eye disease (ADDE). Thermal lid expression and lid hygiene are the most common treatments for MGD; artificial tears and lubricants followed by oral omega-3 supplements are the most common treatments for moderate dry eye (Figure 1).

A related survey found that queried cataract and refractive surgeons think it is very important to understand the etiology of their patient’s OSD, whether it be MGD, ADDE, or both MGD and ADDE, and most are either very confident or somewhat confident in their ability to appropriately match treatments to the etiology and severity of OSD. Approximately 65% strongly agree that anterior segment imaging such as topography and meibography can help guide therapy and patient education about OSD, and a notable 82% agree that treating OSD can benefit their surgical practice.

When asked what they consider to be the greatest barrier to more consistently evaluating the tear film, lids, and meibomian glands in preoperative patients, the leading cause was not having access to advanced tear film or other diagnostic testing in their office, and the second most frequent factor was limited time during the preoperative work-up period.

This supplement presents advice from leaders in our field on the diagnosis and management of dry eye.

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Primary therapies and treatments for managing moderate dry eye disease

Figure 1. Respondents use various treatment options to treat dry eye disease.

Deys II stresses the pivotal role of hyperosmolarity in ocular surface disease (OSD) and suggests that the earlier the diagnosis and management of OSD, the better the outcome. The goal of treatment is to break the cycle of inflammation and restore tear homeostasis.

Mechanisms and management

The central mechanism of DED is an evaporative water loss that leads to hyperosmolar tissue damage and loss of homeostasis. Direct or indirect insult to the ocular surface via inflammation can cause loss of epithelial and goblet cells, and this results in decreased surface wettability with rapid tear breakup time, which initiates tear film instability and amplified hyperosmolarity. DED can be initiated by a variety of causes including Sjogren’s syndrome, rheumatoid arthritis, lupus, decreased estrogen levels in postmenopausal women, MGD resulting in a poor lipid layer of the tear film, allergy medications, and contact lens wear. LASIK and other ocular surgeries that cut corneal nerves can exacerbate pre-existing subclinical dry eye disease as well. All of these are capable of triggering a cascade of pro-inflammatory cytokines resulting in hyperosmolarity; thus the goal of OSD treatment is restoration of the tear film homeostasis.

Visual fluctuations due to increased tear breakup time (TBUT) and the resulting hyperosmolarity are one of the most common, and often overlooked, symptoms of DED. Other factors that influence hyperosmolarity include the lipid layer thickness, palpebral aperture width, blink interval, tear film stability, environmental conditions, and body hydration.

When a hyperosmolar tear film is not treated and homeostasis is not reached, epithelial cells recruit inflammatory cells to the ocular surface, exacerbating inflammation and causing reduced expression of glycocalyx mucins. This leads to ocular surface staining and rapid TBUT, as well as apoptosis of surface epithelial cells and loss of goblet cells.

The DEWS II difference

DED has traditionally been divided into primary aqueous deficient dry eye or evaporative dry eye. DEWS II indicates that these are not mutually exclusive. In fact, up to 85% of DED patients who have aqueous deficiency have some degree of meibomian gland dysfunction (MGD) as well.

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Six steps to establishing a Dry Eye Center of Excellence

by Marguerite McDonald, MD

ocular surface disease (OSD) is pervasive, with at least one study showing that 87% of patients scheduled for cataract surgery are diagnosed with dry eye disease (DED). Even mild OSD can lead to suboptimal refractive outcomes after cataract surgery, and the signs and symptoms of OSD are often poorly correlated; therefore, patients with OSD may be missed if clinicians rely heavily on symptomatology for diagnosis.  

Diagnosing DED starts with categorizing patients into asymptomatic or symptomatic categories and performing testing based on their symptom presentation, then further categorizing them and developing a treatment plan based on their place in the spectrum of symptoms.  

DEWS II makes slight changes to the classic definition of tear film. Previously it was described as a three-layer system encompassing the lipid layer, aqueous layer, and mucin layer. In DEWS II it is described as a two-layer system comprising the lipid layer and a mucro-aqueous layer, which contains biomarkers potentially useful in the diagnostic process.  

Conclusion  
DED is a multifactorial disease, and there is no single one size fits all treatment. It must be diagnosed and treated early in an effort to break the cycle of inflammation and restore tear homeostasis.  

References  

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Given these circumstances, cataract surgeons should treat every patient as a dry eye patient. We have two choices: Create a Dry Eye Center of Excellence to ensure optimal outcomes in your surgical patients, or refer your pre-surgical patients to colleagues who will optimize their ocular surface and send them back to you. If you opt to create a Dry Eye Center of Excellence, I recommend the following steps.  

Stepwise strategy  
1. Have a team meeting to let your staff know that focusing on dry eye is important to you and that it will benefit the practice and the patients. Encourage buy-in by explaining that it can be a lucrative endeavor, and the healthier the practice, the greater the likelihood of staff bonuses and pay raises.  
2. Visit another Dry Eye Center of Excellence. Bring your office manager and lead technician along, and take notes.  

Make sure the practice is an hour or more away, so they won’t consider you as competition. Be gracious; buy the staff lunch. You may want to visit again in a year to observe with fresh eyes.

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ASCRS Cornea Clinical Committee algorithm focuses on OSD in preoperative cataract and refractive surgery patients

by Christopher Starr, MD

Achieving a premium surgical outcome is impossible in the absence of a premium ocular surface. The mission of the ASCRS Cornea Clinical Committee’s Preoperative OSD Cataract and Refractive Surgery Algorithm is to aid busy surgeons in identifying and reversing potentially visually significant OSD preoperatively.  

Proactive screening  
While the TFOS DEWS II diagnostic algorithm and treatment rubric are appropriate for the general dry eye disease (DED) and/or ocular surface disease (OSD) patient, there are special considerations required for preoperative refractive surgery patients with OSD. Symptoms are a primary component of most general DED algorithms and are a key driver in obtaining further diagnostic testing. It’s known that a high percentage of preoperative cataract surgery patients are asymptomatic and yet often have fairly advanced visually significant DED and/or OSD. Thus, the ASCRS Cornea Clinical Committee algorithm emphasizes points-of-care testing (osmolality and MMP-9) and other objective testing in addition to a novel surgery-specific symptom questionnaire in all patients during their pre-surgical office visit. If visually significant OSD is detected by the algorithm, refractive measurements and surgery are delayed until it is fully treated.  

Aggressive treatment  
An important commonality between TFOS DEWS II and the ASCRS Cornea Clinical Committee algorithm is that both recommend identifying the DED subtype, evaporative dry eye (EDE) or aqueous deficient dry eye (ADDE) disease, in addition to any other DED masqueraders (e.g., floppy eyelid syndrome, epithelial basement membrane dystrophy, allergy, conjunctivochalasis) to best tailor effective treatment. In TFOS DEWS II, the first step for most patients with DED is low level palliative treatment. Conversely, the ASCRS Cornea Clinical Committee stresses that patients who are having refractive surgical interventions do not have the luxury of time to start slow and increase treatment incrementally as this will lead to unacceptable long surgical delays. When managing preoperative patients with visually significant OSD, the ASCRS Cornea Clinical Committee recommends starting treatment at the TFOS DEWS II Step 2 level with more advanced interventions and to use a multifaceted approach to achieve a more rapid response. A combination of prescription medications (e.g., topical immunomodulators, steroids, antibiotics, etc.) as well as procedural interventions (e.g., blepharostimulation, thermal pulsation, intense pulsed light, punctal plugs, therapeutic contact lenses, etc.) are typically required for rapid reversal of OSD preoperatively.  

Manage expectations  
The ASCRS Cornea Clinical Committee stresses that surgical patients should know in advance that their vision may fluctuate postoperatively and their OSD may worsen, and thus should expect to continue OSD treatment for at least 3–6 months after surgery. The take-home message is: Diagnose early, educate the patient about their OSD, treat aggressively, and maintain appropriate postoperative surveillance and treatment.

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Develop a consistent diagnostic protocol to detect DED

by Alice Epitropoulos, MD

A n unhealthy tear film can lead to unpredictable preoperative measurements, delayed healing, and suboptimal results postoperatively. The pervasiveness of dry eye among preoperative cataract patients and the understanding that hyperosmolar patients have a greater variability in their K readings and IOL power calculations compared to normal osmolar patients demand that cataract surgeons develop a consistent diagnostic protocol to detect DED.

DEd does not discriminate. While we see it prominently in older patients, peri- and post-menopausal women, contact lens wearers, and patients who have had refractive surgery, the widespread use of computers and digital devices has been the great equalizer with respect to exponential growth of DED among all age groups.

Screening is key to identifying symptomatic and asymptomatic patients. The first thing patients do when they walk into my office is fill out a dry eye questionnaire. My technicians are empowered to perform point-of-care testing if patients are symptomatic. This helps to improve my ability to diagnose and treat DED more efficiently as well as to monitor the response to treatment over time.

Improved diagnostics

The point-of-care options that we have at our disposal to evaluate dry eye today are more sensitive and effective than their predecessors. In combination, these tests provide substantial information about the state of the patient’s ocular surface. For instance, tear osmolarity point-of-care testing is a very accurate way to evaluate dry eye severity and therapeutic response. A normal tear osmolarity is considered less than 308 mOsm/L, or if there is an inter-eye difference less than 8 mOsm/L.

Following tear osmolarity testing, our technicians administer an MMP-9 (Inflamm-aDry, Quidel, San Diego) test to detect the presence of matrix metalloproteinase-9. MMP-9 is usually elevated in tears with DED. Another MMP-9 test, Discovery ( TearLab, San Diego), is in the pipeline and expected to be released soon. This new product is beyond testing for the presence of MMP-9, with the potential to provide a quantitative measurement. This will help us both diagnose dry eye and monitor its response to treatment over time.

Meibography is another excellent point-of-care test. It helps to identify patients with meibomian gland dysfunction (MGD) early before there is irreversible damage. The visuals also help facilitate a discussion with the patient about the extent of the disease and the best course of action going forward.

Topography is another essential test that we should be performing especially for our pre-cataract patients. Corneal pathologies such as irregular astigmatism, keratoconus, and epithelial basement membrane dystrophy (EBMD) can be identified via topography, and we can’t always pick up those pathologies via keratometry, biometry, or slit lamp exams.

Although today’s diagnostics are vastly improved in comparison to what we once had, obtaining a thorough history and performing a careful clinical exam remain the hallmark of DED detection. Evaluation of the lids, the lid margins, and the tear film should be incorporated in every patient exam. Evaluating for any preexisting conditions such as EBMD is also a must.

Conclusion

OSD is underdiagnosed and undiagnosed in cataract patients, and it reduces our surgical predictability and can adversely affect our surgical outcomes. Developing a consistent protocol to help identify and diagnose DED will help improve both outcomes and patient satisfaction.

References


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Case study with Alice Epitropoulos, MD

A 68-year-old female presented complaining of fluctuating vision. She is a lead technician on my staff and had hyperopic LASIK 15 years earlier. We are looking at her left eye in this case study, however, her symptoms were binocular. Symptoms included fluctuation in vision, burning, and tearing, with a SPEED score of 20. Exam and testing revealed high tear osmolarity (295/324), abnormal MMP-9, unstable tear film, iropigmented meibomian glands, and significant corneal staining. Corneal topography showed irregular mires and a fair amount of astigmatism.

In cases such as this, I recommend a good quality oral omega-3 supplement to patients and start them on an immunomodulator and pretreat them with a topical steroid for 2 weeks. I also recommended and performed LipiFlow thermal pulsation treatment (Johnson & Johnson Vision, Santa Ana, California). Her symptoms improved, but she had persistent blurred vision with irregular topography. I subsequently initiated neurostimulation three to four times per day.

On follow-up, the patient reported experiencing symptom relief and improved vision lasting for 4–5 hours following neurostimulation. A recheck of her topography after treatment revealed significant reduction in her pseudo-cylinder and improved mires.

OS before OSD treatment

• Power: 46.1 D
• Radius: 7.33 mm
• Steep K: 47.12 D @90
• Flat K: 44.75 D @130
• Astigmatism: 2.37 D
• CIM: 4.45
• Shape factor: 0.79

OS after OSD treatment

• Power: 40.0 D
• Radius: 7.33 mm
• Steep K: 45.00 D @90
• Flat K: 44.37 D @180
• Astigmatism: 0.63 D
• CIM: 0.82
• Shape factor: 0.95

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3. Implement a simple marketing strategy. Have the staff wear buttons on their uniform that say “Ask me about dry eye.” Place posters throughout the practice calling attention to dry eye symptoms and treatment options. Include a sheet along with every invoice announcing your new Dry Eye Center of Excellence. The dry eye product companies are delighted to provide these materials because your success is their success.

4. Have every patient answer a dry eye questionnaire. When a patient responds to every invoice announcing your new Dry Eye Center of Excellence path: You can help your patients who are suffering; you can vastly improve your surgical outcomes; and it can be extremely profitable. For instance, a thermal pulsation treatment (Johnson & Johnson Vision, Santa Ana, California), HD, Germany), LipiView/LipiScan (Johnson & Johnson Vision, and Thermi. She can be contacted at aepitrop@columbus.rr.com.


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References

Taking a diagnostic approach to dysfunctional tear syndrome

by Kenneth Beckman, MD

As the incidence of ocular surface disease (OSD) continues to rise, the array of preoperative tests aimed at identifying the type and extent of OSD is growing as well. Judicious use of these preoperative tests is integral to the CEDARS Dysfunctional Tear Syndrome Algorithm, a practical approach to targeting treatment for OSD that Mark Milner, MD; Jodi Luchs, MD; and I developed on behalf of the Cornea, External Disease, and Refractive Society (CEDARS) Dysfunctional Tear Syndrome Panel.

The foundation for the CEDARS algorithm is the separation of dysfunctional tear syndrome (DTS) into five diagnostic categories. Treatment is based on which category—or categories—best represents the patient’s pathology.

1. Aqueous deficiency
2. Evaporative dry eye based on goblet cell/mucin deficiency
3. Blepharitis/meibomian gland dysfunction (evaporative and non-evaporative)
4. Exposure keratopathy
5. Co-conspirators

The CEDARS algorithm stresses that while these are distinct categories, they frequently overlap. You should aim to identify a specific category and target your treatment for that, with understanding that you may be treating more than one category. The first four categories are self-explanatory. The co-conspirators category refers to pathologies that exacerbate or masquerade as DTS, such as superior limbic keratoconjunctivitis (SLK); medica- mentosa (topical medication toxicity); superficial punctate keratitis of Thygeson; mucous fishing syndrome; contact lens-related toxicity; chemical toxicity, such as hairspray toxicity, often seen in hairdressers; allergic/atopic conjunctivitis; conjunctivochalasis; and floppy eyelid syndrome.

Most of us are familiar with DTS testing methods such as the Tear Osmolarity System (TearLab, San Diego), MMP-9 (InflammaDry, Quidel, San Diego), lipid layer analysis and meibography (LipiView, Johnson & Johnson Vision, Santa Ana, California), and Sjögren’s test (Sjö, Bausch + Lomb, Bridgewater, New Jersey). Of course, there are also traditional methods such as slit lamp examination of the tear meniscus and meibomian gland function, as well as demonstrations of conjunctival staining. The CEDARS algorithm utilizes the information provided by these tests and evaluations to guide treatment.

For instance, in tear osmolarity testing anything under about 300 mOsm/L is considered normal. The CEDARS algorithm ventures beyond the normal/abnormal classifications; anything in the 300–320 mOsm/L range is considered mild, 320–340 mOsm/L is moderate, and greater than 340 mOsm/L is severe. In addition, the difference of 8 mOsm/L or more between the eyes is abnormal. That is why it’s imperative to check both eyes when you do osmolarity testing.

The CEDARS diagnostic tree (Figure 1) is quite useful in honing in on a treatment plan. For instance, with decreased tear production on Schirmer’s test, decreased tear lake, and keratoconjunctivitis sicca pattern staining with fluorescein, rose bengal, or lissamine green, treat for tear deficiency; rapid TBUT, conjunctival scarring, and goblet cell deficiency, treat for goblet cell/mucin deficiency; anterior blepharitis, posterior blepharitis, and lid erythema/debris, treat for blepharitis/MDG; lagophthalmos, poor or partial blink, lid function problems, treat for exposure keratopathy. In the case of goblet cell disease, rapid TBUT—particularly in the absence of lid margin disease and presence of conjunctival staining—could be useful, as not all evaporative dry eye is related to lid margin disease.

Conclusion

Use testing to arrive upon a diagnosis, then use the diagnosis to guide your treatment plan. Keep in mind that you may have more than one diagnosis—they can overlap—and that you should treat the co-existing pathologies in a logical stepwise approach.

Reference


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Case study with Kenneth Beckman, MD

This case supports the critical need to take a careful patient history. A 65-year-old female presented complaining of dry eyes. Her regimen included antihistamines and antidepressants, and she had previously had blepharoplasty.

Examination revealed excellent vision bilaterally, and Schirmer test results measuring basal and reflex tearing were 2 OD and 3 OS after 5 minutes, without anesthesia. The patient had significant MGD with toothpaste-like secretions (Figure 1), and 2 mm of lagophthalmos reflective of earlier blepharoplasty. She had moderate tearing with rose bengal consistent with keratoconjunctivitis sicca, and a tear breakup time of 7 OD and 8 OS, signifying an evaporative component. Technically, this TBUT would be considered low, although in cold climates such as Ohio, where I practice, low TBUT is pervasive. The patient was treated with cyclosporine drops b.i.d. (to help with tear production and inflammation), warm compresses, oral omega-3 supplements, and erythromycin ointment (to help with lid margin disease and with exposure). She noted significant improvement within a few weeks.

Before OSD treatment

- VA: 20/20 OU
- Schirmer: 2 OD; 3 OS after 5 minutes without anesthesia
- Lids:
  - 2+ MGD with “toothpaste-like secretions”
  - 2 mm of lagophthalmos OU
- Conjunctiva: moderate rose bengal staining consistent with keratoconjunctivitis sicca
- Cornea: no fluorescein staining
- TBUT: 7 seconds OD; 8 seconds OS

After OSD treatment

- VA: 20/20 OU
- Lids:
  - Improved secretions
  - Conjunctiva: Staining resolved
  - Cornea: Still no staining
  - TBUT: 9 seconds OD; 9 seconds OS

Figure 1. An example of a patient with MGD with toothpaste-like secretions

Source: Sam Garg, MD