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Seeing the Signs of Dry Eye Disease

Announcer:
You're listening to *On the Frontlines of Dry Eye Disease* on ReachMD. And now, here's your host, Dr. Steve Jackson.

Dr. Jackson:
This is *On the Frontlines of Dry Eye Disease* on ReachMD. I'm Dr. Steve Jackson, and here with me today to explore current challenges and emerging strategies for diagnosing dry eye disease is Dr. John Sheppard. He's a Professor of Ophthalmology at Old Dominion University and Founding and Senior Partner at Virginia Eye Consultants in Norfolk.

Dr. Sheppard, thanks for being here.

Dr. Sheppard:
My pleasure, Dr. Jackson.

Dr. Jackson:
Well, John, let's dive right in. What makes dry eye disease so difficult to recognize and diagnose in clinical practice?

Dr. Sheppard:
First of all, some doctors ignore dry eye because they have other concerns. They're worried about blepharitis; they're worried about getting some cataract surgery done; they're worried about glaucoma. And dry eye becomes a distraction in what should be tagged first in an otherwise very long evaluation.

What a lot of people don't realize is that three-quarters of the patients who present for a cataract operation also have dry eye, and most of them don't know it. Similarly, a lot of dry eye patients have cataracts, and they may come to your clinic complaining of blurred vision when the cause is actually dry eye. Another reason that people don't identify dry eye is that it is a mimicker of so many other conditions, and because it's so common, people tend to ignore it. Another reason is that it's very frustrating to treat, and you have to individualize treatment for that particular patient's presentation of dry eye.

A lot of people come up with other names like ocular surface disease or chronic ocular inflammation or hypotonic tears, and they just add to confusion. That litany of acronyms is best served by DED: dry eye disease. It's not a condition. It's not a nuisance. It's a significant disease process.

Dr. Jackson:
So when dry eye disease is on the differential, what clinical features can help point us toward diagnosis?

Dr. Sheppard:
There's so many things that we can do in the clinic to elucidate the presence of dry eye disease. Symptoms are wide-ranging and often confusing or even contradictory. The classic symptoms are foreign body sensation, loss of visual acuity, and worsening in the evenings. It can be visual fluctuations, good days, bad days, a certain activity that will trigger blurring, foreign body sensation, or another cryptic symptom of dry eye disease.

So symptoms are very important. But they may not be the most actuating feature because some patients are asymptomatic. These patients may have a chronic neurotrophic component, so they look worse than they feel and many of those patients are older. In fact, all of us become more neurotrophic as we age, and that makes me remember many of my octogenarians who have horrific-looking ocular surface conditions under the biomicroscope, and yet they don't complain. They don't complain about dry eye; they don't complain about foreign body sensation or fluctuating vision. They just accept this gradual deterioration in their acuity as a component of aging.

Symptoms are wide-ranging, and they can imitate other diseases. One common symptom is itching, and we might confuse that with allergies. In fact, there's probably 30 million patients in the United States who have concomitant dry eye and allergy, and they may be seasonal. The allergic component may be more prevalent and more dominating in the springtime, of course, when the pollen comes out, and the dry eye component may be more prevalent in the wintertime when the heat is on, which renders the environment very dry.

So we have to look at many clues and look at a variety of symptoms that may bother the patient, and it may simply be reduced reading time. In fact, there's a standardized metric now that we can do in clinical dry eye trials looking at durability of reading time. In other words, if a patient has dry eye and you ask them to read something, they may get 100 words per minute. But if you do the same patient with the same test in the same conditions 20 or 30 minutes later as they have a chance to dry, their reading speed may be significantly reduced, and that's a sign of frustration. People can't read in the evenings. They have trouble staring at the computer, which itself worsens the dry eye.

Another factor, of course, is devices. Computer screens, phone screens, and all the digital devices that we use reduce the blink rate; it's well-established. How many times do we normally blink a minute? Let's say 10. That's a little on the low side. But when someone stares at a computer or their cell phone, whether they're 80 years old or 10 years old, the blink rate is reduced 50 percent, and that's physiologically unacceptable and deleterious. So we see increasing prevalence of dry eye disease in kids, and this reduced blink rate allows for more convective evaporation of the tear film, and it reduces the continuous pressure on the lids which produce meibum from the meibomian glands.

And so now we have two mechanisms for increased evaporative dry eye and tear loss to the environment.

Dr. Jackson:
For those just tuning in, you're listening to *On the Frontlines of Dry Eye Disease* on ReachMD. I'm Dr. Steve Jackson, and I'm speaking with Dr. John Sheppard about how we can better recognize and diagnose dry eye disease.

John, once you've made a diagnosis, how do you determine which type of dry eye disease a patient has?

Dr. Sheppard:
The standard classification, Steve, is that the patient has either aqueous insufficiency, or hypoproductivity of the tears themselves, or evaporative dry eye loss. The majority of patients have an evaporative component, which implies meibomian gland dysfunction. But there's a significant amount of aqueous production insufficiency in virtually all dry eye patients.

An isolated aqueous insufficiency is a relatively rare form of dry eye disease. So our role, of course, is to at least determine the primary component and identify that. My philosophy is to pick one intervention at a time if the patient has complex dry eye and they've been underserved by previous interventions or providers, and analyze that intervention, not next week not in a month, but in three months. Give them three months to use an intervention to really determine if they've improved with that intervention. You're not going to resolve the symptoms acutely. So we have to determine, first of all, whether they have evaporative or aqueous insufficiency dry eye.

But at the same time, there's some low-hanging fruit in so many patients. One of the most common is contact lens use, and a soft contact lens will increase the level of dry eye significantly. Why? Because the lenses gobble up tears. The lenses, by improving comfort, ironically, reduce the blink rate, and the lenses themselves produce hypooxygenation of the ocular surface, which itself produces inflammation and exacerbates the dry eye. If you can get a patient to reduce their contact lens use, not wear them at night, use daily wear disposables, and take a break in the evenings or on the weekend for a whole day, you can improve their dry eye.

Another bit of low-hanging fruit is oral medications, and there's so many that exacerbate dry eye. Generally, they produce hyposecretion. The most notorious are over-the-counter allergy medications, like antihistamines. This muscarinic mechanism reduces the secretion of tears. It also reduces secretions in the lungs and the nose and the throat, which are part of allergy. And we have to differentiate the importance of the allergy itself to the exacerbated dry eye. Another mechanism that we can intervene with is an anti-lipoxygenase oral medication indicated for allergies. It's called montelukast, and that is a drug that we can take without drying or sedation seen with the common antihistamines.

The other notorious oral medications, of course, are sleeping pills, antipsychotics, antihypertensives, and especially diuretic agents that are so important to a patient's general health and yet so bad for their dry eye.

So a simple inventory of oral medications and habits and contact lens use can take care of a lot of patients who have exacerbating or deteriorating dry eye as they age.

The number one cause of neurotrophic dry eye is diabetes mellitus. And because they're neurotrophic, they may not be complaining. So you have some educational challenges. "You're really dry, and the contact lenses that you love and the diabetes are causing potentially

severe problems with your surface epithelium, the ability to see clearly, and the ability to heal." So neurotrophic dry eye is much more common than we think.

Now, severe neurotrophic disease that we see classified by Mackey as stage two and stage three can be blinding, and that is manifested through epithelial defects or stromal melting. But by far the most common stage of neurotrophic ocular surface disease is stage one, and this is seen in diabetes mellitus, contact lens use, and a number of surgical procedures, either at the brain stem or at the ocular surface itself. Cataract surgery is the most common; LASIK is very common. Limbal relaxing incisions disrupt the corneal nerves, and so does, of course, extensive corneal surgery. So these patients may, because of their severed nerves and their disrupted nociceptor function in the cornea, be relatively asymptomatic with horrific ocular surface changes and, therefore, blurring.

So if we can identify neurotrophic dry eye, accelerate the intensity of our dry eye therapy, and maybe directly address the neurotrophic component, we can intervene intelligently. And there's a number of new medicines coming out as well as established medicines for neurotrophic dry eye.

Dr. Jackson:
So looking ahead, where do you see the greatest opportunity to improve how we diagnose and how we classify dry eye disease?

Dr. Sheppard:
The diagnosis requires what we do every day: a good history. What I see forgotten many times is the oral medication history, and asking the patient about what bothers them.

And then the exam is important. We look for staining patterns. Diffuse staining patterns on the cornea with fluorescein may be toxic or related to medications, especially preserved glaucoma medications or self-prescribed over-the-counter medications for dry eye, which may themselves be extremely dangerous. And certainly, patients often get the red out with topical agents that are drying agents in reality. Vasoconstrictors are bad in many ways for the ocular surface. Inferior staining may indicate blepharitis or exposure. The lids don't close. Exposure keratitis, where the eyes are open the most across the horizontal raphe may indicate classic dry eye. Superior keratitis may indicate tarsal disease in the upper lid, superior limbic keratoconjunctivitis, or even chlamydial infection.

And then we look at the lids. We look for meibomian gland dysfunction to determine the intensity and the priority of MGD-induced evaporative dry eye. And one thing that's very often forgotten is that the lids look down; look for Demodex blepharitis. The classic sign, of course, is collarettes. We have a medication, lotilaner, which will control the Demodex for many months or years, with about six to eight weeks of BID topical therapy. And then flip that upper lid. Lots of signs are valuable up there. Look at the vascular thinning or thickening. Look for papillae, which are from irritants from topical medications or allergies. Look for follicles, which indicate a hypersensitivity reaction. And look for scarring. The white substantia propria changes that are permanent in the upper lid indicate chronic inflammatory disease, many times from contact lenses or allergies that are left to continue to torture the patient unabated.

So that's a very important component of the examination that many doctors neglect, or they think about the dry eye at the end of the exam when they have more pressing concerns like macular degeneration, diabetic eye exam, glaucoma, corneal disease, or, of course, cataract.

And then we look at therapeutic responses. The biggest problem I see in a dry eye referral is that the patient didn't use the intervention long enough. This is a chronic disease. That's why I like the three-month follow-up.

And look at the patient's medical history as well because so many systemic conditions can affect the ability to recover from a given level of dry eye or ocular surface disease.

Dr. Jackson:
And with all of those excellent points in mind, I would like to thank my guest, Dr. John Sheppard, for joining me to share his comprehensive and very practical perspective on dry eye disease diagnosis and classification.

Dr. Sheppard, it was great having you on the program.

Dr. Sheppard:
The pleasure is mine.

Announcer:
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