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Seeing Beyond the Symptoms: Early Diagnosis and Treatment of Dry Eye and Meibomian Gland Dysfunction

Announcer Intro:
This is CME on ReachMD! This CME activity, titled "*Seeing Beyond the Symptoms: Early Diagnosis and Treatment of Dry Eye and Meibomian Gland Dysfunction*," is jointly provided by American Academy of CME, Inc and Spire Learning and is supported by an educational grant from supported by an independent medical education grant from Alcon Vision, LLC.

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DR. DOGHRAMJI:
Dry eye disease is one of the most common ophthalmic complaints. It results from the activity of various pathways and is considered a multifactorial disease. An important factor that contributes to the onset of dry eye disease is meibomian gland dysfunction. Meibomian gland dysfunction causes a disruption in the tear film lipid layer, which affects the rate of tear evaporation. This evaporation leads to tear hyperosmolarity, eventually triggering the onset of dry eye disease. Dry disease and meibomian gland dysfunction are strongly associated with each other, such that many of their risk factors, signs and symptoms overlap.

In this educational podcast, we will review the association between dry eye disease and meibomian gland dysfunction, and highlight best practices for assessment, diagnosis, and management.

I'm your host, Dr. Paul Doghramji, and I would like to welcome back our guest, Dr. Eric Donnenfeld. Dr. Donnenfeld is a founding partner of Ophthalmic Consultants of Long Island, and a Clinical Professor of Ophthalmology at New York University. Dr. Donnenfeld thank you for joining us today.

DR. DONNENFELD:
Dr. Doghramji, it's my pleasure to be here. And this is one of my favorite topics we're going to be talking about today. Because this is the most common reason why patients come into a optometrist's or an ophthalmologist's office is they have symptoms of dry eye disease most commonly due to meibomian gland dysfunction. And so I think this is a very important topic for the great majority of eye care professionals.

DR. DOGHRAMJI:
Excellent. Very nice to hear.

So let's go to Chapter One: A Clinical Protocol for Recognition and Diagnosis of Dry Eye Disease and Meibomian Dysfunction. Presentations of dry eye disease are common, and pose a diagnostic challenge due to the variety of symptoms and the absence of certainty for family practitioners.

For example, I have a 52-year-old woman who presented with complaints of intermittent blur in both eyes for two months with her current pair of glasses. She also stated that over the past year, her left eye has been red on occasion. She has no history of allergies,

and is not currently on any medication. So Dr. Donnenfeld, could you briefly describe the signs and symptoms of dry eye disease, or DED, and the role of meibomian gland dysfunction, or MGD.

DR. DONNENFELD:
I'd be very happy to do that. And I just want to focus on the symptom that you presented here that was intermittent blur. We call that visual fluctuation. And visual fluctuation is by far the most common symptom of dry eye disease, and specifically meibomian gland disease. So any patient who complains of vision fluctuating, think of dry eye first. Dry eye is due to either aqueous deficiency or MGD. But most commonly actually both contribute to dry eye disease. MGD is by far the most common cause. And a paper by Michael Lemp, which is kind of a seminal paper on the subject, he found that 86% of patients who had dry eye, had meibomian gland disease. Now, many of them also had aqueous deficiency, but many patients who come to, see us with MGD, have mild symptoms or may be asymptomatic. Um, and a lot of times the symptoms of dry eye don't correlate the severity the ocular surface disturbance. So it's sometimes a harder diagnosis to make because some patients will have symptoms, some will have signs, and they don't correspond all the time.

As I said, in this case, the intermittent blur is a very common symptom of MGD, referred to as visual fluctuations. The definition of dry eye is really multifactorial. I's a disease the ocular surface characterized by a loss of homeostasis of the tear film and accompanied by ocular symptoms in which tear film instability and hyperosmolarity cause inflammation and damage, and neurosensory abnormalities play roles as well.

I divide dry eye into two main categories, evaporative dry eye, which is not enough oils or not enough meibum from the meibomian glands, and aqueous deficiency dry where you don't produce enough aqueous.

Evaporative dry is due to conditions of the island eyelids such as MGD, poor blinking, lid disorders, or that affect the ocular surface such as prolonged contact lens wear. Frequently, people who use topical medications with preservatives will have dry eye disease, and of course, immune related ocular surface disorders such as atopic keratoconjunctivitis plays a role as well.

Aqueous deficiency is a less common cause of dry eye, and is primarily due to dysfunction of the lacrimal gland. The classic dry eye is seen in patients with rheumatoid disease such as Sjogren's syndrome, in which there is lacrimal gland obstruction and adverse systemic therapies. The dry eye that's seen in p – aqueous deficiency dry eye is usually due to T-cell mediated inflammation with damage to the acinar structure of the lacrimal gland. And this can be reversed with proper therapy.

Epidemiological evidence suggests that dry eye is mainly evaporative, but often associated with MGD. Meibomian glands are in the upper and lower lid. They secrete lipids onto the ocular surface, forming the outermost layer of the tear film. And you can actually look at the - at the lids and see these meibomian glands and – and see how they're functioning.

These lipids spread easily. They promote tear film stability. They prevent - protect the ocular surface and they prevent evaporation of tear film.

And MGD is characterized by a diffuse abnormality of the meibomian glands commonly characterized by terminal duct obstruction or qualitative/quantitative changes in the glandular secretion.

I used to say that meibomian gland disease was due to not enough meibum, but it's also not enough quality meibum being secreted. It may result in alteration of the tear film. Many times, patients will have eye irritation, they'll have inflammation, they may have red eyes, and they may complain of just a chronic irritation, which is a disease which can go on for years and years. And many times patients seek treatment and just are not adequately given therapy to resolve their symptoms.

DR. DOGHRAMJI:
Well, thanks, Dr. Donnenfeld. So now can you discuss any risk factors that may facilitate early recognition of dry eye disease or MGD?

DR. DONNENFELD:
Well, there are a lot of conditions that are associated with dry eye. By far, the most common is female gender. Females have a two and a half to three times greater incidence of dry eye due to both aqueous deficiency and meibomian gland disease. It may be due to hormonal changes, such as changes in androgen or estrogen receptors that are present within the meibomian glands. It's also affected by age. So age and female gender are the most common risk factors.

Many times, topical medications can cause dry eye. So we see this with various etiologies, including allergic reactions and inflammatory response from chemical irritation.

I'm a big believer in nutrition. I think one of the reasons we're experiencing an epidemic of dry eye in the United States today is our nutrition is just not as good as it used to be. We just don't have enough omega-3 fatty acids in our diet. The diet has changed

dramatically over the last 50 years as we've become a corn-based society, rather than a green leafy vegetable society. And that plays a very significant role in dry eye disease. And I encourage people with dry to consume omega-3 fatty acids.

Contact lens wear is associated with dry eye and meibomian gland disease. And every year about three to four million patients stop wearing contact lenses because of dry eye disease. An epidemiological studies showed that 50% of contact lens wearers experience dry versus about half of that and non contact lens wearers.

And as I said before, dry eye is strongly associated with aging. By the time you get into your 60s and 70s, the majority of patients have some form of dry eye.

DR. DOGHRAMJI:
So because DED and MGD are common ophthalmic problems, a clear diagnosis is crucial for appropriate management. So Dr. Donnenfeld, could you describe the appropriate tests to diagnose DED and MGD?

DR. DONNENFELD:
Well, appropriate tests are important to diagnosing dry eye disease. And these have been developed by numerous societies, including the Tear Film and Ocular Surface Society had a, uh, dry eye workshop for defining the treatment and diagnostic criteria for dry eye disease.

But I'll start with the very simple statements. And that is the best way to diagnose dry eye is to ask the patients. We give all patients in our practice a dry eye questionnaire that's given by our technicians prior, uh, to our evaluation. There are a lot of good questionnaires that are out there. The SPEED questionnaire, the OSDI questionnaire are all very good. So ask the patient if they're experiencing dry eye. Things like visual fluctuation, irritation, those are symptoms of dry disease.

Then we want to do the - the appropriate testing. And I empower my technicians to order the appropriate tests. Those tests include diagnostic markers such as tear breakup time, supravital staining of the ocular surface, such as lissamine green, rose bengal, and fluorescein. Schirmer's test, which is almost 100 years old, but still has some importance.

And probably the test that I find the most useful is Tear Osmolarity. When the tear become - tear becomes hyperosmotic, the patient will usually experience irritation, foreign body sensation. That's a good marker for dry eye.

Tear breakup time is non-invasive. We put some fluorescein in the eye, we have the patient open their eye wide as they can, and we watch to see how long it takes tear film to break up. A breakup time of less than 10 seconds is considered diagnostic for dry eye disease. And tear breakup time is most generally associated with meibomian gland dysfunction.

Ocular surface stain is performed fluorescein of the cornea and lissamine green and rose bengal for the conjunctiva. What I look for is interpalpebral staining suggestive of dry eye. And that's a very good marker. By the time you give actual fluorescein staining of the cornea, the patient usually has moderate to severe dry eye. That's an easy diagnostic test.

The earlier diagnosis of dry eye can be accomplished with lissamine green and rose bangle, where you can see breakdown of the conjunctiva before the cornea becomes involved.

Schirmer's test was named after Adolf Schirmer, and involves placing a small strip of filter paper inside the lower fornix with the eye closed. It can be done with anesthesia or without anesthesia, and just looking for how much wetting occurs in that filter paper. After about five minutes, the moisture should have traveled down the paper. And usually anything less than 5 millimeters is considered a diagnostic of significant dry eye disease. Sometimes, however, with meibomian gland dysfunction, you can actually have high Schirmer's but low quality tear film.

Tear osmolarity has really changed everything. And that's a calibrated device. And we just touched in tear film, and in osmolarity of greater than 308 milliosmoles is considered diagnostic, but also a difference between the two eyes of greater than 8 milliosmoles is considered diagnostic as well.

If I had to give one clinical pearl for all of you, and that is that when you're diagnosing a patient, when you examine every single patient comes to your office, look at the eyelids. Sometimes we just pass the lids and go right to the tear film, the cornea, and into the eye. Look at the lids, look at altered anatomical features such as meibomian gland dropout, squeeze the lids and look for the plugging or powdering of the gland orifices. And this should be part of a very routine exam. When I squeeze the lids, if a toothpaste material comes out, that's diagnostic of meibomian gland disease. Sometimes nothing will come out, and then you know the meibomian glands have basically just stopped functioning.

In recent years, a new diagnostic entity has entered our office, and that is my meibography. This allows a view of the actual meibomian glands themselves. When you look at a patient's lid, you only see the tip of the iceberg. You see the surface of the meibomian glands.

When you look at meibography, you actually see the glands themselves. And you can look at the structure, dropout, and changes in morphology of these glands, which is really helpful in diagnosing dry eye disease.

If we circle back to your patient, let's talk about the patient that you just presented to us. This is a classic patient, 52 years old, probably perimenopausal, intermittent blur, telling us that vision fluctuates during the course of the day. And she states that her left eye has been red on occasion. So she has inflammation and irritation. She doesn't have any allergies, she's not itching. She's not currently on any medications. So let's look at her physical findings. The basic physical findings you want to look for in every patient is the visual acuity. The visual acuity here is normal at 20/20 and 25. The pupils are reactive, there's no signs of any abnormality. There's no sign of glaucoma, confrontational fields and pressures are both normal. But her ocular surface disease index is 24, which is pretty high. When I examine her lids, her meibomian glands are inspissated. They're clogged. And when I press on the lids, a thick toothpaste-like material appears. This tells me that the meibomian glands are not liquid at room temperature, or body temperature for that matter. And this is a classic finding. The normal meibum should be like an olive oil consistency. It should be a little yellowish, it should be slippery. It shouldn't be sludgy like it is in this case, which means that the meibum has become saturated, fats they're not melting at body temperature, and they're not getting into the tear film. There's 2+ lissamine green staining of the conjunctiva and 1+ inferior corneal staining, and tear breakup time is markedly reduced at 5 seconds in both eyes. And the rest of her exam is essentially normal. So these are very classic findings of dry eye disease most commonly due to meibomian gland disease.

DR. DOGHRAMJI:
For those who are just joining us, this is ReachMD. I'm your host, Dr. Paul Doghramji, and joining me to discuss dry eye disease and meibomian gland dysfunction is Dr. Eric Donnenfeld.

Earlier we spoke a bit about recognition and diagnosis of DED and MGD. Now let's shift our focus to the management of DED and MGD. So, Dr. Donnenfeld, can you share your thoughts on the latest strategies for treating MGD and DED?

DR. DONNENFELD:
Well, basic therapy for meibomian gland disease is diagnosing it first of all. And then once you diagnose it, very simple therapies. Start with things like artificial tears. And a lot of new tears have lipid in them which is very helpful. Hot compresses play a role in melting the meibum, which is saturated fats. And I like oral therapy as well.

But what's really exciting about meibomian gland disease is we have a variety of new devices that have become very helpful in treating meibomian gland disease. And we use heat and pressure or intense pulsed light therapy.

I also want to give a shout out to probably my favorite new therapy, and that's Blepharoexfoliation. We've learned that Blepharoexfoliation is extremely hel - helpful, and that the lid margin contains glyco - glycoproteins that clog the oil glands and cover them. It's exactly analogous to going to the dentist and having your teeth cleaned. When you go have your teeth cleaned, you're having the tartar buildup on your teeth removed. You can't do that with the toothbrush. And it's exactly the same material that grows on the lid margin as well. And it's - this glycoprotein and by using a device called BlephEx, you can actually remove this blocked glycoprotein, opening up the oil glands, and that lets everything else work more effectively. So I very commonly use Blepharoexfoliation then follow up with a thermal pulsation device.

The first device developed to treat meibomian gland disease with thermal pulsation was LipiFlow which is really a very important development in the field of ophthalmology and optometry which, uh, heats the lids up from the inside, and then massage the lids, expressing the oils.

iLux is another great therapy, which is device which has - is handheld and easily portable. Heats the lids up with light, and then the clinician, uh, will massage the lids to express the blocked oil glands.

TearCare, another great therapy.

Uh, and I have all three of these different treatment devices in my office and we use them very commonly. And they really do augment all the other treatments that we give our patients.

Other new treatments that are similar with heat are MiBo Thermaflo and Thermal 1-Touch, which really are very helpful. Uh, intense pulsed light therapy is good for removing some of the vessels, uh, as well.

But I really think the combination of Blepharoexfoliation and thermal pulsation is a great one-two punch for managing a chronic and very irritating disease. And these therapies last about a year. So once you've been a year gives patients a very good effect. And, uh, there have been a variety of studies that have shown how effective these therapies are.

I mentioned topical lubricants like tears, especially with lipids, but immunomodulators are very helpful as well. Corticosteroids play a role

in reducing inflammation. And some of the therapies we use for aqueous deficiency dry, such as topical cyclosporine and topical lifitegrast, which is a topical lymphocyte-1 antagonist, both have anti-inflammatory processes, they're both very effective. And while they're excellent for aqueous deficiency dry eye, also play a role in meibomian gland disease as well.

For patients who have chronic significant disease, the macrolide antibiotics play a significant role, and doxycycline is extremely effective in managing meibomian gland disease.

The biggest error that clinicians make is they give too much. The microbial therapy for doxycycline is 100 milligrams twice a day. But for an anti-inflammatory effect to reduce collagenase, and, uh, open meibomian glands up, 50 milligrams a day is more than enough. And this is what's used very routinely by dermatologists to treat teenagers with acne.

If the patient doesn't want to take an oral macrolide, then a topical medication can be used as well. And that's topical azithromycin, uh, rubbed into the lid margin, which is probably not as effective as the oral therapy but certainly very effective and very easy to use. Biologic tear substitutes play a role. Amniotic membranes, therapeutic contact lenses also are very helpful. As I mentioned before, I love omega-3 three supplements. I think that they play a very significant role. And, uh, using an omega-3 supplement that, uh, has not been contaminated, uh, and has the actual normal omega-3, uh, supplementation I think plays a very significant role as well.

So there's a lot of good therapies for dry eye. A lot of good therapies for meibomian gland disease, but some of these new devices have really made meibomian gland disease a much more effective therapy for managing a chronic disease.

DR. DOGHRAMJI:
Well, Dr. Donnenfeld this would be a good time to circle back to the patient we discussed earlier in the program. So how would you manage this patient?

DR. DONNENFELD:
Well, again, this is a patient who's 52 years old, has intermittent blur that has been going on for months. Her left eye is red. Her OSDI is 24. We looked at her lids. The oil glands were clogged, secretions were thickened, she had staining. She - so she actually had actual damage to the conjunctiva, and a little bit of the cornea as well. This tells me this is not a mild disease. When by the time you get actual corneal damage, this is a moderate disease. So I'm a little bit more aggressive in managing these patients. I would start with very simple hot compresses, oral omega-3 supplementation, and artificial tears. But I would offer, even on the first visit, Blepharoexfoliation and thermal pulsation of her lids. I think that would be a great one-two punch that would be additive of everything that she's doing currently.

I find that most patients don't really use hot compresses effectively. They're annoying to use. They're difficult, they - they're hot, and they become very cool. So using thermal pulsation gives a long-term effect of almost a year, which I think would be very helpful for this patient.

DR. DOGHRAMJI:
All right, so as we wrap up, what are your key takeaway messages to share with the audience?

DR. DONNENFELD:
Well, thank you for asking. This has been a great session. I just really want to emphasize for everyone listening here today, that dry eye is the single most common ophthalmic problem that you're going to encounter in your office. It's often multifactorial. And it's often been ignored by other clinicians. So when a patient comes to see you, you have the opportunity to significantly improve their quality of life.

Meibomian gland dysfunction is an important contributor to dry eye, owing to an imbalance in lipid secretions that affect the rate of evaporation. When tears evaporate quickly, osmolarity increases, resulting in dry eye disease.

And we know that there are many risk factors that contribute to the onset of both dry eye disease and meibomian gland disease. Many of them overlap, but we know female gender, age, and probably nutrition play a very significant role in the epidemic of dry eye that we're experiencing today in the United States.

But the first step in treating dry eye is having a clear diagnosis so you can manage it effectively. Various treatment options are available for dry eye and meibomian gland disease and a stepwise staged approach is often crucial for ensuring appropriate management.

DR. DOGHRAMJI:
Well, with that, I'd like to thank my guest, Dr. Eric Donnenfeld, for joining me to discuss the dry eye disease and meibomian gland dysfunction. Thank you all for being here.

DR. DONNENFELD:
Well, thank you, Dr. Doghramji, I really appreciate being here with you, and I thank you for your expertise. And I will look forward to working with you in the future.

DR. DOGHRAMJI:
Okay.

Announcer Close:
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