Contact Lens Update
CLINICAL INSIGHTS BASED IN CURRENT RESEARCH

Case Report: Multimodal Treatment of Dry Eye to Overcome Contact Lens Discomfort

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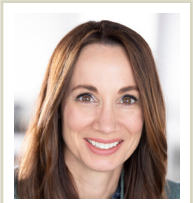

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Background

Contact lens discomfort remains the leading cause of contact lens dropout in long-term contact lens wearers.[1, 2] While most practitioners initial management strategy would be to change the lens material or frequency of replacement to alleviate discomfort and provide longer, more comfortable wearing times, often the discomfort is due to issues with the ocular surface, including dry eye disease, meibomian gland dysfunction, and *demodex* blepharitis.[1] Before focusing on contact lens changes to address comfort issues, it may be more prudent to pause and look deeper at the ocular surface. Sometimes, the real reason for the discomfort is staring us in plain sight. In these cases, utilization of in-office treatments to treat the root cause can be more meaningful for patients and lead to increased wear time and enhanced comfort.[3-5]

History

A 32-year-old female presented to our clinic complaining of limited wear time with her current contact lenses. She reported having to take her lenses out earlier than she desired and had to limit the days she wore them. Her symptoms were worse on the days she was at work, as she spent most of her day on the computer. She was also frustrated with the appearance of her eyes when wearing her lenses because of the conjunctival redness that ensued, which she would often manage with a vasoconstrictor drop and sometimes resort to a steroid drop that had previously been prescribed by another doctor. Her general and ophthalmic history were unremarkable.

The patient's score on the Standard Patient Evaluation of Eye Dryness (SPEED) questionnaire was 14/28. The SPEED questionnaire assesses the frequency and severity of dry eye symptoms: dryness; grittiness or scratchiness; soreness or irritation; and burning or watering.[6] The SPEED questionnaire was validated against the gold standard Ocular Surface Disease Index and mean scores for asymptomatic participants were between 5.00 to 6.25.[7] A higher SPEED score is associated with worse symptoms.

Baseline acuities with spectacles were 20/20 in the right eye (OD) and left eye (OS), and 20/20 at near in both eyes (OU). Extraocular muscles were unrestricted in all gazes and binocular vision testing was within normal limits. Intraocular pressures were 13mmHg OD and 14mmHg OS. Her manifest refraction was OD: -2.25-0.25×175 and OS: -2.50-0.25x005.

The slit lamp examination was positive for eyelash collarettes in both eyes. This cylindrical dandruff is a strong indicator of demodex blepharitis.[8] Bilateral Grade 2 lid margin telangiectasia was present, and meibomian gland expression revealed inspissated glands with only 20% functioning (**Figure 1**). There was grade 2 nasal and temporal bulbar conjunctival injection overlying the insertion of the rectus muscles in both eyes. Fluorescein

tear breakup time (TBUT) was 4.0s OD and 3.5s OS. The *demodex* blepharitis, telangiectasia, meibomian gland disfunction (MGD), reduced TBUT, and conjunctival injection are all indicators of ocular rosacea.[9] Tear meniscus height was normal in both eyes. Osmolarity was 314 mOsm/L OD and 322 mOsm/L OS, which was suggestive of dry eye disease based upon the criteria adapted by the TFOS DEWS II diagnostic algorithm.[10] Inflammadry (Quidel, CA, USA) was positive and equal in both eyes, indicating that ocular surface matrix metalloproteinase 9 (MMP-9) levels were elevated. Tear hyperosmolarity is a component of the inflammatory cascade that results in elevated MMP-9 levels in dry eye.[11] This is because MMP-9 is a proteolytic enzyme produced by stressed epithelial cells.[11] Vital dye of the ocular surface revealed grade 2 bulbar conjunctival staining and trace inferior superficial punctate keratitis in both eyes. The anterior chamber was deep and quiet, crystalline lens clear, and posterior segment unremarkable in both eyes. See Table 1 for a summary of the examination results.

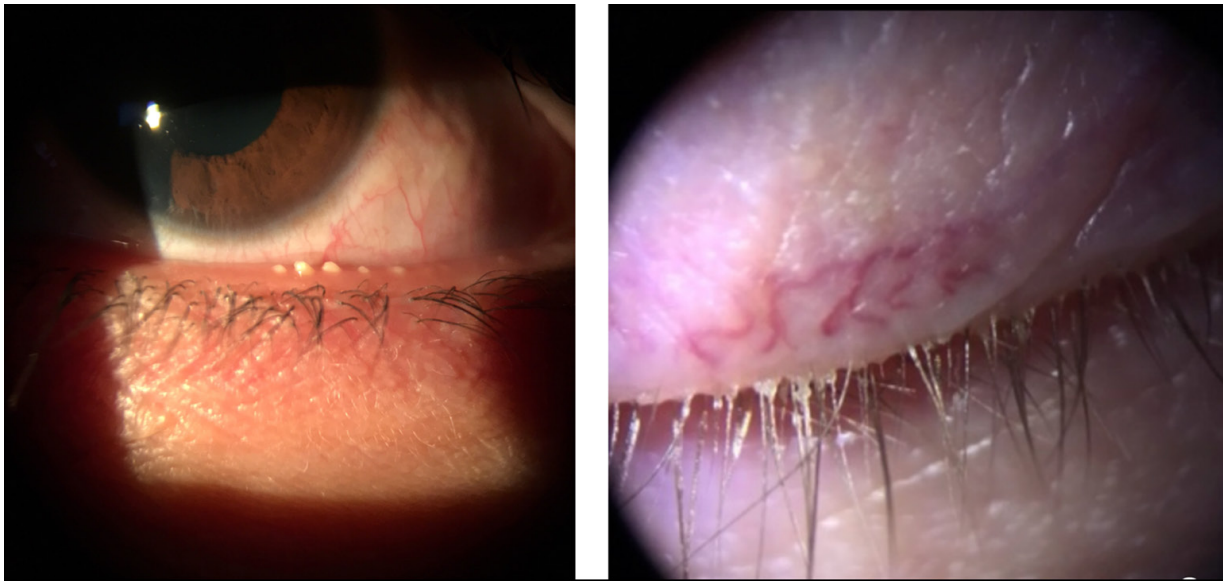

Figure 1: Inspissated meibomian glands, telangiectasia and collarettes.

Table 1: The Dry Eye Workup

Symptoms	
SPEED	14/28
Homeostasis Markers	
Osmolarity	OD: 314 mOsm/L; OS: 322 mOsm/L
Non-Invasive TBUT	OD: 5.4s; OS: 6.2s
Fluorescein TBUT	OD: 4.0s; OS: 3.5s
Ocular Surface Staining	OU: Grade 2 bulbar staining OU: Trace inferior superficial punctate keratitis
Additional Observations	
Anterior Eyelids and Lashes	OU: Grade 2 lid margin telangiectasia; eyelid collarettes and debris
Meibomian Gland Diagnostic Expression	OU: Opaque and inspissated expression
Bulbar Conjunctiva	OU: Grade 2 diffuse temporal injection
Meibography	OU: Grade 2 dropout & distortion lower lid and upper lid

As reports of ocular surface irritation, conjunctivitis, and conjunctival hyperemia, particularly overlying the rectus muscles, have been associated with thyroid eye disease (TED),[12, 13] a full thyroid panel, including thyroid stimulating immunoglobulin, was ordered. The lab results were negative.

Thus, the patient was diagnosed with evaporative dry eye disease, ocular rosacea, *demodex* blepharitis and MGD. As previously mentioned, ocular rosacea is often associated with both *demodex* blepharitis[9] as well as MGD[14-16] and the poor-quality lipid component of the tear film frequently results in evaporative dry eye.[17-19] The patient was educated about the exam findings, prescribed cyclosporine eye drops, and a nutritional supplement for dry eye according to the TFOS DEWS II staged management and treatment recommendations.[17] She was also educated about the negative effects that various ingredients contained in lid cleansers and mascara could have on dry eye disease.[20] Risks, benefits, and questions were answered about lid debridement,[21-23] intense pulsed light (IPL) treatment,[24, 25] along with heat and expression of the meibomian glands.[24, 26, 27] The patient opted to schedule the above series of treatments and was asked to follow-up at the first available time. A detailed summary of the relevant findings, treatment and outcomes is below.

Steps to Treatment

Assessment

The diagnosis of evaporative dry eye disease, *demodex* blepharitis, ocular rosacea and meibomian gland dysfunction was made.

Plan

A treatment approach was taken according to stages one and two of the TFOS DEWS II staged management and treatment recommendations.[17] Firstly, the patient was advised to take a nutritional supplement that has been shown to improve symptoms in dry eye disease (HydroEye, ScienceBased Health, TX, USA).[28] She was educated on the various ingredients in cosmetics, including mascara, that are associated with dry eye.[20] Lid hygiene would be managed using an ocular lid cleanser and hypochlorous acid spray to be used daily at home, and in-office eyelid debridement with ZEST (Zocular, TX, USA). This regimen would help address the *demodex* blepharitis as studies have shown that cleansing wipes and mechanical debridement can reduce bacterial biofilm at the lid margin and clear scruff from the eyelashes.[21-23] Hypochlorous acid is an antimicrobial that can reduce bacterial load on the periocular skin.[29] Medical therapy for dry eye was initiated in the form of cyclosporine A 0.05% ophthalmic emulsion (Restasis, Allergan, CA, USA) twice per day (BID). Cyclosporin A 0.05% is an immunomodulatory agent indicated to increase tear production in patients whose tear production is believed to be suppressed due to dry eye disease.[30] Finally, IPL, as well as heat and gland expression were discussed with the patient. IPL delivers non-coherent light between 500nm – 1200nm and is believed to treat MGD through the following mechanisms: delivery of heat to the meibomian glands which serves to melt and facilitate meibum flow; photocoagulation of telangiectatic vessels, which reduces pro-inflammatory cytokine circulation in the area; reduction of inflammation by reducing *demodex* infestation; and a photomodulatory effect that alters intracellular metabolic activity of the meibomian glands.[24, 25] A combination of heat and pressure applied to the eyelids liquifies meibum and expresses the meibomian glands.[24, 26, 27]

The in-office treatment began with with ZEST debridement and IPL (ICON MaxG, Cynosure, MA, USA). Four sessions were scheduled four weeks apart and then the TearCare (Sight Sciences, CA, USA) device for heat and gland expression would be performed four weeks after the last IPL session. The patient would be started on varenicline nasal spray (Tyrvaya, Oyster Point, NJ, USA) twice per day after the first IPL session. Tyrvaya nasal spray is a cholinergic agonist indicated for the treatment of the signs and symptoms of dry eye disease. It is purported to increase tear production by acting on the nicotinic acetylcholine receptors present on the trigeminal nerve within the nasal cavity and thereby, stimulate the lacrimal functional unit.[31]

Follow-Up

The patient returned ten days later for her ZEST debridement and first IPL treatment. She subsequently returned for three more treatments during the following 16 weeks. Her reported symptoms started to improve after each treatment and the SPEED, osmolarity, and TBUT measures also began to improve.

The final follow-up visit took place one month after the TearCare heat and meibomian gland expression treatment. The patient reported that she was no longer utilizing any type of redness reducing drop and was able to wear her contact lenses on-working days. Her SPEED score improved to 6/28. Meibomian gland expression improved to clear, and her number of functioning glands also increased. Fluorescein TBUT improved to 8.0s OD and 10.0s OS. There was no bulbar conjunctival or corneal staining in either eye. The presence of eyelash debris had reduced and there was no evidence of collarettes.

The patient was advised to return to our clinic in four months for her next follow-up, or sooner should she feel her symptoms return. She was asked to continue HydroEye, varenicline 0.1% BID, cyclosporine 0.05% BID, lid cleanser, acid hypochlorous acid spray. She has continued on one maintenance dose of IPL which is performed every six months. All throughout treatment, and now in long-term management, the patient has been able to continue full-time use of her daily disposable contact lenses. The patient understands that she has a chronic, progressive disease that we will continue to manage together.

Discussion

Ocular rosacea precedes facial rosacea 20% of the time[32] and greater that 50% of those with cutaneous rosacea also have ocular rosacea.[9] TED will often initially present with symptoms and signs of dry eye disease and allergic conjunctivitis.[12, 13] These phenomena highlight the importance of assessing and treating systemic diseases that can initially present in the eyes.

The patient in this case had been suffering from dry eye symptoms for quite some time before she presented to our clinic. Previous practitioners had already changed her to daily disposable contact lenses, yet her symptoms remained. By treating the underlying causes of her dry eye, we were able to improve her SPEED score to a normal level without having to discontinue contact lens wear.

Clinical Pearl

Becoming an astute observer and looking for skin as well as ocular conditions can lead to an early diagnosis of rosacea and *demodex* blepharitis. During slit lamp examination, remind patients to look down so you can properly assess the base of the lashes and surrounding tissues. This intentional step in your assessment can help identify co-morbidities that must be addressed. Often, contact lens discomfort is not due to the actual contact lens but from an underlying issue of the ocular surface. Always utilize in-office procedures, when available, to help patients manage their chronic dry eye conditions.

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