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NOVEMBER/DECEMBER 2017

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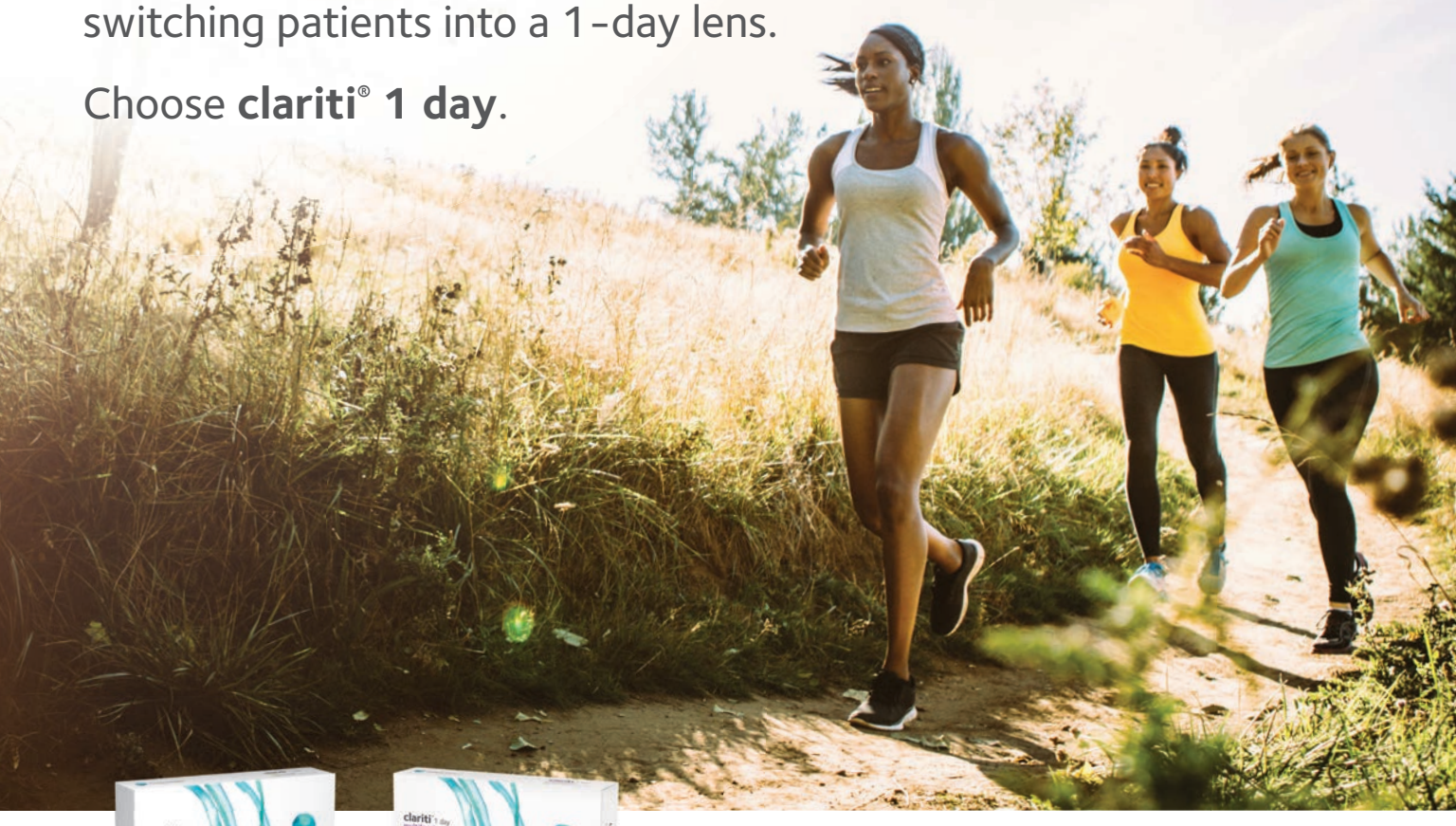

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clariti® 1 day

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IN BRIEF

■ Researchers recently found a benefit to **treating herpetic epithelial keratitis with a cryopreserved amniotic membrane** (CAM) in conjunction with oral acyclovir. Three eyes with dendritic and one with geographic epithelial lesions first underwent epithelial debridement and then amniotic membrane placement. After five (± 3.7) days, all study participants had **significant relief of symptoms, rapid corneal epithelialization, reduced ocular surface inflammation and improved visual acuity**. The patients remained symptom-free on follow up of 2.7 months to 50.8 months. Using CAM allows in-office placement and avoids complications associated with scheduling and performing a surgical procedure, the researchers conclude.

Cheng AMS, Tseng SCG. Self-retained amniotic membrane combined with antiviral therapy for herpetic epithelial keratitis. Cornea. 2017;36(11):1383-6.

■ Starting in 2018, the Centre for Contact Lens Research will become the **Centre for Ocular Research and Education** (CORE). The Center will undergo the name change as it enters its 30th year, citing efforts to work more closely with the optometric profession and improve vision health. Specifically, it plans to expand its scope of focus to include **biosciences, clinical research and education**.

"The new name better reflects what we do," says Lyndon Jones, PhD, CORE's director. "The scope of our work has become larger than just contact lens trials. Now we're much more involved in **dry eye work, drug delivery, microbiology, toxicology and biocompatibility**."

To go along with with the new name, the Center has also announced a new logo, with three colors coming together to represent the roles of biosciences, clinical research and education in CORE's mission. The new brand and logo were unveiled at the American Academy of Optometry's 96th annual meeting in Chicago.

Established at the University of Waterloo's School of Optometry & Vision Science, the Center has been involved in many crucial advances in ocular health and contact lenses, such as the development and testing of silicone hydrogels, as well as studies of contact lens comfort and myopia control.

Centre for Contact Lens Research (CCLR) renamed Centre for Ocular Research & Education (CORE). Business Wire. October 11, 2017. Avaliable at www.businesswire.com/news/home/20171011005022/en/Centre-Contact-Lens-Research-CCLR-Renamed-Centre. Accessed October 23, 2017.

Study Backs Osmolarity Test

A new study recently published in *Cornea* adds to the growing body of evidence suggesting tear film osmolarity is a key indicator for dry eye disease (DED). Researchers looked at three groups of patients—clinically significant dry eye, symptoms-only dry eye and controls—and found the patients without clinically significant, but still symptomatic, dry eye had higher and more variable osmolarity measurements compared with the controls. These study results indicate changes in osmolarity precede clinical findings, the authors conclude, possibly opening doors for better and earlier diagnostics.[1]

The study used the Ocular Surface Disease Index (OSDI) questionnaire to evaluate for dry eye–related symptoms and tested patients for tear osmolarity and other objective measures such as tear break-up time and ocular surface, conjunctival and corneal staining. While it was no surprise patients with clinically significant DED had the highest tear osmolarity (312.0 ± 16.9mOsm/L) and control patients had the lowest (305.6 ± 9.7mOsm/L), the researchers were interested to find 13.5% of symptoms-only dry eye patients had an osmolarity score >320mOsm/L compared with just 2.4% of the controls.[1]

"High tear osmolarity in these individuals may foreshadow significant clinically apparent disease," says Chandra Mickles, OD, MS, an associate professor at Nova Southeastern University College of Optometry. "Routine tear osmolarity testing within the context of the complete clinical picture may foster earlier diagnoses and ultimately improve outcomes."

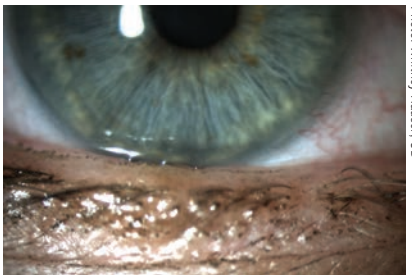

Photo: Whitney Hauser, OD

Patients, such as this one, who are symptomatic but have no clinical signs might still have increased osmolarity, a possible indicator of early DED.

The researchers also found tear film osmolarity was associated with ocular discomfort symptom subscore, corneal staining score, conjunctival staining score, presence of central corneal staining and presence of corneal filaments—further supporting the hypothesis that osmolarity increases and causes ocular surface damage as DED worsens.[1]

DED is notoriously difficult to diagnose properly, given patient symptoms often do not correlate with clinical findings, the study authors said. Researchers continue to hunt for "one single quantitative metric that correlates with both symptoms and clinical findings of dry eye disease"—and many suspect osmolarity may be the answer.[1]

"Although it is clear that hyperosmolarity is at the core of dry eye disease, doctors remain unsure of the utility of tear film osmolarity testing in clinical practice," says Dr. Mickles. "With these study results, doctors can put greater confidence in osmolarity measurement not only for patients with frank dry eye signs, but also for patients exhibiting symptoms without ocular surface staining."

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High-energy Exposure May Cause More Haze Post-CXL

A recent study of corneal density after accelerated corneal collagen crosslinking (A-CXL) found that high-energy exposure may induce more haze in the early post-treatment period for patients with progressive keratoconus.[1]

Researchers from the University of Marmara's Department of Ophthalmology in Turkey surveyed two groups of eyes, 20 in one and 24 in another. The former received A-CXL treatment with continuous ultraviolet-A (UVA) light exposure at 9mW/cm^2 for 10 minutes adding up to a total energy dose of 5.4 J/cm^2, and the latter received A-CXL with continuous UVA light exposure at 30mW/cm^2 for four minutes and a total energy dose of 7.2 J/cm^2.

Follow-up included corneal densitometry measurement, which is used to determine corneal transparency after refractive surgery and to monitor CXL's effect. Researchers used Scheimpflug tomography to track corneal haze at one, three, six and 12 months post-treatment.

At the one-month follow-up, corneal density peaked in both groups. In the group treated with 9mW/cm^2 A-CXL, these results plateaued until six months post-treatment, at which point they significantly decreased. The 30mW/cm^2 group experienced a more gradual decrease in corneal densitometry throughout the follow-up. However, the mean change in density at the one- and three-month follow-ups was higher in the 30mW/cm^2 A-CXL group than in the 9mW/cm^2 A-CXL group, marking a possible correlation between higher-energy exposure and early postoperative haze.

Additionally, the corneal density of both groups returned to baseline after one year. According to the authors, this suggests the haze was transient rather than permanent. Researchers know that A-CXL affects corneal transparency, but these results show the change is reversible, at least in the first year, they note.

While the groups were initially treated with Vigamox drops (moxifloxacin hydrochloride 0.5%, Alcon), Lotemax drops (loteprednol etabonate 0.5%; Bausch + Lomb) and Refresh artificial tears (Allergan) for a brief period after undergoing A-CXL, the study notes that topical corticosteroids do not affect the course of postoperative transient haze.[2]

This study provides a look at the short-term effect of high-energy exposure on A-CXL, but it doesn't address it in the long-term. The researchers view the one-year follow-up period as a limitation and would like to see future studies explore longer post-treatment periods. RCCL

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By Joseph P. Shovlin, OD

Gut Instinct

New research details the relationship between microbiota and the eye. Here's what you should know.

If you were to pick up any professional medical journal today, you would likely find a discussion on microbiota's relationship with diseases and their pathogenicity. In many parts of the body—including the eye—a normal community of bacteria and micro-organisms helps confer resistance to infection.[1-6] Literature suggests the gastrointestinal (GI) microbiome is associated with host health status and its structure and composition define functional gene expression, pathogen abundance and overall host response.

A recent investigation of oral microbiota shows a possible connection to glaucoma. Patients who have glaucoma seem to have higher quantities of oral bacteria than controls without glaucoma.[7]

STRIKING A BALANCE

A significant body of evidence suggests GI microbiota may define states of health and disease in a complex manner. Strategies to improve host health, including the ocular health, may rely heavily on manipulating the delicate balance of this ecosystem.[1] Specifically, the host community must provide balance between maintaining a favorable environment while protecting against invasion or outgrowth of pathogenic species of microbes.[1] Prenatal and postnatal exposures allow the immune system to develop the ability to discriminate between harmful and beneficial microbial species.[1,2,5]

Recent studies have provided evidence of a link between early GI colonization events in those under a year of age and subsequent development of allergic disease.[1] Microbiome deviations at an early age may be associated with immune disease. Studies also show that antibiotic use in infants could change the gut microbiota, which may adversely impact the immune system and increase the risk of atopy in specific groups of children.[5]

Disruption of the microbiome in the adult gut is also associated with many diseases and appears to be characteristic of various chronic inflammatory diseases.[1] The use of antibiotics has raised concerns for the potential adverse affects on gut microbiota by impacting the native microbial community.[1] Introducing pathogens orally through diet provides individual host and micorobiota variability, directly affecting the dynamic ecosystem.[2]

Host resistance to certain infections may be altered by commensal organisms prior to infection occurrence. In herpes simplex, for example, microbiota may enhance, reduce or have no substantive effect on a viral infection, depending on the host response.[2] The influences on an infection may be by direct mechanisms such as virion modification, or indirect mechanisms such as host cell modification.[1,2,4] Conversely, microbial products can indirectly enhance viral replication and herpetic disease.[2]

Probiotics and prebiotics represent some promise for inflammatory disease management by promoting subsets of existing GI bacterial community members capable of degrading and increasing the production of anti-inflammatory compounds.[6]

OCULAR SURFACE STABILITY

Conjunctival diversity of bacteria is actually greater than that of the skin, and the direct impact of contact lens wear on the ecosystem is yet to determined. Host commensal bacteria of the ocular surface interacts with the host immunity to suppress microbial pathogenicity. Manipulation of the microbiome through pro-, pre- and symbiotic supplementation may prove an attractive alternative for improving host health status that might specifically include the eye.[1,4]

Leveraging the role of our microbiota in infections to alter the course of certain infectious diseases may be a reality someday. However, it will require a better mechanistic understanding of microbiota function and a more comprehensive knowledge of how microbes affect resistance and susceptibility to various pathogens.[1,2,4] Will we someday prescribe specific novel supplementation for contact lens wearers to help safeguard against infection or help control inflammation? Perhaps, because human biology and health are inter-related due to the influences of our microbial inhabitants. RCCL

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†Based on a clinical study with AIR OPTIX® AQUA, AIR OPTIX® for Astigmatism, and AIR OPTIX® AQUA Multifocal contact lenses.

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Important information for AIR OPTIX® COLORS (lotrafilcon B) contact lenses: For daily wear only for near/far-sightedness. Contact lenses, even if worn for cosmetic reasons, are prescription medical devices that must only be worn under the prescription, direction and supervision of an eye care professional. Serious eye health problems may occur as a result of sharing contact lenses. Although rare, serious eye problems can develop while wearing contact lenses. Side effects like discomfort, mild burning or stinging may occur. To help avoid these problems, patients must follow the wear and replacement schedule and the lens care instructions provided by their eye doctor.

Important information for AIR OPTIX® NIGHT & DAY® AQUA (lotrafilcon A) contact lenses: Indicated for vision correction for daily wear (worn only while awake) or extended wear (worn while awake and asleep) for up to 30 nights. Relevant Warnings: A corneal ulcer may develop rapidly and cause eye pain, redness or blurry vision as it progresses. If left untreated, a scar, and in rare cases loss of vision, may result. The risk of serious problems is greater for extended wear vs. daily wear and smoking increases this risk. A one-year post-market study found 0.18% (18 out of 10,000) of wearers developed a severe corneal infection, with 0.04% (4 out of 10,000) of wearers experiencing a permanent reduction in vision by two or more rows of letters on an eye chart. Relevant Precautions: Not everyone can wear for 30 nights. Approximately 80% of wearers can wear the lenses for extended wear. About two-thirds of wearers achieve the full 30 nights continuous wear. Side Effects: In clinical trials, approximately 3-5% of wearers experience at least one episode of infiltrative keratitis, a localized inflammation of the cornea which may be accompanied by mild to severe pain and may require the use of antibiotic eye drops for up to one week. Other less serious side effects were conjunctivitis, lid irritation or lens discomfort including dryness, mild burning or stinging. Contraindications: Contact lenses should not be worn if you have: eye infection or inflammation (redness and/or swelling); eye disease, injury or dryness that interferes with contact lens wear; systemic disease that may be affected by or impact lens wear; certain allergic conditions or using certain medications (ex. some eye medications). Additional Information: Lenses should be replaced every month. If removed before then, lenses should be cleaned and disinfected before wearing again. Always follow the eye care professional's recommended lens wear, care and replacement schedule. Consult package insert for complete information, available without charge by calling (800) 241-5999 or go to myalcon.com.

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By Robert Ensley, OD, and Heidi Miller, OD

Managing Corneal GP Complications

Become a troubleshooting master and your GP lens patients will reap the benefits.

The advent of soft disposable contact lenses permanently altered the contact lens landscape, resulting in the decline of corneal gas permeable (GP) lens fitting. GP lenses are increasingly relegated to patients with complex prescriptions or high vision demands, and specialty designs such as custom soft toric, hybrid and scleral lenses are now widely available and steadily growing in popularity. Consequently, corneal GP lenses are often overlooked as a first choice.

Thanks to today's technology, complications related to contact lens overwear and poor lens-to-cornea alignment can be identified early on to help avoid permanent corneal damage. It is important to be aware of common GP lens complications and understand how to troubleshoot in order to maintain good corneal health and lasting comfort.

CORNEAL STAINING

Epithelial punctate staining can occur during GP lens wear for several reasons. Peripheral corneal desiccation, particularly 3 o'clock and 9 o'clock staining, pertains to the area of the cornea not adequately resurfaced with tears (*Figure 1*). This can occur due to the contact lens fit, incomplete blink or the patient's lid positioning.[1]

If the lens has excessive peripheral edge lift or a particularly thick lens edge, the amount of tears swabbing the corneal surface is minimized due to the gap between the eyelids and cornea. This lid gap can also be caused by a low-riding lens or an incomplete blink. The same peripheral staining can occur if the lens has inadequate peripheral edge lift resulting in insufficient lens movement and minimal tear exchange.

If not treated, the areas of desiccation can lead to coalesced punctate staining and, eventually, corneal thinning resulting in ulceration, neovascularization and scarring.[1] The area of reversible peripheral thinning is referred to as a dellen (*Figure 2*). Patients may be asymptomatic in mild cases. In more severe cases, patients may complain of dryness, displacement of contact lenses, redness, lens awareness, light sensitivity and reduced lens wear time.[1]

Since 3 o'clock and 9 o'clock staining is often related to contact lens alignment, centration and movement are critical. If a lens is decentered inferiorly, reducing the center thickness or using a lenticulated design may help minimize weight.[2] Flattening the base curve of a lens with excessive clearance will help with centration. Sometimes a larger diameter lens may be required for centering, but this may need to be coupled with thinning center thickness and reducing sagittal depth to avoid weighing down the lens.

If edge clearance appears excessive when examining with fluorescein and a cobalt blue filter, a steeper peripheral curve radius or a narrower peripheral curve width may be indicated. Also, if there appears to be lens adherence, the case may call for a flatter peripheral curve radius or a wider peripheral curve width. If the lens edge is excessively thick, consider thinning it to provide a closer lid-to-cornea alignment.[2]

Selecting a lens material with good wetting properties can help address corneal staining. Frequent lubrication with rewetting drops may also be beneficial and necessary to maintain optimal corneal health. If the peripheral corneal desiccation is related to partial or incomplete blinking, the patient may benefit from blinking exercises in conjunction with rewetting drops. Ultimately, if changing the lens fit and incorporating adjunct therapies does not resolve the corneal staining, the patient should be advised to minimize contact lens wear time or consider switching to other modalities such as piggy back lenses, soft contact lenses or scleral GPs.

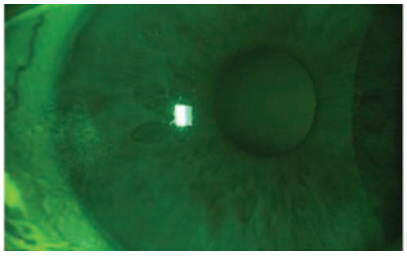
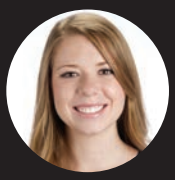


Fig. 1. An example of 3 o'clock and 9 o'clock staining.

VASCULARIZED LIMBAL KERATITIS

A related condition known as vascularized limbal keratitis (VLK) occurs when corneal desiccation is further compromised by peripheral seal off of the lens edge.[3] These lesions will show superficial staining. More advanced cases can result in vascularization, staining and an elevated opacified region.[3] Patients may experience a significant decrease in lens wear comfort, a decrease in wear time and significant awareness of the VLK lesion.

VLK can be easily mistaken for corneal neovascularization, corneal dellen and phlyctenulosis.[3] Corneal neovascularization usually

takes longer to resolve than VLK, however, and is more rare in GP lens wear.[3] Unlike VLK, corneal dellen will pool with fluorescein instillation but will not stain. Additionally, topography reveals a corneal depression in cases of corneal dellen instead of VLK's staining elevation. VLK can be differentiated from phlyctenulosis by how quickly it regresses—VLK can regress in a few days, while phlyctenulosis will often persist for up to two weeks.[3]

Treating VLK revolves around eliminating the cause of desiccation and is usually related to lens fit or contact lens materials. If the lens fit is too tight, flattening the peripheral curves and base curve radius can be beneficial. Decreasing the diameter of the lens can also help eliminate a steep fitting lens.[2] Reducing lens wear time and frequent lubrication is advised to eliminate peripheral desiccation. In more severe cases with corneal thinning or vascularization, practitioners should consider corticosteroid and antibiotic-steroid combination drops.[4]

CORNEAL WARPAGE

Both GP lenses and thick, stiff soft lenses can cause corneal warpage. Often, patients are unaware of the problem and continue to wear their lenses for extended periods of time.[2] Before the advent of corneal topography, corneal warpage was generally described as a condition that included distorted keratometer mires with or without irregular astigmatism and reduced vision on post-lens wear refraction.[5] Because of the increased use of topography, however, the definition of corneal warpage now includes central irregular astigmatism with a loss of radial asymmetry (superior flattening and inferior steepening), and a reversal of the normal flattening corneal contour.[5]

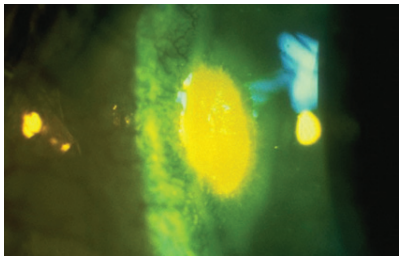


Fig. 2. Corneal dellen with fluorescein pooling.

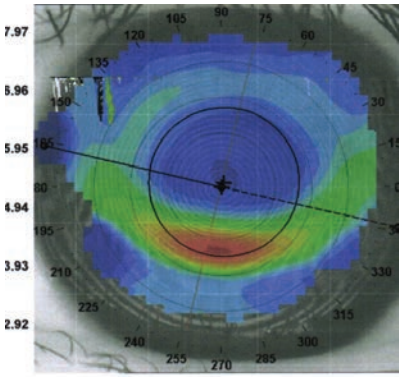


Fig. 3: Corneal warpage due to an inferiorly decentered contact lens.

One cause of corneal warpage is an excessively steep base curve radius. This causes the lens to suction onto the cornea, creating an irregular corneal surface. To combat this, practitioners can flatten the lens base curve and peripheral curves to provide adequate tear exchange and lens movement.

Another cause is spherical GP lens wear in patients with 2.5D or more of corneal astigmatism. By not fitting a patient in an appropriate back surface toric design, topographical changes can occur over time.[6]

If a lens is riding high from a flat fitting lens, superior flattening of the cornea can occur with a steeper contour inferiorly. Depending on the degree of corneal molding, this can be misdiagnosed as keratoconus (*Figure 3*).[5] In these cases, it is important to limit contact lens wear for a few weeks and retake imaging to ensure repeatability of findings.

In all corneal warpage cases, patients tend to rely on contact lenses to see clearly and experience spectacle blur.[7] Irregular astigmatism may be evident on corneal topography and irregular findings on manifest refraction. Patients may complain of halos or distorted vision that is more evident with glasses. To resolve this, practitioners should instruct patients to take a break from contact lens wear and refit them starting with baseline corneal findings.

It is important to identify corneal staining, VLK and corneal warpage early in order to provide lasting GP lens comfort and avoid long-term corneal damage. Working with GP lenses may appear daunting at times, but understanding simple troubleshooting techniques will allow for continued success. RCCL

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Treating Herpes Simplex Virus

Round out your HSV know-how by mastering its treatment.

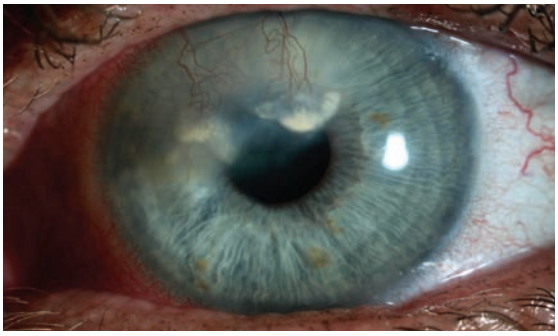
The previous 2017 editions of this column have focused on the wide variety of presentations of herpes simplex virus (HSV) keratitis. While it's almost time to turn the page on HSV keratitis, no discussion would be complete without outlining treatment considerations.

GENERAL APPROACH

During the infectious dendritic stage of HSV keratitis, treatment is based on reducing viral replication and spread using antivirals. Trifluridine was the mainstay antiviral for years. Like acyclovir, trifluridine is a nucleoside analog that interferes with cellular metabolism of virally-infected cells. However, trifluridine is poorly selective for virally-infected cells and is too toxic for systemic dosing. As a rule of thumb, if an anti-infective medication exists only topically, it is probably a sign of toxicity with systemic dosing. Even when only used topically, trifluridine can cause epithelial keratitis with frequent or prolonged use.

Zirgan (ganciclovir 0.15%, Bausch + Lomb) is the only guanosine analog topically available in the United States. This helps practitioners achieve effective antiviral dosing topically with less tolerance issues than trifluridine. Also, varicella's susceptibility to Zirgan has opened up a topical treatment option for herpes zoster ophthalmicus.

Clinically, I've noticed a tendency for practitioners to favor topical antivirals when dealing with HSV. Most think that if the problem is on the eye, dosing the medication directly is helpful. While the mindset that topical anti-infectives are superior to systemically dosed medications is accurate in some situations, such as with bacterial keratitis, it may not be the case in HSV. Meta-analysis of the limited research shows orals and topicals may be equivalent in dendritic HSV.[1] I'll usually lean towards topicals in cases of dendritic keratitis; however, when cost considerations come up, I do feel comfortable using oral medications as front-line therapy when dealing with any type of HSV keratitis. However, severe cases of infectious epitheliopathy or necrotizing stromal disease may call for both oral and topical treatment.



This patient has heavy vascularization and lipid deposition secondary to herpetic keratitis.

Corticosteroid use alone in inflammatory HSV manifestations increases the likelihood of recurrent infectious epithelial disease and further perpetuation of the herpetic cycle. As such, prophylactic dosing with antivirals is required any time a corticosteroid is used. When using a topical antiviral paired with a topical corticosteroid, drop-for-drop prophylaxis dosing is sufficiently conservative in most cases. When using an oral-topical pairing, oral acyclovir 400mg QID will suffice. As with all things, there is no magic dosage that will prevent all flare-ups, and even with both oral and topical antiviral prophylaxis, recurrences are possible.[2]

For long-term maintenance, oral prophylaxis of antivirals was established with the HEDS study, where it was shown to reduce recurrence of stromal disease by 50% in those with a history of stromal disease. Reports of resistance exist, but most cases of acyclovir resistance in HSV have occurred in immune-compromised populations, and resolution with acyclovir is still more common than resistance, even in cases where isolates have low susceptibility.[3] Despite the HEDS-established protocol, effective dosing may vary. For most, the 400mg BID suggested dosing will be sufficient. However, some cases may require titrating upwards to reduce flare-ups, and some may require less frequent dosing.

EXCEPTIONS

While oral acyclovir and its derivatives are the mainstays of HSV keratitis treatment, oral antivirals should be limited or even avoided in some cases. Because these medications are excreted through the kidneys, predictable tissue concentrations are dependent on normal kidney function. Patients with renal failure or failing kidney transplants require altered dosing based on their renal function and weight. In this group, the medication is not excreted at typical rates, so it can systemically build

up to extreme levels with standard dosing, leading to side effects. In these cases, practitioners can call the patient's nephrologist and ask for "kidney-adjusted dosing guidelines." An easier approach to avoid the renal consideration for acute disease is to use topicals.

ODs should avoid using topical corticosteroids when dealing with any infectious epithelial episode due to the risk of worsening the episode and increasing the likelihood of recurrence and stromal involvement.[4] During episodes of herpes stromal keratitis (HSK), herpes endotheliitis and herpetic iritis, however, topical corticosteroids can be used safely and effectively. In some HSV keratitis cases, chronic topical corticosteroids may be required to keep inflammation under control.

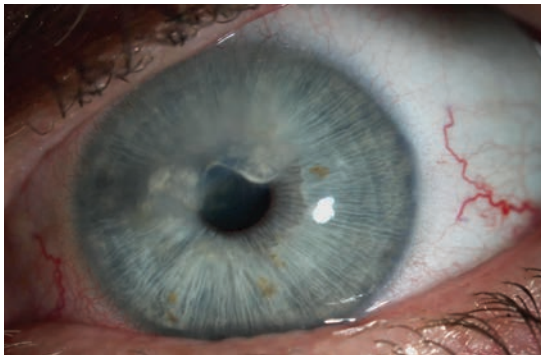
VISION LOSS

Scarring occurs for the roughly 40,000 cases of severe HSV-related vision loss encountered each year, and other treatment options are required.[5]

First, the two most common ways to remove a scar—transplants for deep scars and phototherapeutic keratectomy (PTK) for superficial scars—carry risk of recurrence. Corneal excimer laser application is also a risk factor for reactivation, which may limit the appeal of PTK. In both cases, high-dose prophylaxis with antivirals leading up to surgery and continuing well into the postoperative period, combined with a quiescent pre-surgical period of several months, is advised.

Vascularization associated with herpetic scars further complicates treatment. PTK is not performed on vascularized corneas, and heavily vascularized scars carry a significant risk for rejection with transplants. Surgeons can limit most risk of rejection by performing deep anterior lamellar keratoplasty instead of PTK, but revascularization of the graft remains a concern.

When vascularization is a primary concern, bevacizumab may be dosed topically or intrastromally to reduce perfusion, but any well-established vessels are likely to re-perfuse with time. No herpetic scar is a perfect candidate for scar removal; however, assuming the surgeon is willing and the patient understands the risks, scar removal may be the only option for visual rehabilitation.



At one month post-treatment, vessels are still present but markedly less perfused. This will likely require repeat treatments to maintain effect.

For patients with recurrent or severe endotheliitis, the cumulative stress will reduce the patient's endothelial cell count and may ultimately create a decompensated corneal endothelium that requires an endothelial transplant. As with other HSV surgical interventions, heavy pre- and post-treatment antivirals are warranted in these cases.

PREVENTION

Vaccination for HSV is clinically unavailable. Promising animal models for both viral fragment and attenuated virus vaccines exist, and a human trial with a viral fragment (with middling results) has also been published—but in all cases only a small sample of research actually assesses ocular response. HSK may be a form of autoimmunity turned on by original ocular infection, and it is unknown how a vaccination, which may be preventative or therapeutic, could interact with this immune arc. In either event, it will likely be some time before a vaccination is available.[6]

This concludes our discussion on HSV. My hope is that you will always consider the possibility you are dealing with HSV for any unilateral keratitis you encounter, and also that you will be comfortable with the treatment options available. From a sheer numbers perspective, HSV is the most critical corneal infectious pathology to understand. RCCL

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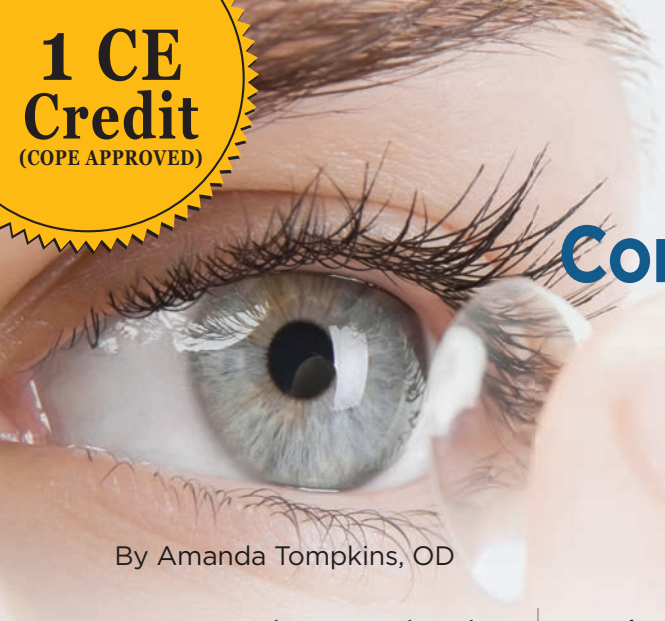

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Contact Lens-associated Red Eye: Causes and Corrections

These cases can be challenging, but they don't always have to be.

By Amanda Tompkins, OD

Red eyes are relatively common in practice but not always straightforward, and adding a contact lens to the ocular surface only complicates things. To avoid potentially vision-threatening scenarios, practitioners need to come up with a timely, appropriate diagnosis and treatment plan. This article discusses how to streamline these processes for contact lens-related red eyes to achieve the best possible outcome.

CASE HISTORY AND EVALUATION

Obtaining a thorough history for the patient right away should help steer the case in the right direction. The first step is to establish an appropriate list of differentials. Asking certain questions can help eliminate the most concerning etiologies and rule out several differentials. Some examples include:

- Can you pinpoint when this started?
- What is your wear schedule?
- What solution do you use?
- Do you have any medical conditions?
- Compared with yesterday, are your symptoms better, worse or about the same?
- How old is your lens storage case?

It is also crucial to know if a case of red eye started while the patient was wearing lenses, if there has been any recent change in environment such as swimming with lenses or being out of the country, if discomfort or pain increases upon lens removal and if there is pain, discomfort or itching in general. For the last point, patients will often say yes to all three. In those cases, practitioners can get them to prioritize their symptoms by asking: "If you could only tell me *one* thing that is bothering you, what would it be?"

Slit lamp observation. This is where working diagnoses are either confirmed or denied. When possible, these observations should be made with the patient's lens on. This will help determine if the red eye is from the lens itself or another etiology. From there, things to look for include lens deposits and defects, whether the lens is inside out and whether it fits properly.

Next is evaluation of the eyelashes, lid margins and tear film quality over the lens. This is a good time to look for any debris trapped under or on top of the lens and take note if there is a chance of poor hygiene with cosmetics or creams. A comprehensive eyelid examination should always include eversion of the lids to determine the presence of follicles or papillae over the tarsal plate.

After evaluation with the lens on, lens removal will allow for accurate analysis of the cornea and conjunctiva. From here, practitioners should look at the limbus for neovascularization. At this point, a corneal evaluation with and without staining is necessary. Before staining, it is important to complete a detailed corneal scan with both parallel pipette and optic sections with full illumination, when possible. Practitioners should evaluate each layer of the cornea to determine which are affected and if any edema or whitening of the cornea is present. Some conditions present with symptoms greater than signs and some may have very subtle signs. Another area in need of close inspection is the anterior chamber. The location, depth and size of any corneal deficiencies or defects will be helpful later, so it is prudent to take detailed notes.

DIFFERENTIAL DIAGNOSES AND ETIOLOGIES

Corneal infiltrates can be one of the most diagnostically challenging anterior segment conditions practitioners have to deal with.

ABOUT THE AUTHORS

Dr. Tompkins is an instructor at The Eye Center at Southern College of Optometry. Her professional interests are in primary care, glaucoma, diabetes, clinical and didactic education as well as local and international medical mission work.

Their presentations and etiologies include both inflammatory and infective processes.

Inflammatory. Contact lens-induced acute red eye (CLARE) occurs in the presence of corneal hypoxia combined with noninvasive gram-negative bacteria that elicit an inflammatory reaction secondary to bacterial endotoxin.[1] No actual corneal infection exists in this case. Symptoms include discomfort, contact lens intolerance and possibly mild pain. One key diagnostic feature is that lens removal relieves symptoms.

CLARE presentation can be unilateral or bilateral and consists of mild to moderate conjunctival hyperemia with associated corneal infiltrates located in the periphery to midperiphery. The infiltrates have no associated epithelial defect, distinguishing CLARE from both contact lens-induced peripheral ulcer (CLPU) and microbial keratitis (MK).[2]

Risk factors for CLARE include extended wear with poor replacement schedule, deficient lens and storage environment hygiene. Of note, *H. influenza*, a gram-negative bacteria, can colonize in the nasopharynx and lead to CLARE in patients who have recently been sick with the flu.[3]

In CLPU, inflammation occurs as the body responds to bacterial exotoxins from the colonization of gram-positive bacteria, namely, *Staphylococcus* species, on the surface of the contact lens.[4]

CLPU presentations are generally mild. In some cases, the condition may go undetected by the patient, and the clinician will observe the inactive infiltrate during routine evaluation. When symptomatic, patients may present with discomfort, foreign body sensation and tearing unilaterally.

Often, CLPU cases are associated with an epithelial defect, but Bowman's layer remains intact so there is minimal spillover of fluorescein into the surrounding stroma. Another key feature of CLPU is that the epithelial defect is usually considerably smaller that the stromal infiltrate and located in the mid-periphery or periphery. The biggest differential with CLPU is MK. CLPU is most similar to a very early stage of MK. Unlike MK, CLPU is typically not associated with photophobia, conjunctival hyperemia or anterior chamber reaction.[5]

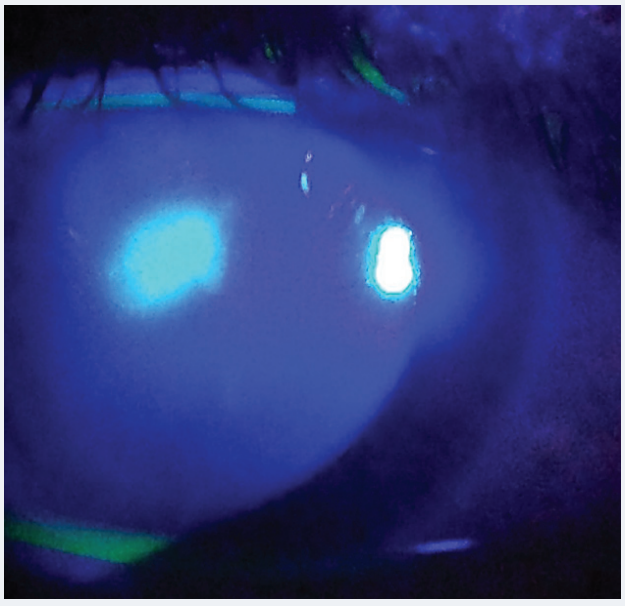

Photo: Dan Fuller, OD

In this MK infection, fluorescein is "spilling" into the surrounding stroma due to excavation of Bowman's layer, the relative size of the ulcer and the central location.

Risk factors for CLPU include sleeping in lenses, prolonged replacement schedule and poor hygiene.

Contact lens-induced papillary conjunctivitis (CLPC) is included in discussions of giant papillary conjunctivitis (GPC), allergic conjunctivitis and vernal conjunctivitis.[6] The pathophysiology of CLPC has been debated and conflicting theories still exist.[7,8]

For this discussion, we will consider CLPC primarily to be an inflammatory condition initiated by protein deposits on the lens surface with an associated mechanical component.[9] The deposition of protein on the lens

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Goal Statement: Contact lens-associated red eyes are among the most challenging presentations seen in everyday practice. However, with a detailed working knowledge of differentials, a thorough examination and swiftly established treatment plan, practitioners can confidently reach an accurate diagnosis and achieve the best possible outcome for their patients and ensure future success with contact lens wear.

Faculty/Editorial Board: Amanda Tompkins, OD

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surface depends on water content of the lens (higher water content means more deposition), as well as polymer content, structure and charge.[1,10,11] Studies have documented that lenses with both high water content and ionic properties have the highest amount of protein deposits.[12] The palpebral conjunctiva undergoes an inflammatory response mediated by a type I hypersensitivity reaction with immunoglobulin E components and a type IV hypersensitivity reaction in response to the presence of these proteins.[13]

Additionally, the movement of the tarsal plate over the edge of the lens contributes to repeated trauma of the tarsal conjunctiva. This results in a papillary reaction mediated by several inflammatory factors.

CLPC symptoms include lens intolerance and awareness, decreased wear time, blurred vision and an urge to excessively clean the lenses due to discharge.[14] Signs include bilateral, large palpebral papillae on the upper palpebral conjunctiva, mucous discharge and excessive lens movement on inspection.[7] Patients with CLPC may report symptoms are worse upon removal of the lens. The age of the contact lenses, frequency of replacement and length of extended wear period, as well as cleaning and case hygiene habits, contribute to increased risk of CLPC.

Contact lens-related hypersensitivity reactions related to lens care systems, drops being used or lens material (a silicone hydrogel combination with certain solutions) are another common source of contact lens-related red eyes.[15] These patients often present with bilateral discomfort and hyperemia. Presentation is also characterized by diffuse superficial punctate keratitis.

Determining the source requires specific questioning regarding the pattern of their symptoms, recent changes in products they are using and an increase in artificial tear usage. Additionally, some clinicians suggest changing one variable at a time if the cause is unknown. Lens removal and copious amounts of preservative-free artificial tears will help alleviate symptoms and reduce superficial punctate keratitis (SPK) initially, but determining and eliminating the source is necessary for a long-term solution.

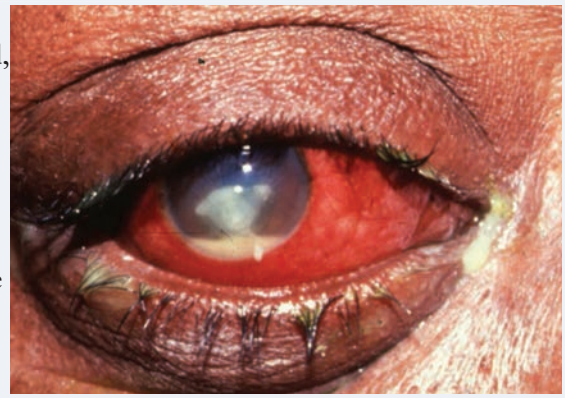
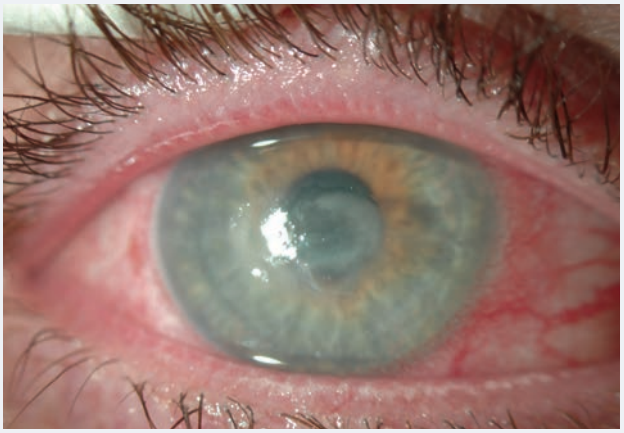


Photo: Elmer Tu, MD

This case of MK was caused by *Pseudomonas aeruginosa*. Note the large central ulcer and gross hyperemia.



Here is an example of *Acanthamoeba* keratitis with deep stromal disease.

Infectious. MK is less common but more severe than sterile etiologies for contact lens-associated red eyes, so it must be considered as well. It is helpful to assume a case is microbial until proven otherwise. Contact lens-related infectious ulcers are typically caused by bacteria, fungi or protozoa.[5]

Bacterial MK is often caused by *Pseudomonas aeruginosa*.[7,16] In early stages, the condition may mimic CLPU both in signs and symptoms, but quickly progresses. Patients with MK typically present with unilateral pain, photophobia and reduced vision, and a history of poor hygiene and contact lens over-wear.

Signs include anterior chamber reaction and moderate to severe conjunctival hyperemia. Ulcers are generally located centrally and associated with an epithelial defect that is characteristically 1:1 with the stromal infiltrate, larger than 2.0mm and irregular in shape. Because of a break in Bowman's layer, instilled fluorescein usually spreads through the stroma beyond the stromal haze. There may also be satellite lesions surrounding the ulcer, which are absent in CLPU.[5] Complicating differentiation from CLPU, both CLPU and MK ulcers can present in the midperiphery, and early MK ulcers are not always 1:1 with the stromal infiltrate.

The culprits in fungal infections typically include *Aspergillus*, *Candida* or *Fusarium* species.[7,8] Classically, these infections are

associated with an injury involving vegetable matter, but can also be associated with contact lens wear.

Patients often present with unilateral pain, photophobia and reduced vision. They may say their symptoms have been gradually increasing over the last several days to weeks. This is secondary to the slow rate of fungal growth, differing from the acute nature of bacterial infections.

The characteristic sign of a fungal infection is a central corneal infiltrate with "feathery" borders and associated satellite infiltrates.[17] As with bacterial keratitis, an epithelial defect may be present over the stroma, but here, the epithelium may be elevated over the infiltrate, possibly intact.

On the protozoan front, *Acanthamoeba* must always be ruled out due to its refractory course and risk for permanent vision loss with these infections.[7,8] Risk factors for this infection include poor contact lens hygiene from tap water use or exposure to hot tubs, swimming pools or other sources of contaminated water.[7,18]

Classically, these patients present with symptoms greater than signs. They usually show up at the office as a result of severe ocular pain and photophobia. Signs include conjunctival redness, dendritiform lesions resembling herpes simplex virus on the epithelium of the cornea in the early stage and a characteristic "ring" infiltrate in more advanced cases.[5]

TREATMENT AND MANAGEMENT

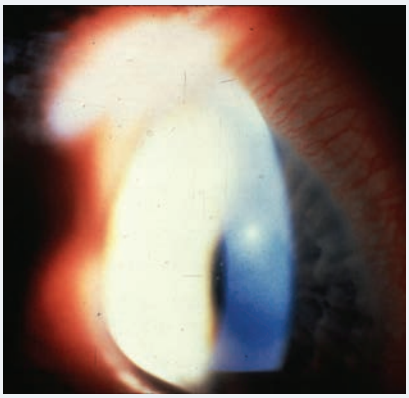
Once an accurate diagnosis is made, focus can shift to developing a treatment plan:

Inflammatory. With all inflammatory etiologies, removing the inflammatory trigger is the first step in treatment. This requires discontinuing contact lens wear until signs and symptoms are resolved. Often, removing the offending agent is sufficient for relief of symptoms, but expediting the process and returning patients to a comfortable state as quickly as possible is a good way to provide reassurance. The addition of an artificial tear will help aid in comfort and return the ocular surface to physiological equilibrium. For CLARE and CLPC, adding a topical steroid such as prednisolone acetate 1% can be helpful (in the absence of epithelial defects). While dosage varies with the severity of the condition, QID is often a good starting point.

Considering CLPU, it's best to assume these cases are MK until proven otherwise and treat with a fourth-generation topical fluoroquinolone. Generally, these cases do not need a loading dose, and TID dosing should suffice. Practitioners should see these patients the next day and on a 24-hour basis until there is resolution of the epithelial defect, at which point adding a steroid will reduce residual scarring. Alternatively, a topical combination drop would work as initial treatment.

For patients who are symptomatic for itching in cases of CLPC, a topical mast cell stabilizer (MCS) or MCS/antihistamine combination is necessary. If the patient's initial treatment includes a steroid, practitioners should avoid the addition of a MCS/antihistamine combination at first because of the anti-inflammatory effects of the steroid. In these cases, a combination drop can be added later and is often beneficial when used as prophylactic treatment for recurrent cases.

In all inflammatory cases of red eye, it is important to continuously follow up with patients and make sure they are not wearing their lenses until symptoms have subsided. At that point, they can resume lens wear on a titrated schedule. In the case of CLPC, papillae may take months to resolve. Here, a daily wear option may prevent recurrence, long-term. In cases where daily disposable lenses are not an option, a hydrogen peroxide-based care system with emphasis on zero tolerance for extended wear or extended replacement schedule will greatly reduce the risk of recurrence.



A mid-peripheral corneal ulcer caused by *Staphylococcus aureus*.

Infectious. For MK, broad-spectrum, fourth-generation fluoroquinolones such as gatifloxacin, besifloxacin and moxifloxacin are the best options for treatment.[19,20] These offer the most broad-spectrum antibiotic properties and are valuable in cases when the organism is unknown. Practitioners should consider a loading dose for all suspected bacterial keratitis cases.[21] Loading dosages vary but can be anywhere from every 15 minutes for the first hour up to every five minutes for the first 30 minutes depending on severity of the ulcer. To determine the organism, culturing and fortification of antibiotics may be required. Additionally, referral to a corneal specialist may be required in severe, visually threatening cases.[22]

Fugal keratitis is notoriously difficult to treat, and the fact that only one commercially available topical antifungal agent, natamycin, exists complicates the treatment plan. Others are available, but in recent studies natamycin provided significant improvement over fortified voriconazole.[11] It is prudent to begin treatment once a fungal etiology is suspected, but referring to a corneal specialist for comanagement can also be a helpful option in these cases.[23]

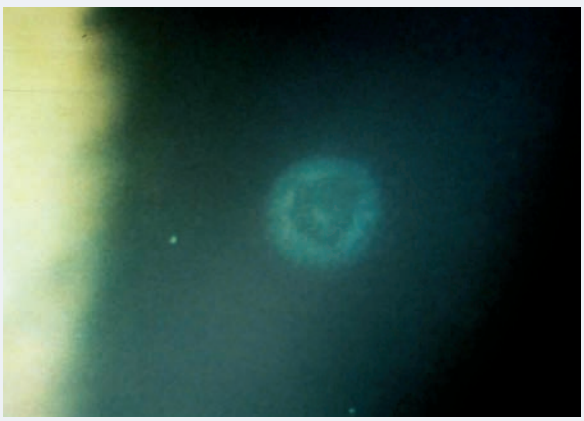
Acanthamoeba is a protozoa, so it is not successfully treated with antibiotics. Because of this, and given the organism's aggressive nature, antiseptics are necessary.[24] The most commonly used antiseptics are polyhexamethylene biguanide and chlorhexidine, both of which have proven effective in clinical trials.[18,25]

After initiating treatment for any of these conditions, practitioners should follow up with patients the next day and then every one to two days until there is consistent improvement. A cautious taper of medication is recommended to best prevent recurrent infection. Fortified topical antibiotics, such as tobramycin and vancomycin, and fortified antifungals, such as voriconazole, may be necessary in the case of large or central ulcers or those not responding to already aggressive use of antimicrobial medication. Again, comanagement is often helpful in these cases.

WHEN TO CULTURE

Typically, when dealing with an ulcer that is small or in the periphery empirical treatment with broad-spectrum antibiotics is sufficient to yield stabilization and resolution of the infection. However, for lesions that either don't respond to empirical treatment, are greater than 2mm in size or are in the central cornea, culturing is indicated. Additionally, infiltrates suspected to be secondary to an unusual organism, such as nosocomial infections or those originating from vegetative trauma, should be cultured so the appropriate treatment can be initiated as quickly as possible.

Contact lens-associated red eyes are among the most challenging presentations seen in everyday practice, and the wide spectrum of etiologies only complicates diagnosis and treatment. However, with a strong working knowledge of the differentials, a detailed examination and swiftly initiated treatment, practitioners can confidently reach an accurate diagnosis and achieve the best possible outcome, securing a successful future of contact lens wear for patients. RCCL



A healed CLPU several months after initial onset.

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CE TEST ~ NOVEMBER 2017

1. **The primary cause for CLARE is:**
   a. A primary infection that leads to secondary inflammation.
   b. A secondary infection due to a primary inflammatory condition.
   c. Gram-positive exotoxins causing an infection.
   d. Gram-negative endotoxins eliciting an immune reaction.

2. **Which of the following microbial keratitis infectious organisms is a protozoan?**
   a. *Pseudomonas aeruginosa.*
   b. *Acanthamoeba.*
   c. *Aspergillus.*
   d. *Moraxella catarrhalis.*

3. **When evaluating a contact lens-associated red eye at initial examination it is important to:**
   a. Remove the lens as soon as possible to prevent further injury.
   b. Leave the lens in place for evaluation, then remove.
   c. Immediately evaluate the eye with slit lamp examination.
   d. Only evaluate the anterior segment with the lens removed.

4. **Which of the following presents with an associated corneal ulcer?**
   a. CLARE.
   b. CLPU.
   c. CLPC.
   d. Hypersensitivity reaction.

5. **When treating a suspected *Pseudomonas aeruginosa* infection, the best treatment approach includes:**
   a. Fortified voriconazole.
   b. Copious artificial tears.
   c. Prednisolone acetate 1% QID.
   d. A fourth-generation fluoroquinolone.

6. **Which of the following would be most appropriate in the long-term management of a patient suffering from chronic CLPC?**
   a. Switching to a daily disposable lens modality.
   b. Initiation of copious lubrication.
   c. Chronic low-dose steroid use.
   d. Administering an anti-histamine/mast cell stabilizer.

7. **Which of the following may present with a dendritiform lesion?**
   a. *Acanthameoba.*
   b. *Candida.*
   c. *H. influenza.*
   d. *Staphylococcus.*

8. **A mid-peripheral ulcer with an associated stromal infiltrate in a 1:4 diameter ratio is likely caused by:**
   a. A gram-positive bacteria.
   b. Filamentous fungus.
   c. A protozoan.
   d. Endotoxins.

9. **A patient presents with a bilateral conjunctival hyperemia and diffuse SPK. Your treatment includes:**
   a. Once-daily gel at night.
   b. Initially eliminating all possible causes.
   c. Copious artificial tears.
   d. Prophylactic antibiotic.

10. **In which of the following scenarios would culturing be most appropriate?**
    a. Peripheral infiltrate with no associated epithelial defect.
    b. Mid-peripheral ulcer slowly resolving with a fourth-generation fluoroquinolone.
    c. Initial presentation of a feathery infiltrate obtained after organic foreign body.
    d. Bilateral diffuse SPK with severe conjunctival injection.

EXAMINATION ANSWER SHEET

Contact Lens-associated Red Eye: Causes and Corrections

Valid for credit through November 15, 2020

Online: This exam can also be taken online at www.reviewofoptometry.com/ce. Upon passing the exam, you can view your results immediately. You can also view your test history at any time from the website.

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Answers to CE exam:

1.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	4.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	8.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
2.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	5.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	9.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
3.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	6.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	10.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
					7.	Ⓐ	Ⓑ	Ⓒ	Ⓓ					

Post-activity evaluation questions:

Rate how well the activity supported your achievement of these learning objectives:

1=Poor, 2=Fair, 3=Neutral, 4=Good, 5=Excellent

11. Better able to obtain a thorough patient history in possible CLARE cases.	①	②	③	④	⑤
12. Better understand how to streamline the CLARE diagnosis process.	①	②	③	④	⑤
13. Better able to identify and initiate effective treatment options for CLARE.	①	②	③	④	⑤
14. Increase my knowledge of presentations and etiologies of corneal infiltrates.	①	②	③	④	⑤
15. Increase my skill in differentiating between treatment options for inflammatory and infectious etiologies.	①	②	③	④	⑤
16. Improve my ability to determine when CLARE cases require culturing.	①	②	③	④	⑤

Rate the quality of the material provided:

1=Strongly disagree, 2=Somewhat disagree, 3=Neutral, 4=Somewhat agree, 5=Strongly agree

17. The content was evidence-based.	①	②	③	④	⑤
18. The content was balanced and free of bias.	①	②	③	④	⑤
19. The presentation was clear and effective.	①	②	③	④	⑤

20. Additional comments on this course:

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By submitting this answer sheet, I certify that I have read the lesson in its entirety and completed the self-assessment exam personally based on the material presented. I have not obtained the answers to this exam by fraudulent or improper means.

Signature: _____ Date: _____

Please retain a copy for your records. LESSON 115666, RO-RCCL-1117

Managing Microbial Keratitis

Early identification and treatment is crucial when dealing with these infections.

By Aaron Bronner, OD

Infectious keratitis is a prevalent source of vision loss. While data from the 1990s report incidence as 30,000 cases per year in the United States, a newer study suggests this number has more than doubled.[1,2] A small but significant percentage of these eyes go on to require corneal transplantation; between 2% and 6% will require an urgent transplant and an even higher percentage will require surgery to ameliorate resultant scars.[3,4] A smaller number, perhaps as high 1.8% of ulcers seen at academic centers, go on to require enucleation or evisceration.[5]

Because of the potential for severe vision loss, microbial ulcer management requires critical thinking at nearly all junctures, as well as careful and thoughtful follow-up. Based on the severity of infection upon diagnosis, the degree of virulence of the particular microbe and patient-specific features, corneal infections can sometimes progress despite aggressive and appropriate intervention. The good news, however, is that when these infections are identified and treated early, the odds of a favorable outcome are much greater.

WHEN TO CULTURE

The first and most critical step in the management of microbial keratitis is determining whether the infiltrate is infectious. Some key features in making this distinction are a supportive risk factor (e.g., contact lens wear), a dense, round or oval infiltrate with ulceration (meaning there is an epithelial defect), overall infiltrate size larger than a pinhead and, with increasing suspicion, chances are heightened the more central the lesion is located.[6] Prior to the initiation of any therapy, practitioners should decide whether to culture in house, refer for culturing or pursue a purely empiric treatment.

For full disclosure, I culture every ulcer that I *believe* is infectious. I find this to be appropriate for several reasons. First, culturing takes practice, and even in the hands of an experienced clinician, it only yields results between 38% and 72% of the time.[7,8] In this regard, practice makes perfect, or at least better. Of course, you don't practice on a patient just to enhance an unnecessary skill, which brings me to my next point: Although culturing removes only a tiny amount of material, removal of infectious material should be a positive thing, at least from a therapeutic standpoint—though this remains unproven. Finally, culturing adds both clinical and medical-legal heft to your treatment decisions. While inappropriate treatment does not dramatically reduce culture yield, you only get one "best chance," and that is prior to initiating treatment.[8-10]

Even ineffective therapy reduces culture yield, and in these eyes the future—like the infected cornea—is opaque. You can't look at a series of ulcers and predict with good accuracy which ones will not respond to therapy. Having the culture results in hand if things deteriorate can prove invaluable. Ignoring my own clinical practice patterns, not all presumed infectious ulcers in community clinics need culturing. In fact, most ulcers respond well to empiric commercially available therapy. However, ulcers that are larger than 2mm, central or paracentral in location, have an unusual appearance or characteristics and an unusual history such as trauma should be cultured or referred for culturing. Also, if you do intend to refer for culturing within 24 hours, you are better off not initiating antibiotic therapy.

CLINICAL DIFFERENTIATION

Regardless of whether or not you culture, nearly all initial treatment

ABOUT THE AUTHOR

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begins empirically, as culture results take one to seven days to process.[11] Establishing effective initial treatment is perhaps the most critical step in setting the stage for a positive outcome. Research shows that patients who fail on incorrect initial empiric therapy have a markedly higher rate of keratoplasty and a significantly greater cost of therapy, so planning should be approached with careful consideration.[7] The managing physician should assess the most likely source of infection based on history, clinical presentation and susceptibility of pathogens. The first consideration here is the likely infectious etiology. Certainly no one would prescribe antibiotics for a fungal infection, so some level of differentiation is necessary.

Beyond general differentiation, distinguishing between the varied bacterial sources of infection is also a key part of managing corneal ulcers. First, you need to consider the influence your geographic location plays. Almost all etiologic reviews of microbial keratitis agree there is tremendous variability in causative pathogens based on location. A study out of Paraguay showed a 79% incidence of *Staphylococcus* etiology, while another from Australia had only a 17% incidence.[12] This trend is mirrored regionally in different zones in the United States. Regardless of history such as contact lens use, *Pseudomonas aeruginos*a contributes to a relatively higher percentage of cases of corneal ulceration in the southeast United States than in more temperate or desert climates.[2,10,13] An increased incidence of fungal ulcers exists in tropical climates, and in the southeast United States, this results in rates of fungal keratitis that double or triple those seen than more temperate zones.[14-16] Geographic differences don't play a role in the clinical appearance of the ulcers, but those practicing in Florida, for example, need to have a higher index of suspicion for fungal ulcers than someone in the southwest United States.

Next in the process of differentiating ulcers is attending to specific risk factors. First of all, age plays a role in which form to suspect. Gram-negative ulcers affect young patients much more frequently than those over age 60, where gram-positive etiologies predominate.[16] It's also been well established that gram-negative species, particularly *Pseudomonas*, have a higher association with contact lens use, whereas postoperative corneal infections and those associated with ocular surface disease are most typically caused by *Staph*. species.[10,16-19] Beyond the widely understood link to fungal infections, trauma also carries the risk for atypical bacterial infections such as nontuberculous *Mycobacterium* and *Nocardia*.[18]

Pathogens that heavily contribute to the normal flora will infect the eye in an opportunistic fashion, so in compromised corneas, *Staph*. species, as dominant organisms of the normal flora, would predominate. *Pseudomonas* is a minor part of the ocular flora, so it requires a delivery vehicle, in this case a contact lens, to be inoculated onto the eye. Fungal species and atypical bacteria are usually absent from the eye and also require some inoculating media, most often in the form of environmental or organic trauma. As a result, ulcers with histories such as organic trauma almost always require initial culturing regardless of clinical appearance. Fungal and atypical ulcers have a dramatically worse prognosis than the more typical etiologies.[8,10]

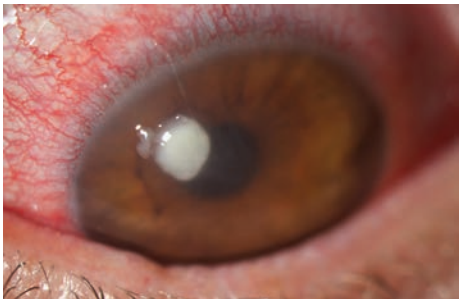

Fig. 1. Here is a look at a typical *Staphylococcus* ulcer. Note its uniform density and well-defined borders. This organism was resistant to fluoroquinolones but responded well to dual fortified agents.

Despite this, history doesn't guarantee presence of any etiology, and a large number of contact lens-related ulcers will be the result of *Staph*. species.[17,20] Risk factors evolve over time and while trauma once was the primary risk factor for fungal keratitis in the United States, contact lens use now probably outstrips it as the top association. Because of this, the final clue guiding clinical suspicion should be the ulcer's appearance.[21-23]

The ulcer's clinical appearance must be mined for hints as to what organism needs combating. *Staph*. species typically have a dry, discrete infiltrate. These may be paired with hypopyon and have surrounding edema. *Pseudomonas* ulcers, in contrast, are much wetter and mucousy in appearance and look like they could be debrided relatively easily. These ulcers may also have a somewhat whirled, non-uniform density, whereas *Staph*. ulcers tend to be more uniform throughout.

Fungal ulcers can give a varied clinical appearance. A significant percentage of fungal infiltrates may display characteristic features such as feathery margins or satellite infiltrates; over half will

have non-specific features, leading to misdiagnosis of the vast majority of fungal ulcers by as much as 87% in one review.[20] Knowing this, fungal infection has to remain on the differential for essentially all corneal ulcers until a positive response to antibiotics occurs.

TREATMENT

Once a clinical differential has been made, focus should shift to therapy. It's becoming more important to use critical thinking to differentiate not only among fungal bacterial and amoebic ulcers, but also among likely etiologies of bacterial microbes when selecting initial therapy.

Twenty years ago, we were practicing in an era of relatively effective monotherapy. Fluoroquinolones were widely effective against most ocular pathogens, and as the class has evolved, their coverage improved.[24] This was the norm among community practices. Empiric treatment with a single fluoroquinolone was successful approximately 90% of the time—even in eyes with demonstrated *in vitro* resistance.[25] Currently, however, as antibiotic resistance to fluoroquinolones continues to expand among gram-positive pathogens, this treatment strategy can be expected to fail more frequently.

Although widespread resistance to fluoroquinolones has essentially been limited to *Staph.* species, and causative etiologies vary wildly depending on geographic location, we can use published data to get a general idea of how frequently the ulcers we run into will be resistant to fluoroquinolones. In the United States, between 10% and 25% of all corneal ulcers should be expected to be resistant to gatifloxacin and moxifloxacin.[1-11,13,14,24,26] If the majority of practitioners who manage corneal ulcers are using a single fourth-generation fluoroquinolone, we have to expect this treatment to fail at levels nearing 25%. Considering the fact that inappropriate initial therapy leads patients to greatly increased risk of needing keratoplasty and a much higher cost of therapy, this is a cause for concern.

Some will wonder about besifloxacin for several reasons. First, it has no systemic equivalent, meaning it should in theory reduce resistance from systemic dosing. The chlorofloroquinolone has a somewhat different molecular structure than gatifloxacin and moxifloxin, theoretically meaning it should reduce the cross-resistance common in fluorquinolones.[14] Additionally, the ARMOR study found the agent to have nearly as potent a minimum inhibitory concentration (MIC) against methicillin-resistant *Staphylococcus aureus* (MRSA) as vancomycin.[27]

Unfortunately, for those of us in community clinics, there is no way to prove any of this information is still valid. The difficulty with besifloxacin starts with it being an ophthalmic-only preparation. Because of this, it is not part of standard MIC testing done with culture and sensitivity testing (a systemically derived test), and most community microbiology labs are unable to provide the same information for it. Therefore, besifloxacin has to be used with blind trust assuming it will work. In our clinic at least, besifloxacin, which is still our go-to commercially available agent, has not performed any better than moxifloxacin or gatifloxacin when faced with fourth-generation fluoroquinolone resistant bugs (as determined by MIC testing).

Still, fluoroquinolones perform very well against nearly all common non-*Staph.* bacterial pathogens and are reasonable forms of monotherapy in most cases. This is especially true for the most common non-*Staph.* cause of bacterial keratitis, *Pseudomonas*, which has extremely low rates of resistance (although it is actually more susceptible to older generation agents).[6,10-15,24,26-29] Considering the overall rate of resistance to fourth-generation fluoroquinolones driven by expanding MRSA and methicillin-resistant *Staphylococcus epidermidis* (MRSE) species, and good coverage of fluoroquinolones against most other ophthalmic pathogens, it's fair to say we are in a bit of a bind. If you are clinically unable to differentiate between "likely" *Staph.* ulcers and "likely" non-*Staph.* ulcers, monotherapy with a fourth-generation fluoroquinolone runs a relatively high risk of encountering resistance and failing initially, therefore increasing the risk of a poor outcome.

Because of all this, I don't believe monotherapy is currently the best practice for management of corneal ulcers. Instead, treating all presumed bacterial but undifferentiated ulcers with

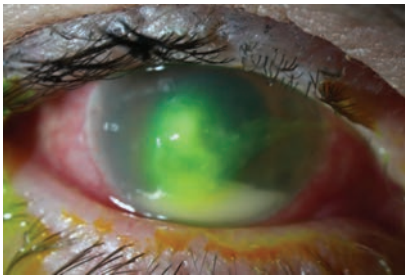

Fig. 2. In contrast to the *Staphylococcal* ulcer in *Figure 1*, this mid-stage *Pseudomonas aeruginosa* ulcer is more mucousy in appearance and slightly whirled. The large hypopyon is not necessarily suggestive of etiology, but more a sign of the inflammatory response's intensity.

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commercially available dual therapy would be wise. Dual-agent treatment may seem unwieldy or undesirable, but this shouldn't be the case. We simply need to look to the example of how cornea clinics have managed ulcers all along—with dual broad-spectrum fortified agents—to realize that commercially available dual therapy redirects our practice patterns back towards what is done at the highest levels.

When selecting agents, I still believe that a fourth-generation fluoroquinolone is the most reasonable starting point for therapy. This could be paired with one of the older commercially available agents that performed well against MRSA and MRSE isolates in the Ocular TRUST 2 study: Polytrim (trimethoprim/polymyxin B sulfate, Allergan) solution, tobramycin solution 0.3%, gentamicin solution 0.3% or bacitracin ophthalmic ointment. With this combination, practitioners should encounter less treatment failures due to resistance. As is the case with any corneal ulcer, initial dosing should be aggressive. With dual agents, I apply the loading dose over 15 minutes and send the patient off with instructions to use both medications every hour—one drop on the hour and the other on the half hour—throughout the day. I also recommend that the patient wake up every two to three hours throughout the night to administer the drops until improvement is noted.

From here, close follow-up is necessary to ensure treatment success, and any unexpected change for the worse should be met with an immediate response. Culturing or re-culturing, empirically changing treatment or referring the patient to a cornea clinic are all appropriate steps, and the only plainly inappropriate step would be to continue watching the eye deteriorate. In addition to failure with original treatment, delay in referral to a cornea clinic is the other significant risk factor leading to keratoplasty with these eyes, so when initial treatment fails, it's imperative to change course.[30] By taking a watchful, conservative approach, using critical thinking to form initial diagnosis and treatment decisions and responding decisively and aggressively to any change for the worse, you are giving your patient the best chance at achieving a positive outcome. RCCL

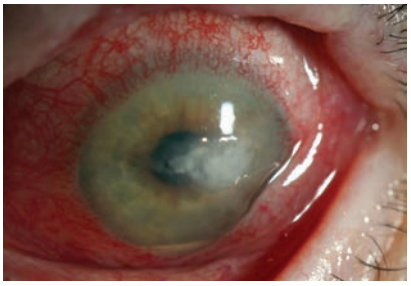

Fig. 3. Note the lack of any defining classic fungal ulceration features In this *Candida* ulcer. There was no history of trauma or injury in this case, suggesting a fungal source. Unfortunately, sometimes which cases should be cultured is only apparent in hindsight.

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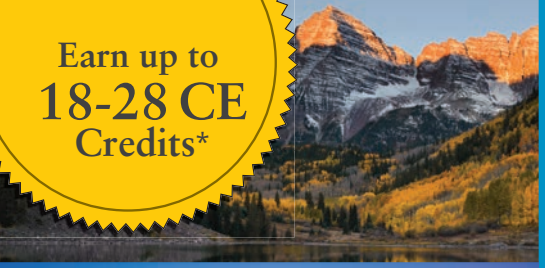

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EXTENDED WEAR: Still an Option?

This modality was all the rage until concerns arose. Is it still worth fitting patients with these contact lenses?

By Evan Kaplan, OD, MS

Extended contact lens wear has long been thought to be the most desirable modality for our patients because it offers great convenience, uninterrupted good vision and suits those with an active lifestyle.[1] Despite these benefits, extended wear is not prescribed with any frequency in the United States—at least not since the late 1980s, when researchers found a much higher incidence of ulcerative keratitis with this modality compared with daily wear.[2]

Currently, only about 5% of new fits in the United States are extended wear contact lens prescriptions.[1] This has not changed over the last 20 years, with the exception of a brief spike in the early 2000s when Ciba Vision released Focus Night and Day and Bausch + Lomb introduced PureVision—the only two contact lenses on the market FDA-approved for up to 30 nights of continuous wear. With these came the term *continuous wear* to distinguish it from *extended wear* (30 days vs. seven days).[1] For the purposes of this article, we will refer to any overnight wear as extended wear.

While a few extended wear contact lens options remain on the market, many uncertainties hinder their use, including concerns that not enough oxygen passes through the lens to support a healthy cornea or that a toxic tear film exists beneath a contact lens that needs to be flushed by removing the lens. In addition, a practitioner's history of contact lens patients with microbial keratitis (MK) or inflammation may lead them to prescribe a more limited wearing time.

This article takes a look at extended wear today, how advances have improved the modality and what questions remain.

LET THE OCULAR SURFACE BREATHE

With contact lens wear, oxygen reaches the cornea both by diffusing through the lens material and mixing with the tears, thereby getting around and beneath the lens. Many studies have looked at the oxygen needs of a healthy cornea, and today's contact lens materials, FDA-approved for extended wear, meet those requirements (Dk/t of 125 x 10-9) according to the widely accepted, relatively straightforward calculation of Dk/t.[3-5] The new high Dk silicone hydrogel lens material used for extended wear contact lenses provides several benefits, such as:

- Reduced incidences of endothelial polymegethism, which occurred as a result of chronic hypoxia with hydrogel lenses.[3]
- Only rare instances of vascular changes (i.e., limbal hyperemia and neovascularization) commonly seen in hydrogel lenses worn on an extended wear basis.[3]
- No more myopic shifts, once observed when changing patients from hydrogel to silicone hydrogel materials—presumed to result from chronic hypoxia.[3]

Although research has yet to identify the actual amount of oxygen diffusing into the cornea underneath a contact lens, investigators suggest it may be an alternate means for evaluating contact lens performance.[3] Further research may help to reveal the rate of oxygen consumption of the cornea, and thus help clinicians to properly evaluate contact lens performance and the impact of extended wear.

Cornea metabolism is also affected by contact lens wear.

ABOUT THE AUTHOR

Dr. Kaplan has been teaching contact lenses to optometry students at SUNY College of Optometry and ophthalmology residents at NY Eye and Ear Infirmary for over 25 years. After graduating from Washington University in St. Louis, he completed his optometry degree from SUNY College of Optometry. Dr. Kaplan continued studying at SUNY to earn a master's degree in vision science and complete his fellowship in cornea and contact lenses. Additionally, he is the co-owner of two group optometry practices in New York City.

Epithelial cells originate at the limbus and travel across the cornea before sloughing off at the ocular surface—a process that might be affected by covering the cornea with a contact lens, possibly inducing hypoxia. When measuring basal epithelial cell proliferation in extended wear contact lens patients, researchers found that oxygen transmissibility of the contact lens material does make a difference with chronic hypoxia, causing a decrease in the metabolic activity of the corneal epithelium and decreased epithelial oxygen consumption. These changes negatively impact normal epithelial growth.[6] Research also shows epithelial thinning and conjunctival hyperemia in extended wear patients, possibly due to alterations in proliferation or exfoliation, which was exaggerated in patients wearing lower Dk materials.[6] Such epithelial thinning due to decreased metabolism might make it prone to infection.[6]

However, Dk/t is only a physical measurement and does not incorporate any physiological component, such as changes due to trapped debris behind a lens worn overnight in a warm environment (e.g., a closed eyelid traps heat from the eye), which is more conducive to bacterial growth.

WARD OFF INFLAMMATION

Researchers know that any contact lens on the eye initiates an inflammatory response, and extended wear options are no different.[7] Even with daily disposable contact lens wearers, clinicians sometimes see evidence of a prior inflammatory event on slit lamp examination, such as subepithelial infiltrate scars, despite the patient denying any clinical event. Studies have yet to uncover whether extended contact lens wear moves the inflammatory response from subclinical to clinical with a statistical difference over daily contact lens wear.

What researchers do know is that corneal erosions—another potential risk for infection—are more likely to occur during extended wear.[8] Overnight, the posterior lens surface adheres to the epithelium; upon awakening and the resumption of blinking, an epithelial disruption is more likely, removing the barrier to bacteria and increasing the risk of an infection.[9]

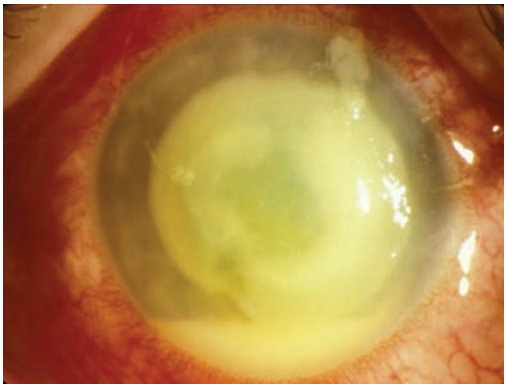

Photo: William D. Townsend, OD

This extended wear patient presented with a large hypopyon ulcer (*Pseudomonas* cultured).

Hypoxia during extended wear also leads to increased binding of bacteria to corneal epithelial cells. Soft contact lens wear significantly increases the amount of binding relative to baseline (no contact lens use), and this binding only increases with extended wear, leading to an increased risk of infection.[10]

MK is the most serious complication of contact lens wear, and while the prevalence is low in the United States, it is still an ocular emergency.[11] Immediate treatment is crucial to minimize the overall size and depth of the ulcer and the inevitable scarring.[11] The greatest risk factor for MK is overnight lens wear—a risk that is similar for both hydrogel and silicone hydrogel lenses.[12]

In the United States, contact lens use and misuse—including overnight wear—accounts for 19% to 42% of culture-proven MK.[13] However, only a small number of those patients were prescribed lenses as extended wear, leading researchers to believe the oxygen permeability of most contact lenses is sufficient to prevent contact lens-related issues.[13,14] Thus, in most cases, complications such as

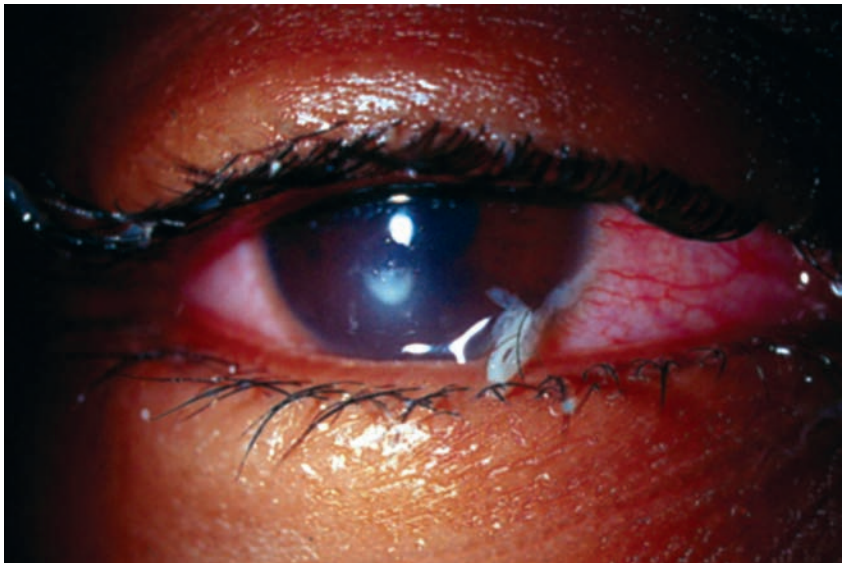

Photo: Joseph Sowka, OD, and Alan G. Kabat, OD

This extended wear patient developed culture-proven *Pseudomonas* keratitis that has mucopurulent discharge.

MK must be caused by some other factor, such as entrapped debris, mechanical irritation or drying of the lens and eye, for example.

Other inflammatory events negatively impact contact lens wear and can be a particular concern for extended wear users:

Contact lens-associated red eye is typically caused by a unilateral acute inflammation as a result of endotoxins—heat-stable toxins associated with the outer membranes of certain gram-negative bacteria—and often occurs in patients suffering from upper respiratory infections.[7]

Contact lens peripheral ulcer is typically due to an inflammation caused by a hypersensitivity reaction to the endotoxins of gram-positive bacteria. These sterile ulcers usually scar and have the potential to negatively affect best-corrected visual acuity even after the acute inflammation is gone.

Despite the widespread thought—by patients and even the government—that contact lenses are a commodity and not a medical device, it is imperative doctors address contact lens wearing schedule, handling and care. A patient's compliance with contact lens wear and care is a crucial factor when prescribing contact lenses. Proper patient education and early detection can prevent most of these events from becoming sight-threatening problems.[15]

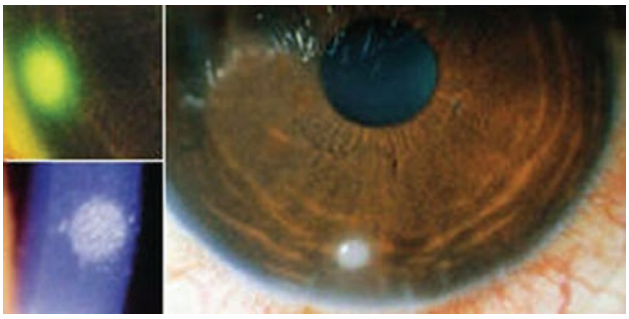

Photo: Clark Chang, OD, MSA, MSc

Management of an acute contact lens peripheral ulcer, as seen here, requires only temporary discontinuation of lens wear until all signs of inflammation are resolved.

THE EYE'S ENVIRONMENT

The contents found naturally in tears, in addition to what the eye has been exposed to during the day—which can dissolve or became entrapped in the tear film under a contact lens—are huge factors to consider with contact lens wear.

The tear film is full of potential inflammatory stimulants, including lipids, proteins and mucin, exfoliated cornea epithelial cells, byproducts of corneal metabolism and chronic blepharitis, to name a few. In addition, exposure to a smoky or toxic environment can initiate any number of ocular side effects that can complicate contact lens wear. The eye's ability to respond to any of these factors may be affected by the individual's genetics, the wearing schedule and contact lens material.[7]

Contact lens wear changes the natural flow of tears across the corneal epithelium. It also hinders the mechanical effect of blinking to clear toxins.[8] Tears are even more stagnant with extended lens wear, further increasing the exposure of these potential toxins to the cornea. Research suggests mucin balls, which are typically seen under extended wear lenses, might act as protective agents. They were once thought to be innocuous, but some research now suggests they are protective against inflammatory events, as they appear to form due to corneal surface irregularity and might help to smooth out the cornea and activate keratocytes.[16,17]

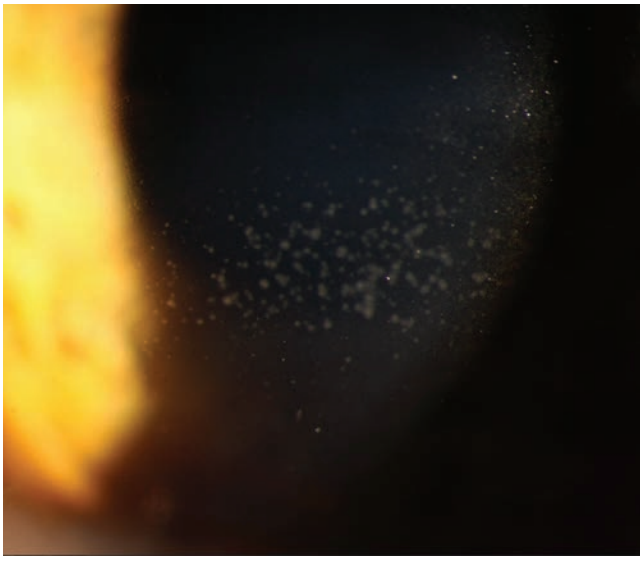

Photo: Christine W. Sindt, OD

Mucin balls, as seen here, are a typical finding under extended wear contact lenses.

INDICATIONS FOR EXTENDED WEAR

Other factors to consider when choosing the right contact lens modality are the patient's ocular and systemic medical conditions that might impact their contact lens use. While extended wear is necessary for bandage lenses in patients with corneal abrasions, bullous keratopathy and keratoprostheses, these patients are often prescribed antibiotics and anti-inflammatory drops while wearing the lenses. Contraindications to extended contact lens wear include patients with collagen vascular diseases and diabetes, for example.

Over the past decade, overnight orthokeratology has been renewing the discussion on the safety of extended wear, particularly with

pediatric patients. Even though extended wear is generally thought to be a safe modality, MK continues to be a concern in this population.[18] Poor lens hygiene and noncompliance with cleaning regimens are risk factors for *Acanthamoeba* keratitis, for example, especially in pediatric patients caring for their own lenses.[18] To keep this a safe modality, clinicians must overemphasize the wearing and cleaning regimens with patients and their parents.

UNANSWERED QUESTIONS

Research has yet to reveal whether a difference in inflammation or infection rates exists for patients who are prescribed extended wear lenses vs. those who choose this modality without a prescription. A better understanding of the impact of patient education on extended contact lens wear will help clinicians to avoid negative outcomes. Inflammatory events are common in contact lens wearers—and research shows these events are more likely in extended wear patients—but it is unclear whether an increase in inflammatory mediators in the tear film while sleeping is the only cause. Future studies are needed to explore patients' possible genetic predisposition to inflammation, as well as affordable in-office tests for genetics, goblet cell density and antigen-presenting cells. A better understanding of specific environmental factors that make extended wear an impossibility for at-risk patients would also help clinicians with patient selection. Some contact lens wearers are perpetually noncompliant and wear their lenses continuously without any negative events—these patients may be the key to understanding who can and cannot tolerate extended wear due to their genetic makeup.

Most clinicians know their patients would prefer extended wear contact lenses. To help make that desire a reality while maintaining safe contact lens wear, we must further the research. Worldwide, contact lenses are a multi-billion dollar industry—we should harness some of that money to continue to research ways of determining what makes some patients excellent contact lens wearers with no or minimal complications. RCCL

Extended Wear of the Future

Our society is obsessed with wearable technology. In the coming years, we might see more extended wear use with the advancement of therapeutic contact lens wear. Lenses may soon monitor glucose levels in tears and send the data directly to the patient, their provider or both.[1,2] They may be able to record diurnal curve measurements for our glaucoma patients and provide sustained release of medications, just to name a few innovations in the pipeline.[3]

Many companies are even looking at contact lenses to project a televised image directly to your retina for a larger-than-life virtual reality experience or to aid as a reflecting telescopic for our low vision patients.[4]

All of these advances, particularly those used for health monitoring, might lead to more extended wear of contact lenses and will demand rigorous patient education to ensure they stay safe in contact lens wear.

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Contact Lens Care: Advice from an Expert

As we look to the future of contact lens safety, it's important to remember our past and how it shaped our present.

By Christine W. Sindt, OD

Despite today's improved contact lens care technology fueled by decades of research and the myriad of options currently available, there is still work to be done. Contact lens dropout rates due to discomfort are still an issue, and as a result, finding the right approach to best suit our patients can be difficult.[1]

Ralph Stone, PhD, had a long career leading teams in the development of multipurpose solutions, as well as creating an FDA classification system of lens materials for testing lens-solution interactions. Dr. Stone is currently president of RP Stone Consulting and continues as a member of the American National Standards Institute.

As contact lens safety continues to be an important consideration in today's optometric world, and we discussed with Dr. Stone how the concerns of the past have evolved into the challenges of the future:

CS: When did you start in the contact lens industry, and what were the lens cleaning solutions of the time?

RS: I started in the contact lens and lens care industry in 1981, 10 years after the initial introduction of soft contact lenses into the United States in 1971. By that time, the primary mode of lens care was the use of heat units designed to disinfect lenses at above 80°C for at least 10 minutes. This was based on a number of experiments with a range of microorganisms. Cold disinfection solutions were introduced based on chlorhexidine and thimerosal. Both of these systems were only for disinfection, and separate enzymatic cleaners were used in conjunction with disinfection. Separate cleaners were necessary, since the life of a lens was expected to be a year, primarily because of the cost of the lens. Shortly after I began my career, hydrogen peroxide was introduced. This used the ever-present platinum disk, but also a faster, two-step system: first, soaking 3% hydrogen peroxide for at least 20 minutes, followed by a chemical neutralizer such as thiosulfate or catalase.

As time progressed, the introduction of novel cold disinfection and multipurpose solutions evolved, initially with single biocides and later with multiple active ingredients.

During this evolution, the standards for efficacy evolved as we began to understand the risks associated with contact lens wear. This did not seem to affect the rates of microbial keratitis (MK), however.

CS: Good points. Thimerosol, a mercury and thiosalicylate-based compound, causes a delayed hypersensitivity reaction in approximately 10% of people.[2,3] According to the North American Contact Dermatitis Group, it is the 5th most common allergen.[2,3] Also, nearly one million clinic visits are made yearly for MK, with contact lens wear being the single biggest risk factor.[4] Specific issues include overnight lens wear and inadequate replacement, cleaning and disinfection of contact lenses and lens cases.[4]

What is the history of the contact lens classification system?

RS: The classification system (*Tables 1 and 2*) came as a result of the changing materials used in the contact lenses. For most of the first 10 years, all the materials had water contents less than 50%, primarily based on the use of HEMA. As companies looked to expand the offerings and

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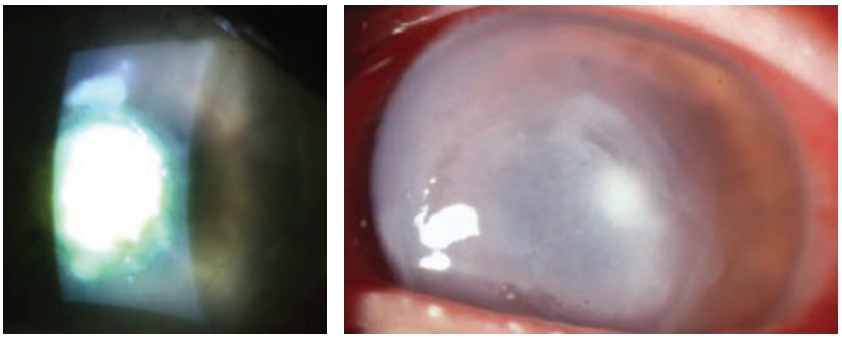
recognized that we needed to get more oxygen through the lens, the water content was increased by using acids such as methacrylic acid or other polymers with better water content based on polyvinyl pyrrolidone. Those of us working on keeping lenses clean found the association of proteins with lenses was remarkably different with these newer, higher-water, acid-containing materials.

At about the same time, we started seeing that the disinfecting compounds in use were being absorbed into the lenses, creating clinical issues. In investigating these properties, we found that some clear divisions existed in the lens properties based on water content and concentration of ionic compounds in the lens materials. This was based on descriptions of how water incorporated into the polymers. The classification system was proposed and vetted throughout the industry for the testing of care systems before acceptance first in the United States and then Europe as the early descriptors of low-, medium- and high-water materials did not provide adequate ability to communicate the properties of the lenses.

With the advent of silicone hydrogel around the year 2000, we combined the properties of the rigid gas permeable materials and hydrogel materials. While that solved the issues of providing enhanced oxygen to the cornea, these lenses had all the issues related to the chemistry of both types of materials. From early on, we recognized this was a new entity, and as early as 2002 many of us were commenting on these differences in lectures and publications. The American National Standards Institute (ANSI) and the International Organization for Standardization (ISO) then



Photos: Scott G. Hauswirth, OD, and Richard Mangan, OD

***Staphylococcus aureus*, left, and *Pseudomonas aeruginosa*, right, were among the five organisms tested by the ISO to develop its recent contact lens solution disinfection testing guidelines.**

put this information into systems, leading initially to the recognition of a new group: Class V materials (silicone hydrogels). As we have had increased numbers of developments using these materials, they have been divided into three subclasses, VA, VB and VC, based on the use of ionic ingredients and water content. Further subgrouping to account to the chemistries used to provide for improved surface wettability has also been added to the grouping process for class V materials. These have recently been recognized as a part of the ANSI and ISO standards for contact lenses.

CS: Thanks for mapping that out for us. To expand a little on the ISO subdivision, its recent version of the contact lens solution disinfection testing guidelines includes recommendations for lens soaking and storage periods when contaminating microorganisms are introduced through patient handling.[5] To develop these recommendations, the antimicrobial efficacy of the test solution was evaluated at specific time intervals of 24 hours, seven days and the maximum labeled storage as stated by the lens case manufacturer.[5] Five challenge organisms were tested: *Staphylococcus aureus, Pseudomonas aeruginosa, Serratia marcesens, Candida albicans* and *Fusarium solani*.[5] This standard is reviewed every five years.[5]

Moving on, what are the biggest misconceptions about contact lens care solutions? What do practitioners need to know to keep patients safe?

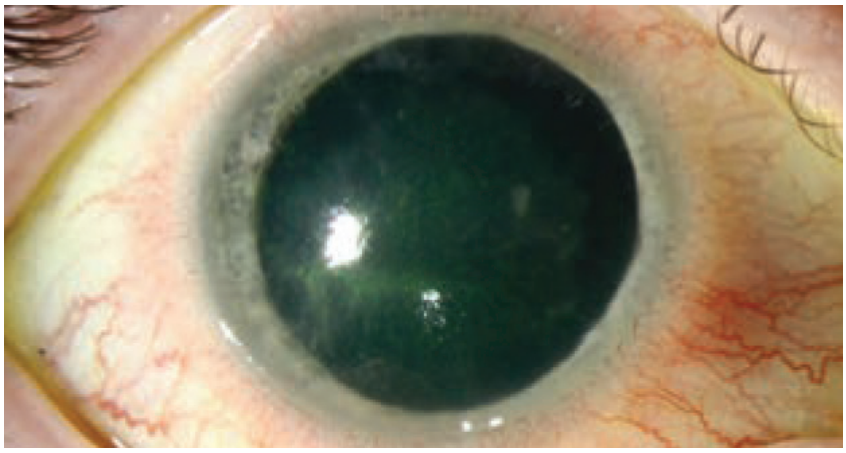
RS: The first misconception is that they produce sterile lenses with no bacteria or fungi associated with the lens or the case. The disinfecting properties are designed to reduce the level of organisms to "safe" levels. In testing, this means that when used for disinfection or in a regimen, the solution by itself reduces the presence of organisms from a million to less than 10. While this requires activity far higher than the levels observed on lenses in normal usage by a factor of a 100 to 1,000, it is not zero.

The second misconception is that lenses going back into the eye are as good as in the case at the end of the care cycle. The biggest source of contamination, other than dropping lenses, is the patient's hands during lens handling and insertion. Studies looking at lenses taken from the eye using sterile techniques show very low levels of contamination, in the order of 100 to 200. After handling, however, lenses going

into the disinfection process have levels reaching into the thousands, which is the level of disinfection the systems have to deal with. When we put a lens back on the eye, the organisms from the finger are introduced onto the lens. This is probably why we do not see a big difference in the rate of infections between daily disposable lenses and frequent replacement lenses, although severity appears to be less.

The third misconception is that the care systems are equally effective against all organisms, even the different species and strains. Although the biocides used for lens care are designed to work through mechanisms that minimize organism survival, the biocides are not equally effective in all instances, and sometimes survivors remain. This is often seen as contamination in the cases of various organisms. The simplest example, although not the only one, is hydrogen peroxide. Some organisms produce catalase, an enzyme that may inactivate peroxide, reducing its efficacy. This points to our need for regular case cleaning and replacement by patients. How many cases have we seen that are visibly dirty?

The fourth misconception is in our teaching of compliance. Overall, we don't do a good job of this. Every major recall related to infections has been linked to "topping off." Multipurpose solutions are designed for use with a single bottle meant to last about one month, but usage rates are only about 3.6 to 3.8 bottles a year. While we recognize the need for emptying, cleaning and refilling cases with fresh solution, patients do not do it. These solutions are designed for use for a single cycle, whether it is hydrogen peroxide, which is neutralized, or a multipurpose solution. We are not emphasizing this enough in our practices. Contact lenses are recognized as a medical device used in the eye, and patients need to understand the importance of good hygiene.



While *Acanthamoeba* infections, shown here, aren't the most frequently encountered of entities seen in clinical practice, they are serious events that require swift treatment.

CS: Very true. A couple side notes: Overnight wear, increased exposure in daily wear, smoking and poor hand hygiene are significant risk factors for MK.[6] However, the organisms isolated on the lenses are consistent with less severe disease.[6] Also, the most efficacious way to clean a contact lens case is to rinse the case with multipurpose solution, followed by wiping out the case with a clean tissue and air-drying.[7]

Next question: What has been the biggest advance in solution knowledge during your career?

RS: I think of two areas that have enhanced the proliferation of safe contact lens wear. The first is the development of better, safer and less toxic biocides. During my career we have introduced the current array of novel biocides, starting with relying on heat and chlorhexidine/thimerosal disinfectants and advancing to hydrogen peroxide, polyhexamethylene biguanide, polyquaternium-1 and, more recently, Aldox and alexidine. We have learned that we can be even more effective by using combinations to provide a broader spectrum of activity.

The second and more difficult area has been keeping lenses wet and lubricated. We have known that soft contact lenses tend to get dry areas over time, causing the polymer to accommodate by migrating the like hydrophobic portions of the polymer chains to the surface. This dryness may be at least partially responsible for the major issue facing the field: contact lens dropouts.

We recognized early on that the eye does try to accommodate and make lenses wet. Studies focused on spoilation of extended wear lenses showed that comfort was not best on day one, but rather on days two, three and sometimes four, with a decrease going forward as the fouling became more of an issue.

Since the lid may be the primary driver in all this, practitioners have been looking for ways to keep lenses wet and lubricated. New wetting agents have helped

solutions maintain their action over the course of the day. This has improved the maintenance of the lenses during care, and the introduction of silicone hydrogels has made this even more important. Lenses are often modified at the surface with bonded chemistry that minimizes the appearance of non-wetting areas.

CS: What changes still need to be made?

RS: The first and most prominent need in this industry is to make compliance a number-one priority. I recently read an article indicating the importance of reducing chair time. However, practitioners should not skip patient education just for the sake of reducing chair time. It is imperative that both doctor and staff put emphasis on teaching patients the importance of using a medical device.

The second need is development of better case technology. We still are using the same technologies developed in the 1970s and '80s. Significant levels of case contamination continue to be reported, even in controlled clinical trials. Development of cases that engender compliance and have finite lifetimes is critical.

While several of these concepts are on the drawing boards, few have seen the marketplace. This is needed to minimize the inoculation of organisms into the eye.

The third is to understand the occurrence of *Acanthamoeba* infections. These continue to occur at a low level, but are still serious events. There appears to be an increase from levels seen around 10 years earlier. While many of our current products have some activity, we are still trying to find a reproducible measurement system to understand the effectiveness.

Testing methods are currently undergoing multi-laboratory evaluations to validate these options. Researchers speculate that the increase noted in 2007 was first related to a single product and, overall, probably related to changes in our drinking water requirements.

The final need is continued exploration of biocide technologies, as clinical isolates will continue to develop that are outside the range of current efficacy standards or products. No single option or combination can be totally effective against all organisms and the multitude of strains present as they continue to evolve. It is imperative to note that testing of clinical strains outside of standard culture collections is not necessarily reproducible between laboratories, as the organisms continue to mutate. As such, we must pursue continued monitoring of the organisms causing infections.

While the industry has made great strides in contact lens care such as improved biocides and lens wetting systems, there is still much that can be done to create a better experience for our contact lens patients. In order to continue our growth, we only need to look to our past and how it has shaped our present. Dr. Stone has seen this evolution firsthand, and looking to experience like his for guidance will be helpful as we move forward. RCCL

Table 1. Conventional Hydrophilic Material Groups

Group	Description
I	Low Water Content (<50%), Nonionic*
II	High Water Content (>50%), Nonionic*
III	Low Water Content (<50%), Ionic*
III	High Water Content (>50%), Ionic*
*Being ionic in pH = 6.0 - 8.0	

Table 2. Silicone Hydrophilic Material Groups

Group	Description
V-A	No Water Specification Ionic*
V-B	High Water Content (>50%), Nonionic*
V-C	Low Water Content (<50%), Nonionic*, Hydrophilic-monomer Only
V-Cm	Low Water Content (<50%), Nonionic*, Surface Tested (ST)
V-Cr	Low Water Content (<50%), Nonionic(, Non-ST, Semi-interpenetrating Network
*Being ionic in pH = 6.0 - 8.0	

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KERATOPROSTHESIS: When Standard Transplants Fail

Here's what you need to know about this specialized procedure for treating blinding corneal disease when traditional therapies don't work.

By James Esposito, OD

While traditional penetrating keratoplasty is successful in treating many corneal diseases to prevent blindness, sometimes standard corneal transplants fail. When this happens, a keratoprosthesis is an important alternative treatment modality. Furthermore, for the pediatric population and other high-risk conditions such as chemical burns, ocular trauma, herpetic keratitis, Stevens-Johnson syndrome and mucous membrane pemphigoid, researchers have increasingly advocated it as a primary penetrating corneal procedure over standard penetrating keratoplasty.[1-3]

The basic principle of keratoprostheses is the insertion of a clear "window" into the ocular surface to maintain a clear visual axis, independent of the state of the surrounding cornea. Numerous materials with favorable optical properties are used for this purpose, as are diverse strategies for biointegration into the surrounding ocular surface tissues. Thus, patients can accomplish a prompt recovery of best achievable visual acuity and good stability of the implant.

Most advances in the field of keratoprosthesis research have occurred during the second half of the twentieth century, as recognition of the limitations of penetrating keratoplasty, an inadequate supply of donor tissue and the development of new synthetic polymers sparked increased interest in keratoprostheses.[2,4-7] This article focuses on the most widely used keratoprosthesis in the United States, the Boston keratoprosthesis type 1 (KPro).

BOSTON KPRO BASICS

This keratoprosthesis was developed by Claes Dohlman, MD, PhD, at the Massachusetts Eye and Ear Infirmary in 1968.[4,8,9] Dr. Dohlman and colleagues have introduced many improvements to the original collar button design over the past five decades.[9,10] The device obtained FDA clearance in 1992, and more than 12,000 of these devices have been implanted worldwide.[5,8]

Two types of Boston keratoprostheses exist, both consisting of two plates joined by a polymethyl methacrylate (PMMA) stem, which is the optical portion. They are designed to be incorporated into a ring-shaped cadaver corneal graft, which can be donor corneal tissue not suitable for endothelial replacement, since healthy endothelium is not required.

DESIGN

The Boston KPro type 1 has a PMMA front plate that is 5.5mm in diameter, into which the appropriate dioptric power has been ground.[4] The front plate is fused to a 3.35mm cylindrical optical stem, onto which the surgeon slides the

One or Two?

The KPro type 1 is used for patients with adequate tear function, while type 2 is reserved for rare cases of abnormal lid function and tear secretion and ocular surface keratinization.[1] Even in conditions at extremely high risk for transplant and repeat graft failure, surgeons will still try a type 1 device before performing a type 2 prosthesis. Type 2 involves a complete tarsorrhaphy and has an additional anterior extension for implantation through surgically closed eyelids.[1]

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ABOUT THE AUTHOR

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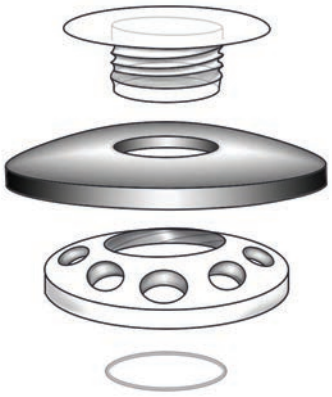


This schematic drawing illustrates the anterior to posterior assembly of a Boston KPro type 1.

different assembly components. The PMMA back plate is 8.5mm in diameter and has 16 fenestrations, each 1.3mm in diameter. The fenestrated back plate is important for nutrient exchange with graft keratocytes from the aqueous and to keep the corneal graft hydrated. Finally, a locking ring, either titanium or PMMA, is placed behind the back plate to secure the assembly.

New research suggests titanium may be superior to PMMA for the back plate.[6,11] The Boston KPro with a titanium back plate now has FDA approval and is largely replacing the older PMMA version, but both material options remain available and marketed under the same name, the Boston KPro type 1. Not only is titanium superior from a manufacturing and resilience standpoint, but as an inert material it also appears to cause less postoperative inflammation and retroprosthesis membrane formation.[6]

PATIENT SELECTION

The Boston KPro type 1 is best suited in cases of refractory corneal blindness with repeated graft failure and pediatric congenital corneal opacities, such as Peter's anomaly.[1-3,12]

Blink rate and tear production are important patient selection factors because this device often fails in the setting of a dry ocular surface.[5,6,13] Therefore, clinicians should perform a Schirmer's test, note lagophthalmos or other lid pathology and estimate the rate and completeness of blinking preoperatively (when the patient is not aware they are being observed) before referring a patient for a keratoprosthesis. Practitioners should also inspect the conjunctiva for any evidence of inflammation, surface keratinization and symblephara, which may need to be addressed concurrently.

The Boston KPro type 1 can be successful in patients with cicatrizing diseases (e.g., Stevens-Johnson syndrome, mucous membrane pemphigoid, chemical burns) when there is a good blink and tear film.[5,6,13] However, these groups of patients remain a high-risk category and a type 2 Boston KPro is often considered in these conditions.

PROCEDURE

The surgery for a Boston KPro type 1 implantation is analogous to regular penetrating keratoplasty. Either general or retrobulbar anesthesia can be used. Surgical steps include:

1. The surgeon slides an 8.5mm corneal donor button—in which a central 3mm hole has been punched—onto the stem of the front plate.

2. The titanium or PMMA back plate is then advanced onto the cylindrical stem to sandwich the donor graft between the two plates.

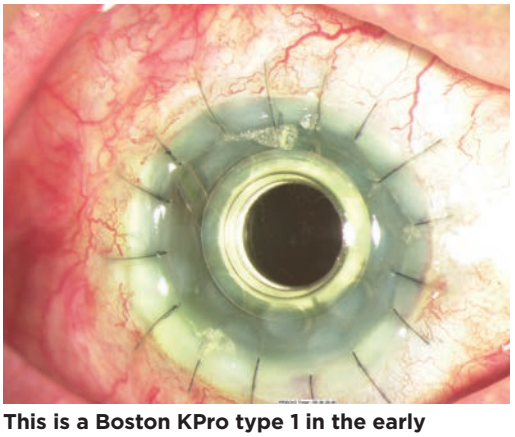
3. The surgeon slides the titanium ring onto the stem and snaps it into place, securing the entire assembly. The patient's cornea is then trephined in the standard manner.

4. The donor button/keratoprosthesis complex is sutured into the patient's cornea, as in a standard penetrating keratoplasty, using nylon interrupted sutures.

5. At the end of the procedure, the surgeon places a 16.0mm diameter, 9.8mm base curve, plano soft Kontur contact lens onto the eye as a bandage lens.

POST-OP CARE

Postoperatively, patients are started on prednisolone acetate 1% QID for one week and tapered over a period of two to three months. A slower taper is advised if one notes a fast rate of a retroprosthetic membrane formation. However, a prolonged taper may increase the risk of a fungal infection, especially in endemic areas. Patients are also started on topical polymyxin B/trimethoprim QID for one week, then maintained on a once daily prophylactic dose. Vancomycin (14 mg/mL) at the same frequency is additionally recommended in patients with chemical burns or autoimmune conditions. Alternatively, a combination of a fourth-generation fluoroquinolone and vancomycin can be used.



This is a Boston KPro type 1 in the early postoperative period.

OUTCOMES

When implanted in low-risk eyes, the Boston KPro type 1 has a high retention rate—greater than 80% beyond 40 months, according to two recent studies.[14,15] In a prospective cohort study, 300 patients demonstrated a retention rate of 93% after an average of 17 months.[16] The preoperative mean acuity was 20/1205, and final mean acuity was 20/150.[16]

In another prospective multicentered study, 60% of 133 patients achieved a best-corrected visual acuity of 20/200 or better, which was retained in 90% of eyes during the reported follow-up of 8.5 months.[17] Multiple small studies support these numbers, and many show improved outcomes with the newer device designs.[14,15]

The prognosis for high-risk patients, such as those with severe autoimmune disease or chemical burns, is more guarded with variable results. Retention rates generally are lower, ranging from 59% to 89%, with a visual acuity of 20/200 in approximately two-thirds of cases.[6,14-18]

COMPLICATIONS

Several possible concerns exist for this procedure, including:

Retroprosthetic membranes. These are commonly reported from 13% to 60%, and likely result from intraocular inflammation.[17,19,20] Histopathologic studies with the Boston KPro show that the membranes originate from the host's corneal stroma.[5,6,11] Luckily, the development of the titanium back plate has reduced their incidence.[6,11] Peribulbar steroid injections may be useful in the early stages of membrane formation to slow progression.

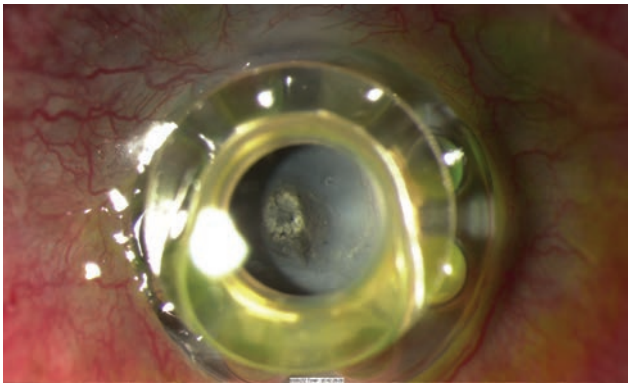
Once the membrane has formed, an Nd:YAG laser, followed by topical steroids, can create an opening in the membrane and restore vision. If the membrane becomes too thick and vascularized, surgical management becomes necessary.

Melts and extrusion. Corneal melts tend to occur at the edge of the front plate. Biomicroscopy and anterior segment optical coherence tomography are helpful in detecting and monitoring corneal thinning around the keratoprosthesis. A leak on Seidel testing should prompt an ultrasound to check for choroidal effusions. Kissing choroidal detachments frequently lead to retinal detachment and have a very poor prognosis for visual recovery.

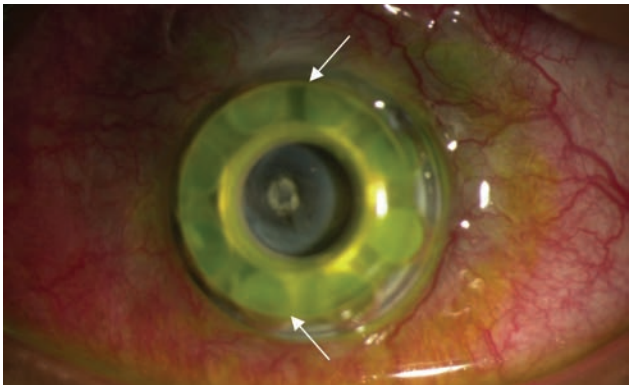
Research shows a good bandage soft contact lens fit and retention reduces the risk for corneal melts and extrusion.[21] Also, a shortened use of topical corticosteroids and avoiding toxic drops, such as fortified vancomycin, are helpful in reducing the risk of corneal melts.

Infectious endophthalmitis. This remains the most common posterior segment complication following keratoprosthesis surgery.[5,14-16,22] It usually presents following microbial keratitis with decreased vision in an injected and painful eye, and often shows a leak on Seidel testing. Prophylactic topical antibiotics have greatly reduced bacterial endophthalmitis rates, with recent studies reporting 0% to 14.7%.[5,14-16,22-25] Treatment generally consists of leak repair, intravitreal tap and antibiotic injection, and use of topical antibiotics.

While wearing an extended wear contact lens, fungal infections can also arise with chronic treatment involving corticosteroids and antibiotics. In one series, four out of 202 eyes with a keratoprosthesis developed a confirmed fungal keratitis or endophthalmitis over a cumulative 6,893 patient-months of follow-up.[26] If clinicians see fungal colonization of the contact lens, they should change the contact lens



A dense retroprosthetic membrane in a Boston KPro type 1, as seen here, required surgical removal, but Nd:YAG laser was attempted in the early stage of membrane formation. This patient also has extrusion with the front plate vaulting from the ocular surface.



The white arrows in this lower magnification view of the same patient delineate the edge of the front plate. This patient has extrusion with the front plate vaulting from the ocular surface. The donor corneal graft is no longer visible after corneal melt.

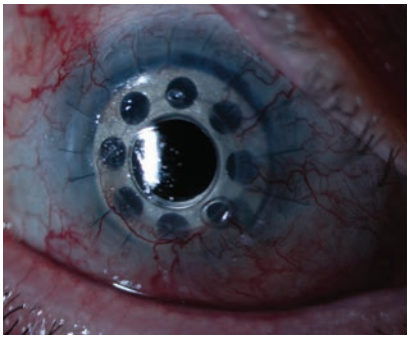


Photo: Clark Chang, OD, MSA, MSc

This KPro patient was fit with a custom bandage lens because the standard KPro bandage lens would not stay on eye.

and administer a course of topical amphotericin B 0.15%. The prognosis in fungal infections is usually good if identified early.

Another manifestation of ocular inflammation is a sterile uveitis and vitritis. This condition masquerades as a bacterial endophthalmitis with a reduction in vision; however, no pain, tenderness or redness is typically observed. Treatment with topical and peribulbar steroids typically results in restoration of baseline vision.

Glaucoma. This is currently the single most serious complication following Boston KPro surgery. Although many patients have glaucoma preoperatively, it frequently worsens in some and develops in others following keratoprosthesis surgery.[5,14-16,22-25] Research suggests chronic low-grade inflammation, progressive angle closure and anterior displacement of the iris are all factors in glaucoma progression.

Intraocular pressure (IOP) is the most important risk factor for developing glaucoma, yet it cannot be reliably measured in Boston KPro patients, even though clinicians should estimate IOP with finger palpation at every visit. The lack of objectivity of such a measurement has prompted the development of alternative methods such as scleral tonometry, which has been tried with variable success.[27,28]

Patients with glaucoma or suspicion should be managed in conjunction with a specialist. Often, surgeons consider implanting a glaucoma drainage device concomitantly during surgery.

Contact lens-related complications. Soft contact lenses have become the standard of care to protect against evaporative damage to the corneal surface around the Boston KPro. They can be worn for many months and even years without replacement. Extended contact lens wear is actually recommended, and the lens helps to protect a fragile ocular surface from mechanical stress and desiccation, reducing the risk of stromal thinning and corneal melt.[21] Although uncommon, extreme lens deposits can reduce visual acuity. This seems to be more related to a low blink rate and incomplete blinking, rather than low Schirmer values. Refitting into a hybrid or large diameter rigid gas permeable lens can be beneficial for reducing lens deposits.

The Boston keratoprosthesis type 1 is now considered a viable alternative to standard corneal transplantation for specific corneal conditions. Careful patient selection is paramount, since these patients will require lifelong monitoring, often from multiple subspecialists. Notwithstanding, there is no question that the Boston KPro has helped restore vision in many blinded by severe corneal disease. RCCL

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By Elyse L. Chaglasian, OD, and Tammy Than, MS, OD

Juggling Dry Eye and Glaucoma

Overlap increases as our patients age, so we need to be aware of how treatment of one disease affects the other.

Recently in our dry eye clinic, I (Dr. Chaglasian) saw an 83-year-old female with significant subjective and objective signs and symptoms of ocular surface disease (OSD), including redness, burning, itching, watering, foreign body sensation, corneal and conjunctival staining, increased tear osmolarity and decreased tear production.

THE COMPLICATION

This is not an unusual presentation in our clinic, but she was also being treated for moderate primary open-angle glaucoma. She reported side effects, such as irritation and redness, to hypotensive drops including prostaglandins, carbonic anhydrase inhibitors and alpha agonists.

At the time, she was using Restasis (cyclosporine, Allergan), preservative-free artificial tears and nighttime gel. She had tried bandage contact lenses with minimal success. She had also undergone selective laser trabeculoplasty to reduce some of her topical medication usage and was only using Simbrinza (brinzolamide/brimonidine, Alcon) at the time of initial presentation. She admitted that she sometimes purposely skipped taking her glaucoma medication because of the irritation.

This serves as a reminder that as our population ages, there will be greater overlap between the dry eye and glaucoma populations we see in our practices.[1] We need to be cognizant to not exacerbate one condition while treating the other. Although multidose, preserved, topical eyedrops are the mainstay of glaucoma management, other available options need to be considered, including "softer" preservatives and preservative-free preparations.

While there is no disputing the benefits of reducing microbial contamination with preservatives, significant negative side effects to the ocular surface exist and cannot be ignored in vulnerable eyes, such as destabilization of the pre-corneal tear film, increase in evaporation of the lipid layer, corneal neurotoxicity, inflammation and reduction in aqueous tear production.

THE CULPRIT

Often, the problem is benzalkonium chloride, a cationic surfactant, which can damage the corneal and conjunctival tissues, even in small concentrations.[2-4] Other preservatives, such as Purite and Sofzia, offer antimicrobial properties while minimizing (though not eliminating) ocular surface compromise. Purite, found in Alphagan P (brimonidine, Allergan), is a stabilized oxychloro complex that destabilizes into non-toxic components upon instillation. Sofzia, found in Travatan Z (travoprost, Alcon) contains borate, zinc and sorbitol. Upon instillation, it is inactivated via tear enzymes.[5]

In patients with concurrent OSD and glaucoma, preservative-free medications have been effective in treating intraocular pressure (IOP) while also improving patient compliance and symptoms and reducing inflammation, osmolarity and clinical signs.[6,7]

THE DOWNSIDES

Without the antimicrobial benefit afforded by preservatives, these medications need to be packaged in individual, single-use containers. These can be difficult for patients to open. Another downside is cost, as these medications tend to be more expensive than multiuse bottles. A bottle of latanoprost can cost as little as $12, while a 30-vial package of preservative-free Zioptan (tafluprost, Akorn) is $195 on GoodRx.

Getting back to our patient: After consultation with a colleague who was managing the patient's glaucoma, we switched her to Zioptan. At her last dry eye follow-up one year later, she reported full compliance with its use and improved comfort with stable IOP. Her osmolarity and clinical signs had also improved.

Glaucoma and OSD are chronic diseases that need long-term therapies. Individualized treatment plans to maximize outcomes are essential, and alternative preservative or preservative-free solutions need to be in the forefront of our minds. RCCL

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One Size Does Not Fit All

Custom-designed contact lenses can be a big help for your outlier patients. Here's one case that displays the benefits.

The contact lens manufacturing process has become more sophisticated and efficient since soft contact lenses were first introduced in the 1970s. Optometrists are now able to provide high quality lenses at a reasonable price to the general population.

To make the manufacturing of prefabricated lenses more cost efficient, companies design contact lenses to fit patients who fall in the center of the bell curve for shape and size. To create this bell curve, manufacturers evaluate the size and curvature of many different eyes and pick the base curve and diameter that work for as many of them as possible.

While this strategy is good for the majority of patients with average eyes, it's not ideal for patients who are outliers. Luckily, practitioners can significantly improve fit and vision for these patients by turning to lens customization. This patient, for example, failed in traditional soft contact lenses, but found success with a little extra care.

CASE

A healthy 13-year-old female was referred by her ophthalmologist for a contact lens evaluation. Her motivation for wearing contact lenses was to get rid of her glasses before starting a new school in the fall.

Her ocular history was significant for nystagmus and mild amblyopia. Her medical history was positive for allergic rhinitis, for which she took Zyrtec (Johnson & Johnson) as needed.

Most importantly, she had a history of contact lens intolerance, and multiple doctors in the past two years had yet to find a successful contact lens fit. Every lens she tried would pop out. Her previous doctor went through multiple trials before settling on silicone hydrogel monthly disposable lenses, which she still couldn't tolerate for long. She felt the lenses made her eyes dry.

The patient's presenting visual acuity was 20/25 OD, 20/30- OS. Her presenting spectacle prescription was -1.50+0.75x089 OD and -2.25 sph OS. Manifest refraction was -1.75+0.50x085 20/25+ OD and -3.75+0.50x080 20/25 OS. Simulated keratometry per topography measured 41.01/42.21@011 OD and 41.15/41.98@171 OS (*Figure 1*). Horizontal visible iris diameter (HVID) was noted as 12.9mm OD and 13.3mm OS.

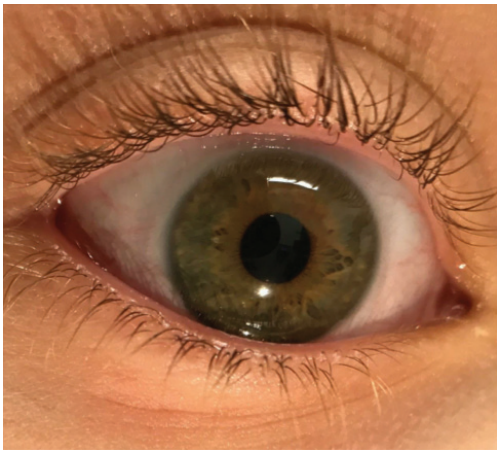

Fig. 2. A 14.5mm diameter Biofinity toric lens, barely covering the corneal surface.

A slit lamp exam revealed clear lashes and cornea OU and 1+ papillae in the palpebral conjunctiva OU. The anterior chamber was deep and quiet OU. The iris and lens were normal OU. Intraocular pressure was 14mm Hg OD and 13mm Hg OS by Goldmann applanation. An undilated view of the posterior segment was within normal limits.

CONTACT LENS FITTING

The average HVID in the general population is 11.8mm.[1] Due to the large diameter of her HVID, we tried Biofinity toric (Coopervision) lenses instead of spherical lenses. The new lens parameters were 8.7 BC/-1.00-0.75x175/14.5 diameter OD and 8.7BC/-2.50-0.75x170/14.5 diameter OS. The lens diameter barely covered her cornea, and the lens edge sat below

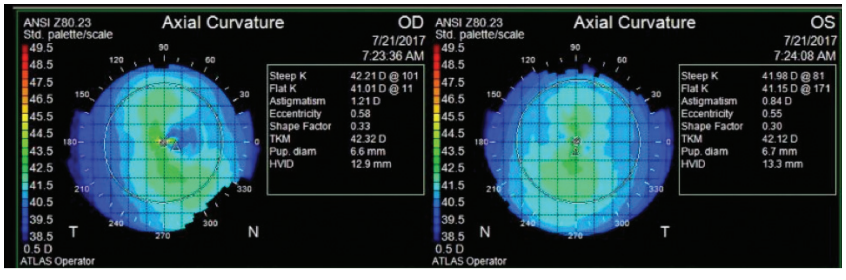

Fig. 1. Corneal topography depicting large horizontal visible iris diameter OU.

Image: SpecialEyes

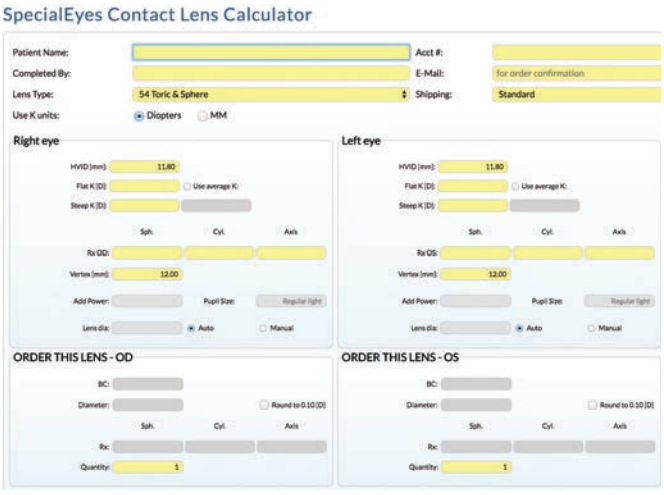

Fig. 3. A custom toric lens was made for the patient using the SpecialEyes Contact Lens Calculator.

the lid margin (*Figure 2*). A hard blink would eject the lens out of her eye. There was low movement of the lens, which appeared to be too steep on the flat cornea. We realized her large, flat corneas fell outside of the "one-size-fits-most" bell curve used by prefabricated contact lenses. From this evaluation, it was apparent she needed a custom lens.

We designed a lens empirically using the arc length soft lens calculator on the SpecialEyes website (*Figure 3*). Taking into account the HVID, base curve and prescription, we ordered a toric SpecialEyes lens of the following parameters: 8.6BC/-1.25-0.50x175/15.7 diameter OD and 8.5BC/-3.25-0.50x170 /15.9 diameter OS.

She also had ocular allergies, for which we prescribed Pataday (olopatadine hydrochloride) QD OU.

DISPENSING

The patient presented for her lens dispensing two weeks later with no new complaints. With her new soft lenses in place, her visual acuity was 20/20 OD and 20/25 OS. The lens tucked nicely underneath the upper lid and was well-centered with adequate movement (*Figure 4*). We instructed the patient to use hydrogen peroxide cleaning solution for nightly cleaning to address her allergies and sent her home.

Two weeks later, the patient returned with no complaints. She was thrilled with her vision and comfort and had no issues with lens ejection.

SIZE MATTERS

Soft contact lenses are traditionally fit without taking corneal diameter into account. In most cases, contact lenses are fit based on the patient's refraction and keratometry reading. When HVID and sagittal depth are factored in, practitioners can improve patient comfort and vision by formulating a lens with better stability.

When customizing lenses, it is important to follow the fitting guidelines of each individual manufacturer. Lens materials have a large impact on the fitting characteristics of the lens.[2]

Custom-designed soft contact lenses will help produce loyal patients because most patients who need these lenses have given up on contact lenses. Simply listening to the patients and gathering more information can help practitioners fine-tune visual needs and personalize the fitting experience. With custom-designed contact lenses, practitioners are able to refine lens powers in smaller increments, prescribe cylinder axes to the exact degree, adjust multifocal zone sizes and specify the exact base curve and diameter of the lens to achieve the best vision and comfort.[3] RCCL

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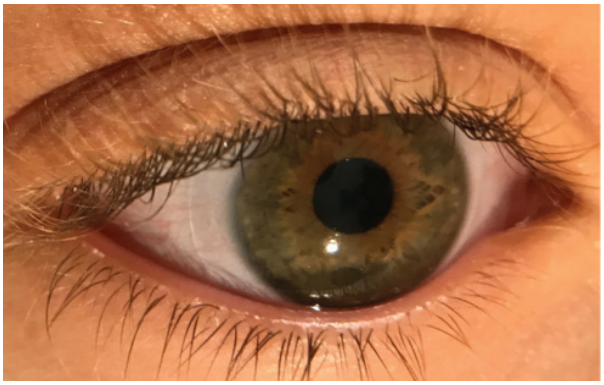

Fig. 4. The patient's new lens is well-centered with adequate cornea coverage.

By Mile Brujic, OD, and David Kading, OD

Let's Make It Less Complex

New scleral lens technology makes the fitting process more efficient than ever. Here's a look at what has changed.

Specialty contact lens designs help us restore significant visual function in patients with difficult prescriptions and corneal conditions. As these designs have flourished, so too have available options for these individuals. Several lens manufacturers aim to provide designs that improve predictability and efficiency in the fitting process.

Scleral lenses in particular have seen a number of advancements over the past several years. These lenses are intended to clear the cornea and are separated from it by a nonpreserved saline solution. As our knowledge of scleral lens fitting improves, the process has become easier. Here, we discuss the factors that have simplified scleral lens fitting for today's practitioners.

FILLING SOLUTIONS

For years, those fitting scleral lenses had few options when filling the lens bowl. Unisol 4 saline was popular for a while, but has been discontinued. Addipak saline, a preservative-free sodium chloride solution, was also widely used.

Recently, however, the release of two new FDA-approved filling solutions for scleral lenses has given practitioners more options. LacriPure (Menicon) is packaged in unit-dose vials containing 5mL of saline. This filling solution is ideal for both rinsing the lenses when removing them from multipurpose solutions and filling the lens bowl prior to lens insertion.[1] ScleralFil (Bausch + Lomb) is also FDA-approved for rinsing and filling the bowl of the lens prior to insertion.

APPROPRIATE CENTRAL CLEARANCE

Central corneal clearance can be measured a number of ways. The most commonly employed strategy is estimation at the slit lamp. Here, an optic section is created and the thickness of the lens is used to compare with the clearance.

The ideal corneal clearance is between 100µm and 300µm, and it will decrease throughout the day.[3] In one study, corneal clearance decreased from the initial amount by 83µm.[4] Frequently, we will attempt to fit these lenses initially closer to 300µm of clearance to allow for some expected settling throughout the day. Interestingly, the amount of settling is independent of the viscosity of the solution used to fill the lens prior to insertion.[5]

Although each scleral shape is unique, they tend to have an asymmetric profile that is flattest in the nasal region and steepest in the temporal and inferior regions.[5] Because scleral lenses rest entirely on the sclera, they often follow the steepest regions of the scleral and migrate infero-temporally.[6] Technology now allows us to visualize the central corneal clearance through optical coherence tomography (OCT) measurements. OCT cross-section scans through the lens, clearance and cornea provide a highly accurate look at the amount of clearance. This helps practitioners communicate the central corneal clearance with labs to determine any needed lens modifications.

OCT also provides unique views of the limbal region of the cornea and its relationship with the scleral lens. Practitioners can easily review a scan to confirm clearance in the limbal region (*Figure 1*).

POST-LENS CLOUDING

Clouding of the post-lens solution can be an irritating side effect for some of your scleral lens wearers. Studies show that visual quality tends to decrease over time when this happens.[7]

Post-lens clouding seems to occur when fluid enters and exchanges with the post-lens tear film. An

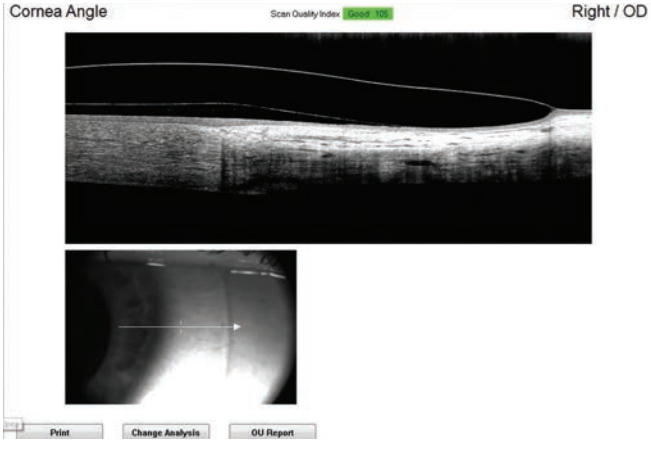

Fig. 1. Limbal clearance as seen on OCT.

inappropriate fit often causes an inadequate scleral lens fitting relationship with the conjunctiva and underlying sclera, allowing fluid exchange beneath the lens. To minimize tear exchange, some have tried using a more viscous preservative-free solution in the bowl of the lens prior to insertion. This can delay clouding, but in many cases it will still occur.

If you place a spherical landing zone on a toric sclera, there will be regions where the lens is either impinging upon the conjunctival tissue or elevated from the surface of the conjunctiva. For example, a spherical scleral lens on a sclera steeper vertically than horizontally may be elevated in the superior and inferior regions of the lens if it is aligned in the horizontal meridian. Here, the lens inappropriately aligns with the sclera.

This may occur inadvertently and cause fluid exchange under the lens in the vertical meridians. To combat this, some manufacturers have made toric peripheral curves more readily available in their scleral lens designs. This allows the lenses to better fit toric scleras. Additionally, this creates more of a chamber behind the lens, preventing fluid exchange and minimizing the effect on post-lens clouding. When used appropriately, toric peripheral curves can help alleviate a number of instances in which chronic clouding occurs.

MAPPING THE OCULAR SURFACE

Recent developments and advances in corneo-scleral topography provide new scleral lens fitting methods. Unlike placido-based corneal topographers, corneo-scleral topographers measure the entire anterior ocular surface, including the sclera. Elevation data is used to determine sagittal height and scleral shape. The sMap3D (Visionary Optics) corneo-scleral topographer creates a three-dimensional model of the eye and automated fitting software aids in designing a customized scleral lens (*Figure 2*). Customizable sMap3D options include toric haptics, quadrant specific haptics, multi-meridian (more than four quadrants), notches and edge vaults.

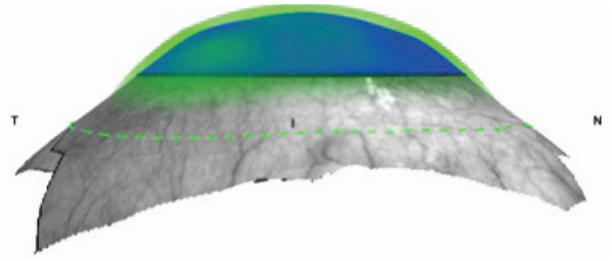

Image: Gregory DeNaeyer, OD

Fig. 2. The sMap3D allows practitioners to create a three-dimensional model of the eye.

LENS SURFACE DRYING

A major cause of discomfort for gas permeable (GP) lens wearers is lens surface drying. Because of this, solution manufacturers have optimized their multipurpose disinfecting solutions to help improve lens surface conditioning. Also, plasma treatments for GP surface materials have provided greater wettability.

Recently, a new treatment, Hydra-PEG (Tangible Science), has helped increase surface wettability for GP lenses.[8] This involves bonding polythylene glycol to activated GP surfaces, resulting in a lens surface completely encapsulated with Hydra-PEG polymers.[8]

Sclerals give practitioners the opportunity to improve vision for many patients who otherwise couldn't wear contact lenses. While these lenses require appropriate training, new technologies and designs have made them less complicated. As a result, the fitting process and patient wearing experience are more efficient than ever. RCCL

The authors would like to thank Gregory DeNaeyer, OD, for his contribution to this month's column.

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By Christine W. Sindt, OD

Rubbed the Wrong Way

Complications developed after epithelial debridement in a routine crosslinking procedure.

A 34-year-old male who had undergone LASIK in 2003 experienced progressive ectasia and was referred for corneal collagen crosslinking (CXL). Two weeks s/p, he presented with entrapped mucus in the epithelium. Superficial keratectomy was performed to clear the cornea, and a bandage contact lens was placed to alleviate pain and encourage healing. He was prescribed levofloxacin QID, prednisolone acetate 1% TID with taper and ibuprofen 800mg Q8hr PRN for discomfort. One week later, the patient presented with a central corneal ulcer, depicted here.

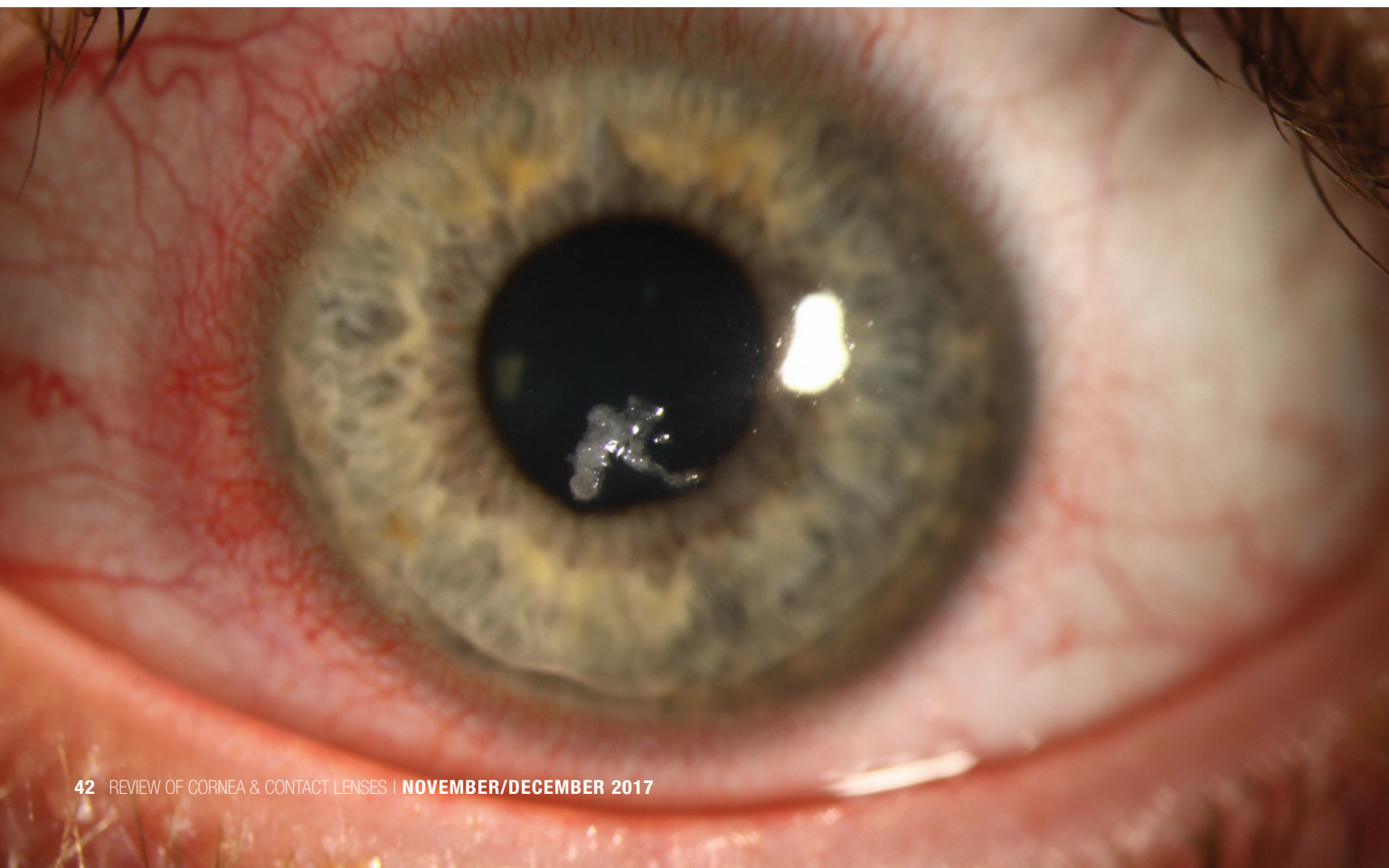
Pinpointing causality in the chain of events is difficult in such cases. This patient underwent the standard FDA-approved 'epi-off' CXL procedure, had an epithelial complication that required superficial keratectomy and, despite antibiotic coverage, developed ulceration.

In crosslinking, as in all surgical procedures, there may be unforeseen adverse events (AEs) postoperatively. The FDA trials of Photrexa and the KXL system (Avedro) reported corneal epithelial defect in 28% and keratitis in 3% of ectasia patients.[1] Other notable AEs in this group included corneal opacity/haze (71%), striae (9%), dry eye (14%), pain (26%), punctate keratitis (20%), photophobia (19%), reduced acuity (11%) and blurred vision (17%)—all expected sequelae after epithelial corneal debridement that occurred at a higher incidence than in controls.[1]

Epithelial defect, striae, punctate keratitis, photophobia, dry eye, pain and decreased acuity took up to six months to resolve, and corneal opacity/haze took up to 12 months.[1] In 6% of ectasia patients, corneal opacity remained at 12 months.[1] Looking at the study population as a whole, most other AEs were mild and resolved during the first month.[1]

Although risk of vision-impairing complications with CXL is low, clinicians should remain vigilant, particularly during epithelial regrowth when the cornea is most vulnerable to infection. Our patient was started on a broad-spectrum antibiotic while we await culture results. RCCL

1. Highlights of prescribing information, Photrexa Viscous and Photrexa, for use with the KXL System, Avedro. Revised Sept. 2017.



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