

# A case of managing a contact lens peripheral ulcer (CLPU).

Dr. Paymaun Asnaashari OD

## **Abstract**

Contact lens peripheral ulcer (CLPU) is a corneal ulcer that is stimulated from contact lens wear. Despite advancement in contact lens properties, patients are still at risk of developing contact lens-related complications. Even for the most hygienic and compliant contact lens patients, complications can still occur. Although the nature of CLPU is typically not sight-threatening, understanding the pathophysiology of CLPU is important and requires immediate management and monitoring. This case will outline a discussion involving the treatment and management for contact lens peripheral ulcer (CLPU).

**Keywords:** *Corneal ulcer, contact lens peripheral ulcer, CLPU, microbial keratitis, bacterial keratitis, viral keratitis, fungal keratitis, contact lens*

## Introduction

CLPU poses a diagnostic dilemma for optometrists for two reasons. The distinction between sterile inflammation and microbial infection is often not clear. CLPU is a less serious and typically not sight-threatening adverse effect of contact lens wear. On the other hand, Microbial keratitis (MK) is a very serious sight-threatening adverse effect of contact lens wear. It is estimated that there are over 70,000 cases of microbial keratitis annually in the US (1). Differentiating between MK and CLPU can often be blurred because both have similar presentations. Recognition and correct diagnosis is important for optimal outcomes.

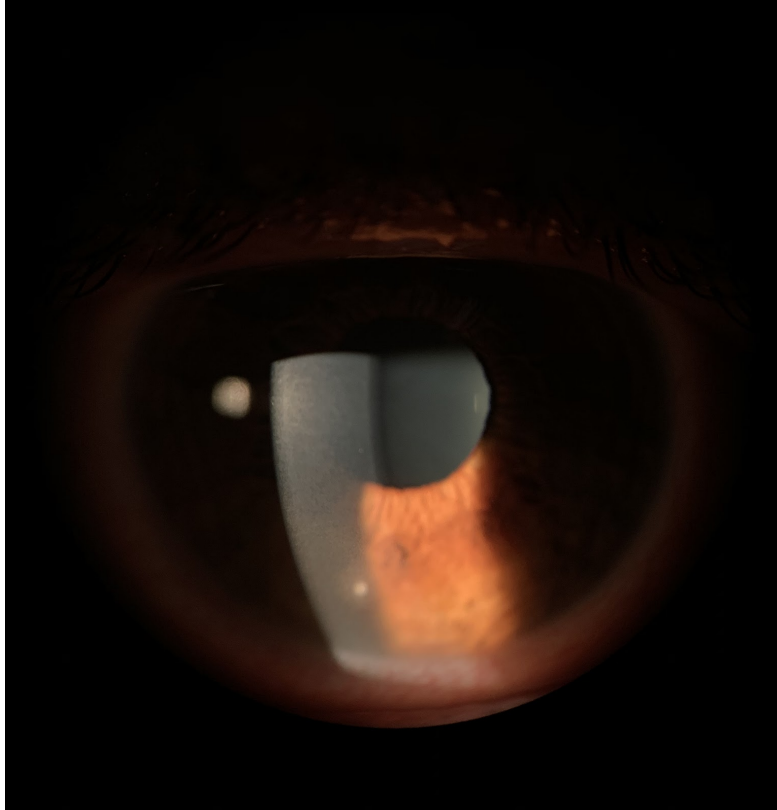
## Case Report

A 29-year old caucasian female presented with complaints of acute eye discomfort and redness of the right eye. The patient became symptomatic the day prior in the morning when she woke up. The patient was a current everyday contact lens wearer and denies poor compliance with lens wear. Despite waking up with discomfort and redness the day prior, the patient still instilled her contact lenses. This morning, her symptoms worsened. Discomfort level was 4 out of 10. Additional symptoms included excessive tearing and mucous discharge. The patient describes the mucous discharge as yellow, crusty and sticky debris. The patient denied changes in vision and photosensitivity. The patient's last eye exam was 1 year and 2 months ago. The patient's past ocular history and family ocular history were unremarkable. The patient is a habitual 2-week disposable soft lens wearer; wearing Acuvue Oasys OU. The patient does not report taking any medications and has no known drug allergies. The patient's blood pressure was not measured. The patient was oriented to time, place, and person. Color vision testing was not performed. Confrontation fields were normal OD and OS. Extraocular muscles were unrestricted in all gazes without pain or diplopia. Cover test was orthophoric at distance and near. Habitual spectacle correction measured via lensometry was -6.00 DS OD, -4.75 -0.25 x 035 OS. Her corrected visual acuity was 20/20 at distance in OD and 20/20 OS.

Non-contact tonometry measured 10 mmHg OD, 11 mmHg OS at 5:24pm. Anterior segment evaluation was performed using a slit lamp biomicroscope. The adnexae, lids, lashes, puncta, palpebral conjunctiva, iris and lens were normal in both eyes. The left bulbar conjunctiva and cornea were normal, however, the right eye revealed 2+ general hyperemia of the bulbar conjunctiva and small (about 1mm) subepithelial infiltrate at 6:00 in the mid-peripheral cornea. The corneal lesion displayed an opaque base with an overlying epithelial defect (about 0.25mm), which stained with sodium fluorescein. Anterior chambers of both eyes were quiet without evidence of cell or flare and angles were 4/4 via the Van Herrick method. Pupils were equally round and reactive to light, no afferent pupil defect was noted OU. No pupil dilation was performed. Posterior segment evaluation was performed using a slit lamp biomicroscopy with a 90D lens. Fundus assessment revealed optic nerves with a cup-to-disc ratio of 0.30/0.30 OD and OS. The cups were shallow; there was no evidence of pallor or edema or the neuroretinal

rim. Both macula's were flat and evenly pigmented. The vitreous was clear and the vasculature was normal in both eyes. Retinal periphery evaluation was not performed.

Figure 1 (Initial presentation)



The differential diagnoses considered at this point included:

- Corneal abrasion
- Acanthamoeba keratitis
- Microbial keratitis (MK)
- Contact lens peripheral ulcer (CLPU)
- Herpes simplex keratitis (HSV)
- Herpes zoster keratitis (HZV)
- Fungal keratitis
- Contact lens associated red eye (CLARE)
- Marginal keratitis

A corneal abrasion usually presents unilaterally with an acute moderate to severe ocular pain, hyperemia, epiphora, photophobia and reduced visual acuity depending on the location and size of the lesion. Corneal abrasions typically precede with a recent history of scratching or trauma to the eye. The epithelial defect stains with fluorescein in the absence of underlying corneal opacification. Depending on the severity of the abrasion, an AC reaction may be present.

Acanthamoeba keratitis presentation can vary from foreign body sensation to severe ocular pain (out-of-proportion to clinical signs), hyperemia, and photophobia over a period of several weeks. Often associated with a history of swimming or showering with contact lenses. Early clinical signs may be epitheliitis, pseudodendrites and subepithelial infiltrates. Later signs include ring-shaped stromal infiltration, epithelial defects, radial keratoneuritis, scleritis and anterior uveitis (with possible hypopyon). Advanced signs include stromal thinning and corneal perforation. Vision often can be affected depending on severity and stage.

MK is an infectious condition of the cornea. It usually presents unilaterally with ocular pain that can moderate to severe, photophobia, mucous discharge, hyperemia, reduced visual acuity, depending on the size and location of the lesion. The epithelial defect stains with fluorescein and is accompanied by underlying corneal opacification. Lesions tend to be large and more centrally located. An AC reaction typically is present. Predisposing factors include contact lens wear, ocular trauma, corneal surgery, ocular surface disease and immunosuppression.

CLPU is a non-infectious inflammatory event of the cornea. Inflammation occurs from accumulation of bacterial exotoxins on the surface of a contact lens. CLPU presentations are typically mild and unilateral; which include discomfort, foreign body sensation, hyperemia and tearing. Clinically they can appear very similar to MK; an epithelial defect is usually present with underlying opacification. However, lesions typically are small and peripheral and vision is unaffected. An AC reaction is typically not present. Predisposing factors include contact lens wear or history of extended wear use with contact lenses.

HSV keratitis usually presents with unilateral redness with variable degrees of pain or ocular irritation, often associated with epiphora and decreased corneal sensitivity. Punctate or dendritic epithelial lesions often stain with vital dyes. Unilateral eyelid vesicular rash can be present. Vision can be affected.

HZV keratitis usually presents unilaterally with similar symptoms as described in HSV. Pseudodendrities are typically seen within the cornea. Other signs include painful facial and skin lesions or rashes that respect the midline. These lesions form along the branches of cranial nerve V, particularly affecting the V1 and V2 dermatomes. Clinical signs may be preceded by headache, fever or malaise. Hutchinson sign predicts a higher risk of ocular involvement. Vision can be affected.

Fungal keratitis is a serious fungal infection of the cornea. Often associated with a history of minor trauma with vegetative matter, contact lens wear, chronic ocular surface disease or a history of poor response to conventional antibiotic therapy. Patients usually present with foreign body sensation or ocular pain, photophobia, hyperemia, epiphora and discharge. Satellite lesions may surround subepithelial infiltrates. A hypopyon and anterior chamber reaction can be present. There is potential for catastrophic visual outcomes.

CLARE is an inflammatory reaction that is stimulated by the presence of corneal hypoxia combined with noninvasive gram-negative bacteria from contact lens use. Symptoms include discomfort, contact lens intolerance and possibly mild pain. No corneal infection exists. Presentation can be unilateral or bilateral, consisting of conjunctival hyperemia and corneal infiltrates located in the periphery to midperiphery. The infiltrates have no overlying epithelial defect, distinguishing CLARE from CLPU and MK.

Marginal keratitis is an inflammatory reaction of the peripheral cornea in response to staphylococcal aureus antigens. Presentation includes stromal infiltrates which are often associated with epithelium break down or ulceration. Infiltration appears in areas of direct contact between the peripheral cornea and the eyelid margin. Majority of patients have longstanding blepharitis and meibomitis. Patients can complain of pain, foreign body sensation, photophobia and conjunctival injection. Vision is typically not affected.

The appearance of the corneal lesion in the right eye suggests a diagnosis of a CLPU based on the following; an isolated peripheral corneal lesion that consisted of an epithelial defect with underlying subepithelial infiltration. In addition, the patient was wearing contact lenses at the time of the incident. The patient's medical history was unremarkable and had no symptoms of pain, malaise and fever nor any abnormal skin lesion or any recent contact with vegetative matter. No AC reaction or mucous discharge were present and vision was stable. Furthermore, there was no punctate or dendritic epithelial staining present on the cornea. The patient was ordered to discontinue contact lens wear in both eyes and prescribed Vigamox QID in the right eye. Standard dosing for Vigamox is TID but QID dosing was prescribed for additional antibiotic prophylaxis as MK was part of the differential. The patient was scheduled to return for follow up in 2 days.

#### Follow up #1

The patient returned in 2 days for an anterior segment evaluation. The patient reported good compliance with Vigamox in the right eye. The patient's symptoms were significantly improved. The patient denied discomfort and discharge in the right eye. Visual acuity remained stable with corrected distance acuity of 20/20 OD and 20/20 OS. Slit lamp biomicroscopy of both eyes revealed normal lids, lashes and irides. The cornea and conjunctiva of the left eye were normal. No epithelial defect was observed in the right cornea with sodium fluorescein but mild underlying infiltration still remained. No AC reaction was present OU. Non-contact tonometry measured 11 mmHg OD, 11 mmHg OS at 5:30pm. Posterior segment evaluation was deferred in both eyes. The patient was advised to continue Vigamox QID OD and contact lens cessation. The patient was scheduled to return for follow-up in 1 week.

#### Follow up #2

The patient returned in 1 week for an anterior segment evaluation. The patient reported good compliance with Vigamox in the right eye. The patient had no visual complaints. Visual acuity

remained stable with corrected distance acuity of 20/20 OD and 20/20 OS. Slit lamp biomicroscopy of both eyes revealed normal lids, lashes, cornea, conjunctiva and irides. No staining was observed in the right eye with sodium fluorescein. No AC reaction was present OU. Non-contact tonometry measured 11 mmHg OD, 11 mmHg OS at 5:00pm. Posterior segment evaluation was deferred in both eyes. The patient was ordered to discontinue Vigamox. The patient was advised she could resume contact lens use one week from today's visit. The patient was doing well and no additional follow-up was ordered.

## **Discussion**

A corneal ulcer has the potential to be a vision-threatening ocular emergency. It can cause severe visual loss if untreated, which is why it is one of the leading causes of blindness worldwide (3). The annual incidence of corneal ulcers in the US is estimated to be between 30,000 and 75,000 (2). Corneal ulcers are much more common in those who wear contact lenses, especially extended wear lenses (2). It is estimated that 85 million people are using contact lenses worldwide today (3). It has been estimated that up to 66% of cases of corneal ulceration seen in the US and UK are contact lens-related (6). Studies have determined that extended wear of hydrogel lenses presents the highest risk for contact lens-related ulcerative keratitis (6). The risk of ulcerative keratitis with hydrogel extended wear is up to 10 to 15 times greater than during hydrogel daily wear. Patients can suffer significant complications that can lead to vision loss or blindness. Therefore, prompt management and treatment is essential.

Almost any organism can invade the corneal stroma if the normal corneal defense mechanisms or corneal epithelium are compromised (4). Microorganism infiltration such as bacteria, fungi, parasites or viruses can play an important role. The most common etiology of corneal ulcers involves bacterial pathogens (2). The most common bacterial pathogens are *Staphylococcus aureus* and *Pseudomonas aeruginosa* (2).

Corneal ulcers can be divided into infectious and non-infectious (aka 'sterile') categories. Differentiating between the two types is essential for any practitioner involved in managing these conditions. CLPU is a non-infectious, inflammatory reaction from contact lens wear. It is characterized as an acute adverse event observed with extended wear of contact lenses (8). It is characterized by moderate bulbar and limbal redness with the presence of a single, small (0.1 to 1.2mm in diameter), circular, subepithelial stromal infiltrate in the corneal periphery (8). The focal infiltrate is associated with overlying epithelial loss (8). CLPU occurs in response to bacterial exotoxins colonizing on the surface of the contact lens (7). Histological studies have shown that exotoxins originate from *staphylococcus aureus*, a gram-positive bacterium (8,9). When deposits develop on contact lenses, the lens surface becomes roughened, and epithelial defects develop as a result (9). This disruption of the epithelium provides an opportunity of entry for toxins into the corneal stroma, stimulating an inflammatory response, leading to focal infiltration and ulceration (9). The inflammatory response consists of infiltration with polymorphonuclear leukocytes (PMNs) and the infiltration is found to be localized just beneath Bowman's layer (8). Bowman's layer generally remains intact in CLPU, which helps to minimize

diffusion of exotoxins into the underlying stroma (8). In addition, an intact Bowman's membrane helps stromal defense mechanisms to prepare and cope with an invasion.

CLPU presentations are generally mild. When symptomatic, patients can complain of discomfort, foreign body sensation and tearing unilaterally (10). Corneal lesions have been shown to resolve upon removal of the contact lens without any antibiotics (8). The standard of care for CLPU is not clear; CLPU has been shown to resolve without treatment, however, because of the similarity to MK, conservative management with medical therapy is considered. A study showed patients with CLPU healed within 14 days without medical therapy, often leaving behind a scar (9). Because of the full thickness loss of the epithelium, CLPU can predispose an infection, therefore prophylactic treatment with antibiotics can be used for CLPU (6, 11, 12). As a result, the patient was treated with topical antibiotics to minimize the risk of developing MK. Topical antibiotics can also help minimize bacterial overgrowth on the lid margin and ocular surface which can help to quell the inflammatory response and bacterial exotoxins. Fluoroquinolones are recommended, preferably third or fourth generation, due to their broad spectrum profile and increased potency against gram-positive organisms (6,12,16). Topical steroids could also be used in management of CLPU due to the inflammatory nature of the condition (17,18). Corticosteroids have been used alone to treat CLPU, however the concern is that it can result in masking or enhancement of other infectious masqueraders. The SCUT study found adjunctive corticosteroid use may be associated with improved long-term outcomes in bacterial corneal ulcers not caused by *Nocardia* species; highlighting the safety of corticosteroids (19).

CLPU can occur with any type of contact lens (soft or hard) or wearing regimen (11, 12). Soft contact lenses pose a greater risk factor than rigid gas permeable lenses, and disposable extended-wear lenses have a greater association with peripheral corneal infiltrates than any other lens type (11). CLPU events are most often associated with extended wear of contact lenses (13,14). It is well documented that extended wear use of contact lenses significantly increases rates of corneal infiltrates (14). Other predisposing factors include sleeping in lenses, lens solution hypersensitivity, poor hygiene and poorly fitting lenses. Poorly fitting lenses can create mechanical insults to the cornea. In addition, increased bacterial load on lenses from blepharitis could also be a significant risk factor (12). Clinicians should carefully review the fit of the contact lenses and patient compliance with lens wear, lens replacement, disinfection protocols and counsel appropriately.

## **Conclusion**

CLPU are considered mild adverse reactions from contact lens wear. Often stimulated by extended wear of contact lenses. Events of CLPU are benign, noninfectious and often self-limited. However, clinical features of CLPU and MK can be very similar early on and the lack of clear distinguishing features necessitates events of CLPU to often be managed conservatively with medical therapy.

## References

1. "Bacterial Keratitis PPP - 2018." American Academy of Ophthalmology, 13 Nov. 2018, [www.aaopt.org/preferred-practice-pattern/bacterial-keratitis-ppp-2018](http://www.aaopt.org/preferred-practice-pattern/bacterial-keratitis-ppp-2018).
2. Byrd, Liza B. "Corneal Ulcer." StatPearls [Internet]., U.S. National Library of Medicine, 13 May 2019.
3. Loh, Ky, and P Agarwal. "Contact Lens Related Corneal Ulcer." Malaysian Family Physician : the Official Journal of the Academy of Family Physicians of Malaysia, Academy of Family Physician of Malaysia, 30 Apr. 2010.
4. Garg, P, and G N Rao. "Corneal Ulcer: Diagnosis and Management." Community Eye Health, International Centre for Eye Health, 1999.
5. Holden BA, La Hood D, Grant T, et al. Gram-negative bacteria can induce contact lens related acute red eye (CLARE) responses. CLAO J. 1996;22(1):47-52.
6. Grant T, Chong MS, Vajdic C, et al. Contact lens induced peripheral ulcers during hydrogel contact lens wear. CLAO J 1998 Jul; 24(3):145-151.
7. Silbert, Joel A. "Is It an Ulcer or an Infiltrate?" *Review of Optometry*, 3 July 2007, [www.reviewofoptometry.com/article/is-it-an-ulcer-or-an-infiltrate](http://www.reviewofoptometry.com/article/is-it-an-ulcer-or-an-infiltrate).
8. Holden B, Reddy M, Sankaridurg P, et al. Contact lens-induced peripheral ulcers with extended wear of disposable hydrogel lenses: histopathologic observations on the nature and type of corneal infiltrate. *Cornea*. 1999;18(5):S38-S43.
9. Wu P, Stapleton F, Willcox MDP. The causes of and cures for contact lens-induced peripheral ulcer. *Eye Contact Lens*. 2003;29(18):S63-S66.
10. "Contact Lens-Associated Red Eye: Causes and Corrections." *Review of Optometry Continued Education*, 2017, [www.revieweducationgroup.com/ce/contact-lens-associated-red-eye](http://www.revieweducationgroup.com/ce/contact-lens-associated-red-eye).
11. Donshik, P C, et al. "Peripheral Corneal Infiltrates Associated with Contact Lens Wear." *Transactions of the American Ophthalmological Society*, U.S. National Library of Medicine, 1995.
12. Keir, Nancy. "Contact Lens-Induced Peripheral Ulcers." *Epubs.democratprinting.com*, 2011, [epubs.democratprinting.com/article/Contact+Lens-Induced+Peripheral+Ulcers/701281/0/article.html](http://epubs.democratprinting.com/article/Contact+Lens-Induced+Peripheral+Ulcers/701281/0/article.html).
13. Dumbleton K. Adverse events with silicone hydrogel continuous wear. *Cont Lens Anterior Eye*. 2002 Sep;25(3):137-46.
14. Szczotka-Flynn L, Diaz M. Risk of corneal inflammatory events with silicone hydrogel and low dk hydrogel extended contact lens wear: a meta-analysis. *Optom Vis Sci*. 2007 Apr;84(4):247-56.
15. *The Wills Eye Manual: Office and Emergency Room Diagnosis and Treatment of Eye Disease*, 7th Edition. (Philadelphia, Pa.) Wills Eye Hospital, et al. Lippincott, Williams & Wilkins, 2017.
16. Wong, Raymond, et al. "New Treatments for Bacterial Keratitis." *Journal of Ophthalmology*, Hindawi, 6 Sept. 2012, [www.hindawi.com/journals/joph/2012/831502/](http://www.hindawi.com/journals/joph/2012/831502/).



17. Carnt, Nicole, et al. "The Diagnosis and Management of Contact Lens-Related Microbial Keratitis." Wiley Online Library, John Wiley & Sons, Ltd, 16 Aug. 2017, [onlinelibrary.wiley.com/doi/full/10.1111/cxo.12581](http://onlinelibrary.wiley.com/doi/full/10.1111/cxo.12581).
18. Baum, J, and O H Dabezies. "Pathogenesis and Treatment of 'Sterile' Midperipheral Corneal Infiltrates Associated with Soft Contact Lens Use." Cornea, U.S. National Library of Medicine, Nov. 2000.
19. Srinivasan, Muthiah, et al. "The Steroids for Corneal Ulcers Trial (SCUT): Secondary 12-Month Clinical Outcomes of a Randomized Controlled Trial." American Journal of Ophthalmology, U.S. National Library of Medicine, Feb. 2014.