



# A Handbook of Contact Lens Management

4<sup>TH</sup> EDITION

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# About This Handbook

As contact lens (CL) designs and materials have developed over the years, more patients than ever can now wear CLs successfully. With new lens materials that differ in their characteristics and interaction with lens care solutions, the role of the eye care professional is more important than ever in selecting the optimal combination to deliver sustained comfort and minimal physiological impact on the wearer. Thorough aftercare plays a critical role in this management as many of the tissue changes that may occur during CL wear are asymptomatic. Comprehensive aftercare and follow-up enable practitioners to ensure continued healthy contact lens wear by early detection and appropriate management to minimize adverse events, increase retention to lens wear and increase patient satisfaction.

The primary purpose of this clinical resource is to support practitioners with guidelines in monitoring, recording and taking appropriate action during the care of their CL patients. It is intended to be a clinical guide for everyday clinical practice on viewing the individual structures affected by CL wear, with examples of the changes that might occur. This guide provides information on soft, rigid corneal (formerly known as rigid gas permeable, RGP or GP lenses; name updated in line with new recommendations), orthokeratology and basic scleral lens patient management. Also provided

are possible causes, signs, symptoms and management strategies. Each condition is illustrated with examples of its typical clinical appearance, some of which have been arranged in order of severity. This, along with recommendations for descriptive recording of the pathology, provide an idea of how to grade and monitor the progress of the condition. In many cases, validated grading schemes exist to support best-practice medical record completion and use of those scales is recommended where possible.

The first three editions of the handbook were printed reference guides. The new fourth edition is available in print but is also available online for the first time, making it accessible from all digital devices, providing searchable menus to enhance the user experience. Embracing the opportunities afforded by moving to an online format, video content depicting clinical techniques and examples of pathologies have been added for the first time. The content has been reviewed and updated in collaboration with the Centre for Ocular Research & Education (CORE) at the University of Waterloo, Canada.

**John Meyler BSc (Hons), FCOptom, DipCLP. Global Head, Professional Education & Development. Johnson & Johnson Vision**

# Slit Lamp Examination of the Eye

In order to be successful, a CL must provide the wearer with optimum vision and comfort whilst maintaining the normal physiology and ocular integrity of tissues which may be affected by lens wear. Recent advances in CL designs, materials, care systems and manufacturing techniques are making it possible for more and more patients to wear CLs. To maintain the patient as a successful CL wearer, the practitioner must make provision for thorough aftercare and regular check-ups. Not all CL induced changes are symptomatic, hence it is essential for the practitioner to regularly monitor the eye objectively. The slit lamp biomicroscope provides one of the most important tools in achieving this.

Slit lamp biomicroscopy is an essential aspect of pre-assessment of the potential CL wearer (neophyte) and aftercare of the existing wearer. Practitioners must carry out a physical assessment of those tissues which can be affected by CL wear, including the cornea, conjunctiva, limbus, lids and tear film.

The slit lamp examination of the neophyte has two purposes: to assess the suitability of the eye for CLs and to provide baseline data to which any changes during the course of CL wear can be compared. Furthermore, in the fitting process, the slit lamp has a role in assessing the physical fit of rigid and soft lenses in situ. In CL aftercare, the slit lamp provides the means to objectively judge the interaction between the lens and the eye, as well as a crude means of assessing lens deposition and in-eye wettability.

## Calibration

The correct set-up of the biomicroscope is essential for a valid examination. The illumination and observation systems must be coupled and in focus for the observer. The patient must be seated comfortably, with their chin in the rest, head firmly against the headrest and eye level at the centre of the vertical travel of the instrument. The stages needed to achieve this are as follows:

### (i) Instrument focusing

Using the focusing rod provided with the slit lamp ensures that a narrow slit beam is seen clearly in focus through each eyepiece individually. Then adjust the inter-pupillary distance of the instrument, binocularly. Assuming that only one person is using the instrument, this procedure only needs repeating periodically (*Figure 1*).

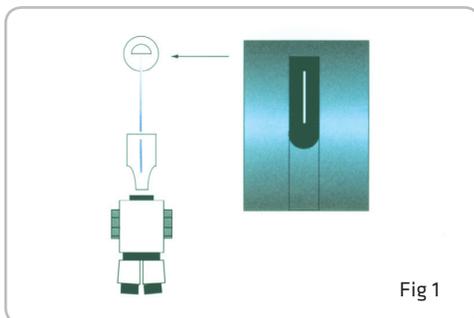
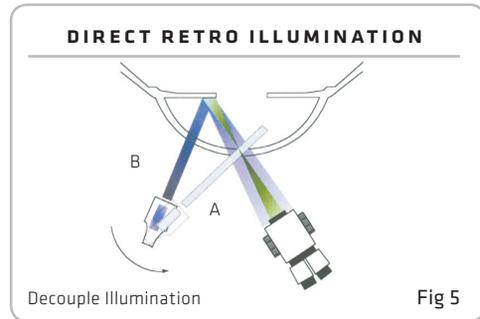
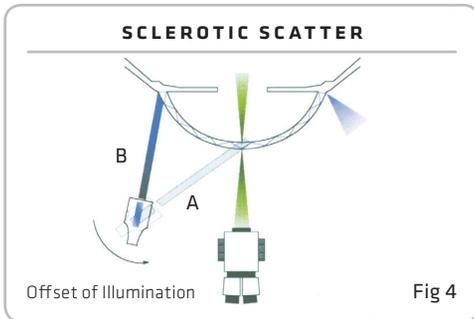
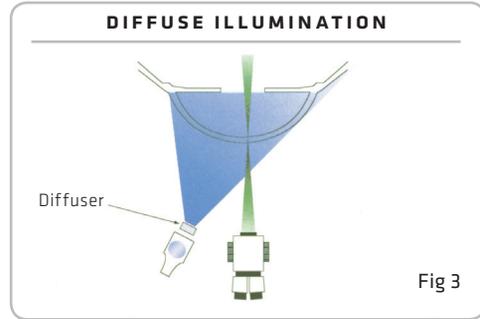
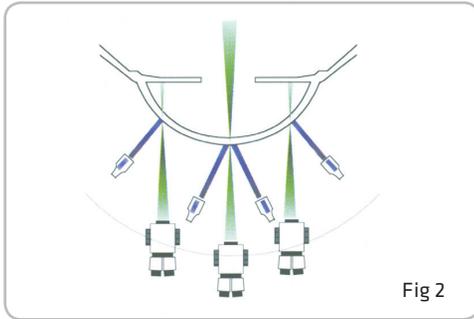


Fig 1



## (ii) Patient position

It is important to explain to the patient the nature of the examination and ensure that they are seated comfortably as described earlier. This is critical because if the patient is uncomfortable, the examination becomes significantly more difficult. Similarly, if the eye level is not in the middle of the instrument's vertical travel, the examiner may have difficulty examining the inferior and superior parts of the eye. Most slit lamps have a notch on the headrest which should be lined up with the outer canthus of the eye for optimal positioning.

## (iii) Focusing check

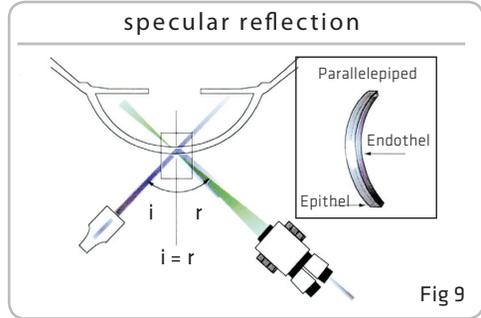
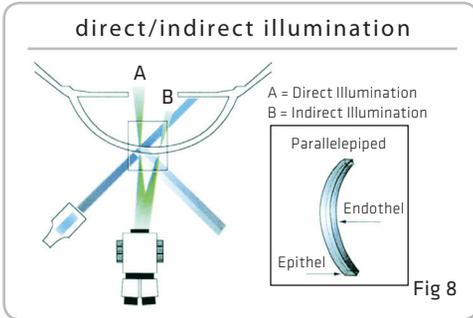
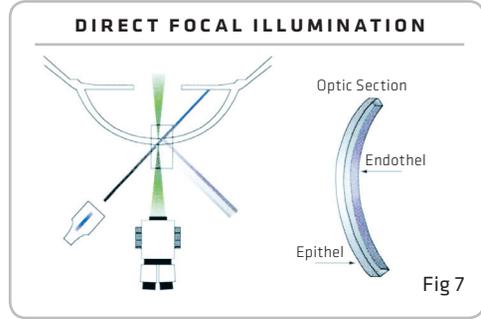
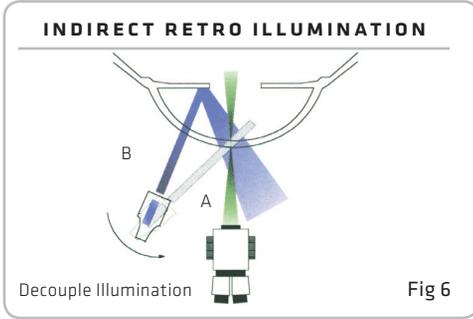
With the eyelids closed, the examiner should now focus the light on the lids and check its focus by rotating the illumination system round from side to side. As it rotates, the light should remain stationary on the lid. If it is showing relative movement, then the instrument is not in focus. With the slit lamp now focused and coupled with the illumination system, the whole cornea can be viewed in focus (Figure 2). As with many aspects of CL and ocular examination, the practitioner should

develop a routine to ensure that the examination is conducted in a thorough, logical and consistent manner.

## Slit Lamp Routine

### (i) Overall view: low magnification, wide diffuse beam (Figure 3).

The practitioner should carry out a number of sweeps across the anterior segment with a broad beam and low magnification. Starting with the lids closed, the lid margins and lashes should be examined for signs of blepharitis or hordeolum. Next, with the eyes open, the lid margins should be examined for patency of the tear drainage ducts and general appearance of the meibomian glands. Once upper and lower margins have been examined, the practitioner should review the bulbar conjunctiva to assess hyperaemia and the presence of Pinguecula or pterygia. The illumination should also be used to view the superior and inferior palpebral conjunctiva for hyperaemia, follicles and papillae, although this should ideally not be until the end of the routine to minimize disruption of the tear film during lid eversion.



**(ii) Corneal and limbal examinations: medium magnification, 2mm wide beam**

The corneal examination typically starts by placing the slit at the limbus and, with room lights off, observing the cornea using sclerotic scatter to search for gross opacifications or central corneal clouding produced by rigid lens wear (Figure 4).

The viewing system needs to be uncoupled from the illumination system if the cornea is to be viewed under magnification by this means, although viewing with the naked eye may be sufficient. Once the cornea has been examined by sclerotic scatter, the illumination and viewing systems need to be re-coupled and a series of sweeps carried out across the cornea. The practitioner should start by moving around the limbus looking at limbal vasculature to assess the degree of physiological corneal vascularization (blood vessels overlaying the cornea) and differentiate from neovascularisation (new blood vessels growing into clear cornea). Blood vessels are seen in both direct retro-illumination (Figure 5[KW6]), looking directly at the area of cornea illuminated, or indirect, retro-illumination (Figure 6), looking to the side of the illuminated cornea. Utilizing a red-free (green)

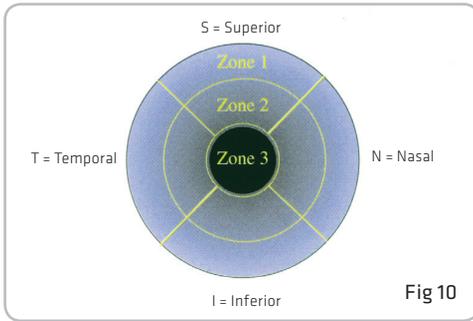
filter aids in the detection of vascularization. The practitioner should also look for peripheral infiltrates or dellen during this part of the examination.

**(iii) Corneal examination: high magnification, narrow beam**

It is at this stage of the examination that the slit width is reduced to its minimum, allowing the practitioner to view the cornea in cross section (Figure 7). [KW7] With high magnification, the cornea is swept systematically, and a routine is essential to ensure that none of the cornea is missed.

In addition to looking for opacifications and recording their depth and location, the practitioner also looks for microcysts (Figure 8[KW8]), striae and folds. During the aftercare of a soft lens wearer, this process should be one of the first parts of the slit lamp examination to be carried out, as signs of oedema disappear shortly after lens removal.

The final aspect of the corneal examination under white light and high magnification is observation of the endothelium. This is one of the most difficult corneal structures to examine; even at 40 times magnification,



only a gross clinical judgment can be made as it is not possible to view individual cells in detail and only a small area of the endothelium will be seen at any one time. The technique employed for viewing the endothelium is specular reflection. This uses a slightly broadened slit lamp beam and sets up the illumination system and microscope such that the angle of incident light is equal to the angle of reflection (*Figure 9[KW9]*). The area of specular reflection is only visible monocularly. As the practitioner focuses on the back of the corneal section, the endothelium comes into view as a mosaic of cells with a dull gold color appearance.

#### (iv) Fluorescein examination

It is essential that the cornea and conjunctiva be examined following vital stain instillation both prior to CL fitting and at every aftercare appointment. Sodium fluorescein is a vital dye which stains damaged epithelial tissue and is the best means of judging corneal integrity, in addition to the assessment of tear film stability, conjunctival integrity and palpebral conjunctival roughness. Practitioners should not shy away from using fluorescein in soft lens wearers as it will reveal changes in corneal integrity which could not otherwise be seen. Although fluorescein has the potential to stain hydrogel lenses, only a small amount is needed in the tear film to observe any disruption to corneal integrity. A fluorescein strip is first wetted with sterile saline, shaken clear of excess fluid and lightly touched onto the bulbar conjunctiva. During the examination, the dye will dissipate quickly to allow soft lenses to be applied to the eye within 10 minutes, without risk to the lens. The appearance of fluorescein in the eye can be enhanced by placing a yellow barrier filter in the observation pathway of the slit lamp. This acts to filter away blue light to make the fluorescent green stand out clearer.

Lissamine green staining is ideal for evaluating conjunctival integrity during CL and dry eye assessments and shows greater specificity for symptomatic patients than fluorescein. It is best viewed with white light, and can be made more visible by use of a red filter in the observation pathway. Lissamine green avoids the stinging associated with the use of the vital dye Rose Bengal while being equally effective. A mixture of 2% fluorescein and 1% lissamine green aids in assessing the cornea and conjunctiva to avoid having to instill the two dyes separately.

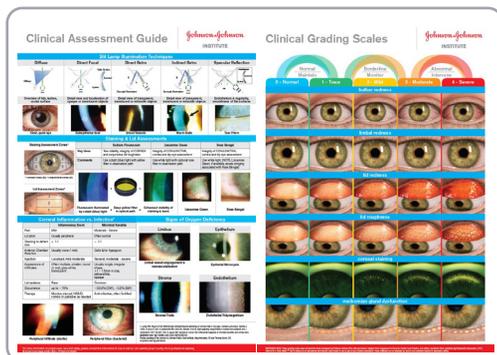
### Recording The Results

Of equal importance to carrying out the examination is the recording of the results. Legally, if an action is not recorded, it is not deemed to have taken place. It is not sufficient to say “Cornea clear” or “N.A.D.”; the practitioner must attempt to record and quantify what is seen. With a graticule in situ, some conditions can be measured, while others can be graded. Corneal location can be recorded with reference to zones of the structure (*Figure 10*). In this handbook, the key structures are listed and a recommended grading system shown for many of the complications.

### Grading Scales

In addition to the use of this digital resource, there are several other widely available grading scales for use in clinical practice to more accurately and concisely record and monitor any physiological changes. Examples of validated scales include the Brien Holden Vision Institute (BHVI; formerly CCLRU) and the Efron grading scales. The former was first published in 1993 and uses four photographic images (corresponding to grades 1 to 4) for a range of conditions which may occur during CL wear; in 2017, a more clinician-oriented version of the BHVI scale was introduced, which shows updated reference photographs for a smaller number of complications. The Efron scales (first edition 1997) uses five pictorial images (corresponding to grades 0 to 4). While there are differences in the number of reference images shown for these scales, both scales are intended to be used by using grades that range from 0, where no clinical action is required, through to grade 4, where clinical action is urgently required. The use of these scales has been clinically validated, with practical yet

highly sensitive ranges for a variety of conditions. Management will be based on how much the normal ocular appearance has changed, with a grade of 2 or less typically considered within normal limits. It has been recommended that, for maximum precision, recording of clinical signs using grading scales should be undertaken to the nearest 0.1 scale units and that a change in grading scale units of 1.0 is typically considered clinically meaningful, with Efron recommending a change of 0.7 units for the use of his scale. Grading scales are a very useful clinical tool with a common language for improved accuracy, allowing direct comparisons, enhancing record keeping and are sensitive to monitoring changes. In addition, they can also be useful as an educational tool to help explain changes to the patient and so keep them fully informed. THE JOHNSON & JOHNSON INSTITUTE (JJI) has developed a more condensed version of the full Efron Scales as a readily accessible chair-side reference (*Figure 11*), to include six key ocular signs that practitioners regularly grade in clinical practice, and a useful clinical assessment guide summary. While clinicians are free to choose the grading scale that they feel most comfortable with, it is crucial to consistently use the same grading scale and not to interchangeably use different grading scales, due to inherent differences between the available scales.



## Other Investigative Techniques

During the aftercare of CL wearers, there may be signs and symptoms that indicate other investigative techniques are required. It should be remembered that CL wearers may also exhibit non-CL related pathology, which may require appropriate diagnosis and management. For this differential diagnosis, the following investigative techniques may need to be used:

- Slit lamp – Van Herrick’s technique (estimation of anterior chamber and angle depth)
- Slit lamp – gonioscopy (examination of anterior chamber angle)
- Slit lamp – examination of crystalline lens, vitreous or fundus (using plus power condensing lenses)
- Keratometry (investigation into corneal conditions such as keratoconus, corneal warping)
- Pachymetry (assessment of corneal oedema during CL wear, prior to refractive surgery or monitoring keratoconic patients)
- Viewing of tear film via interferometric techniques
- Fundus examination (conditions affecting visual acuity unexplained with slit lamp biomicroscopy)
- Retinoscopy (over-refraction, detection of keratoconus)
- Tonometry (applanation or non-contact for differential diagnosis of acute glaucoma with a red eye)
- Perimetry (conditions affecting visual acuity unexplained by slit lamp biomicroscopy)
- Amsler Chart (for assessment of unexplained changes to central vision)

Fig 11 The Johnson & Johnson Institute Clinical Grading Scales and Assessment Guide

## Imaging Techniques

Anterior segment imaging techniques have been developed to accurately record clinical findings which both allows clinicians to monitor conditions over time and demonstrate specific points to patients. Enhanced imaging can visualize tissue not visible on the slit lamp. The following imaging techniques may be used to monitor CL wearers:

- Slit Lamp photography and video recording
- Digital camera with anterior segment imaging
- Meibography with infrared imager
- Tear Interferometry (assessment of lipid layer thickness)
- Corneal topography (orthokeratology, post-refractive surgery fitting, keratoconus management)

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3. Schulze, M et al. The use of fractal analysis and photometry to estimate the accuracy of bulbar redness grading scales. *Invest Ophthalmol Vis Sci* 2008; 49(4), 1398-1406

# Lids & Lashes

## Blepharitis

### Slit lamp viewing

Diffuse beam, low magnification (10x), direct illumination

### Incidence

- Generally not CL related, although may impact lens comfort and wear time
- True incidence unknown, although common
- Staphylococcal blepharitis – younger, more common with females
- Seborrhic blepharitis – generalized condition involving scalp, brow and face
- Demodex blepharitis – more common in CL wearers than non-wearers, mites in eyelash follicle, incidence of Demodex infestation increases with age.

### Aetiology

- Staphylococcal most common – in isolation or with sebhorreic or as local immune reaction
- Seborrhic (oily) can exist in isolation – disorder of glands of Zeiss or Moll
- Demodex blepharitis results from active Demodex infestation of lash follicles
- Associated with MGD and increased presence of gram-positive organisms on the lid margin

### Symptoms

- Foreign body sensation, itching, burning, gritty, lids stuck together on waking, mild photophobia
- Dryness symptoms (especially morning)
- Lens intolerance. Often little correlation with signs

### Signs

- Hard crusts at base of lashes (rosettes)
- Cylindrical dandruff or collarette (signs of Demodex)
- Dilated vessels lid margin (telangiectasis)
- Lid margin notching and thickening (tylosis)
- Loss and whitening of lashes (madarosis and poliosis)
- Misdirected lashes
- Lid margin redness and oedema, hordeolum, keratoconjunctivitis sicca (50%)
- Superficial inferior punctate epithelial erosions, peripheral corneal infiltrates (nasal and temporal due to hypersensitivity to staphylococcal antigens)

## Management

- Manage  $\geq$  grade 2 or if symptoms occur
- Temporary cessation of lens wear if grades 3 or 4 until resolved
- Risk factor for corneal inflammation (pathogenic microbes at ocular surface); treat prior to CL fitting; chronic condition contraindication to overnight wear
- Warm compresses with commercially available eye masks and lid hygiene with commercially available lid scrubs to be carried out 2–3 times a day
- Lid wipes/foams, and if for Demodex blepharitis they must contain an agent that can eradicate Demodex (eg: Terpinen-4-ol derived from Tea Tree Oil)
- In office microblepharon exfoliation
- Ocular lubricants
- Antibiotic therapy may be required in staphylococcal blepharitis (oral tetracycline, topical fusidic acid gel or bacitracin ointment)
- Daily Disposables (DD) advisable (or lenses with a shorter replacement frequency)

## Prognosis

- All types typically respond well to correct treatments, but can reoccur and/or be chronic in nature, requiring ongoing lid hygiene maintenance regimen.
- Differential diagnosis
- Ocular rosacea
- External Hordeolum
- Chalazion



Figure 1: Primarily Staphylococcal Blepharitis, with evidence of some Demodex collarettes too



Figure 3: Cylindrical dandruff/collarettes of Demodex Blepharitis



Figure 2: Exposed Demodex tail visible in centre of image

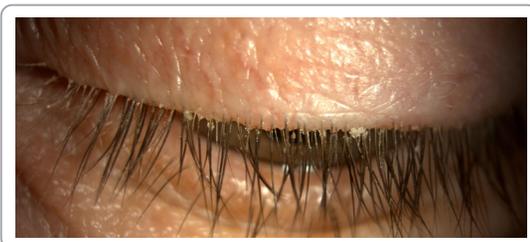


Figure 4: Seborrheic Blepharitis

## Describing clinical appearance

- 0: No debris/scaling/collarettes
- 1: Trace debris/scaling/collarettes
- 2: Some debris/scaling/collarettes; faint lid hyperaemia and telangiectatic vessels
- 3: Moderate debris/scaling/collarettes, lid hyperaemia and oedema, telangiectasia vessels
- 4: Marked debris/scaling/collarettes, lid hyperaemia and oedema, telangiectatic vessels

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# Lids & Lashes

## Meibomian Gland Dysfunction (MGD)

### Slit lamp viewing

Diffuse beam, medium magnification (16x), direct illumination

### Incidence

- 4 – 20% (Caucasian population) to over 60% (Asian population);
- Increases with age, blepharitis and rosacea

### Aetiology

- Chronic, diffuse abnormality of meibomian glands (MG), with duct obstruction and/or qualitative/quantitative changes in glandular secretion.
- Progressive inflammatory process associated with blepharitis, mechanical trauma, lowering temperature of eyelids, microbial contamination, CL wear and make-up.

### Symptoms

Ocular discomfort, dryness, irritation, itching, CL intolerance, smeary vision (greasy lenses), photophobia

### Signs

- Absent or cloudy meibomian gland secretions on gland expression
- Frothy tears with reduced tear film quality and break up time
- Thickened lid margins with distorted, possibly capped, meibomian glands

- Marginal dry eye signs (inferior corneal staining, recurrent corneal erosions) clinically apparent inflammation and ocular surface disease
- Discrete lipid deposits or greasy lipid layer over lens surface

### Management

- Explain chronic nature of condition to patient
- Manage > grade 2 or if symptoms
- Lens wear can be continued if tolerated
- Daily eyelid hygiene including warming with proprietary masks followed by moderate to firm massage and expression of MG secretions
- In office microblepharon exfoliation
- Device assisted thermal pulsation and expression in office
- Consider CLs with shorter replacement frequency
- Artificial tears
- Advice on diet (increase omega-3 fatty acid intake), effect of work/ home environments on tear evaporation and possible drying effect of certain systemic medications
- If severe, systemic tetracyclines may be necessary

### Prognosis

Generally good resolution of symptoms and improvement in tear quality following treatment.

### Differential diagnosis

- Hordeolum (external/stye and internal/ meibomian cyst) and chalazion
- Associated with reduced TBUT and inferior corneal staining

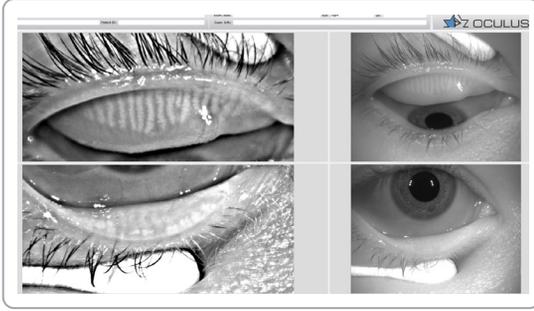


Figure 1: Meibography example showing gland drop out, more severe in lower than upper lid



Figure 2: High magnification view of telangiectasia, capped meibomian glands and irregular lid margin



Figure 3: Telangiectasia, capped meibomian glands

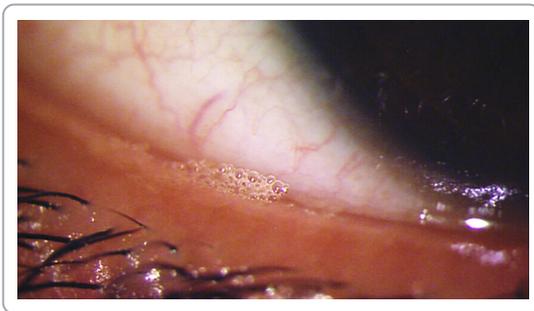


Figure 4: Example of frothy tears

## Describing clinical appearance

### Diagnostic expression

- Count the number of visibly capped glands
- Grade meibum quality.

Use finger, Q-tip or Meibomian Gland Evaluator (MGE) to assess 5 glands in each of 3 areas: nasal, central and temporal. Look for number of glands releasing meibum and grade meibum quality (0-45 score). Maximum score is 45 across 15 glands. NOTE: this is a reverse scale where the highest 'grade' is given to the best appearance:

- Grade 3 Liquid, clear
- Grade 2 Liquid milky
- Grade 1 Thick (toothpaste/ inspissated)
- Grade 0 No secretions

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# Lids & Lashes

## Lid Wiper Epitheliopathy (LWE)

### Slit lamp viewing

- Diffuse beam, medium magnification (around 16x).
- 2 drop instillation of lissamine green and observe 1-5 mins later; or 2 drop instillation of fluorescein and observe with cobalt blue filter 3- 5 mins later

### Incidence

- Up to 85% of habitual soft lens wearers
- 88% of non-CL wearers with dry eye symptoms (32%  $\geq$  grade 2) and 16% of asymptomatic non-wearers
- Can occur in the absence of positive dry eye test findings
- Correlates with lid parallel conjunctival folds (LPCOF), tear film stability and tear volume, mucin quantity, bulbar and limbal hyperaemia, ocular surface staining and dry eye symptoms
- More common with rigid corneal (RCL) and SiHy lenses

### Aetiology

- Alteration in epithelium of advancing lid margin due to friction during lid movement across the lens surface
- In dry eye, tear film thickness insufficient to separate ocular surface and lid wiper
- Other causes include blinking disorders, lid and ocular surface abnormalities

### Symptoms

Increased lens awareness, scratchiness on blinking, reduced wearing time

### Signs

Characteristic staining at upper and lower lid margin

### Management

- Manage  $\geq$  grade 2 or if symptoms present
- Refit with more lubricious lens surface (lower coefficient of friction)
- Change lens type (rigid corneal (RCL) to SiHy or hydrogel) or wearing schedule (increase replacement frequency)
- Maintain good lens cleaning including rub and rinse step
- Manage any tear quality issues
- Rewetting drops

### Prognosis

Generally good resolution of symptoms with appropriate management, although signs may remain

### Differential diagnosis

Staining associated with Marx line



Figure 1: Upper lid wiper epitheliopathy with lissamine green



Figure 2: Higher magnification view of upper lid wiper epitheliopathy with lissamine green



Figure 3: Lower lid wiper epitheliopathy with lissamine green

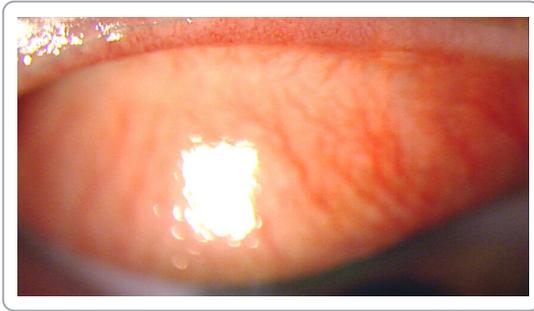


Figure 4: Differential diagnosis: Marx's line

## Describing clinical appearance

Mean of grade for staining length (mm) and grade for % average sagittal staining width

### Length

- 0: <2mm horizontal length of staining
- 1: 2-4mm horizontal length of staining
- 2: 5-9mm horizontal length of staining
- 3: >9mm horizontal length of staining

### Width

- 0: <25% average sagittal width of staining
- 1: 25-50% average sagittal width of staining
- 2: 50-75% average sagittal width of staining
- 3: >75% average sagittal width of staining

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32. McMonnies C. Incomplete blinking: exposure keratopathy, lid wiper epitheliopathy, dry eye, refractive surgery, and dry contact lenses. *Cont Lens Ant Eye* 2007; 30(1): 37-51
33. Muntz A et al. Cytomorphological assessment of the lid margin in relation to symptoms, contact lens wear and lid wiper epitheliopathy. *Ocul Surf* 2020; S1542-0124(19) 3030: 1-5
34. Pult H et al. The relationship between clinical signs and dry eye symptoms. *Eye* 2011; 25(4): 502-10.
35. Schulze M et al. Lid Wiper Epitheliopathy in Soft Contact Lens Wearers. *Optom Vis Sci* 2016 August; 93(8): 943-954
36. Varikooty J et al. Atypical manifestation of upper lid margin staining in silicone hydrogel lens wearers with symptoms of dry eye. *Cont Lens Ant Eye* 2008; 31(1): 44-6
37. Yeniad B et al. Lid- wiper epitheliopathy in contact lens users and patients with dry eye. *Eye Contact Lens* 2010; 36(3): 140-3

# Bulbar Conjunctiva

## Pinguecula

### Slit lamp viewing

Diffuse beam, medium magnification (16x), direct illumination

### Incidence

- Unknown, increases with age
- Not CL related, although may be aggravated by lens edge

### Aetiology

- Degenerative sub-epithelial collagen often associated with calcification
- Associated with excessive exposure to hot, dry, windy climates and UV radiation

### Symptoms

Typically none, although may cause dryness and discomfort with CL wear.

### Signs

Raised yellowish nodule on bulbar conjunctiva (nasally and temporal adjacent to limbus)

### Management

- Not a contraindication for CL wear
- Avoid mechanical disturbance for comfort reasons
- Ocular lubricants can be helpful
- Use of UV protection with wrap-around sunglasses to prevent further deterioration

### Prognosis

Generally remains constant, not affected by CL wear  
If inflamed may require medical treatment

### Differential diagnosis

- Pterygium
- Vascularised limbal keratitis (VLK) – chronic inflammatory complication due to rigid lens design resulting in elevated, semi-opaque epithelial lesion at limbus with conjunctival hyperaemia, corneal staining and neovascularisation



Figure 1: Prominent temporal Pinguecula

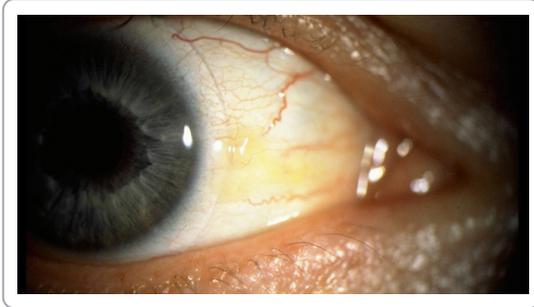


Figure 2: Pinguecula interacting with soft contact lens edge

## Describing clinical appearance

Position: nasal/temporal

- 0: None visible
- 1: Slightly raised area <0.5mm
- 2: Slightly raised area 0.5 – 2.5mm
- 3: Raised yellow area
- 4: Unsightly

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# Bulbar Conjunctiva

## Pterygium

### Slit lamp viewing

Diffuse beam, medium magnification (16x), direct illumination

### Incidence

- Environment affects development - incidence varies depending on geographical location (levels UVR exposure) and if closer to equator, more likely to develop pterygium
- Prevalence rates vary from < 2% in upper latitudes to 36% in lower latitudes.
- Heredity affects incidence (more common in persons of Spanish and Oriental origin)
- Not CL related but may impact CL comfort and wear time

### Aetiology

Degenerative collagen bundles in bulbar conjunctiva due to excessive exposure of the bulbar conjunctiva to hot, dry, windy climates and/or UV radiation

### Symptoms

- Some discomfort with or without lens wear, dryness
- Cosmetic concerns
- Vision affected if becomes large

### Signs

- Triangular growth fibrovascular tissue on bulbar conjunctiva, usually nasal, which encroaches onto cornea and destroys Bowman's membrane
- Often bilateral

### Management

- Only if discomfort occurs or if it interferes with vision – avoid mechanical trauma
- Not a contraindication for CL wear
- Occasional use of vaso-constrictors and ocular lubricants
- If severe, surgical removal may be required, followed by application of steroids, Mitomycin C or amniotic membrane

### Prognosis

- CL fitting possible as long as satisfactory physical fit can be obtained
- If surgical removal required, prognosis fair although re-growth occurs in 40% cases

**Note: condition associated with 2 to 3x increased risk of incident late and early ARMD**

### Differential diagnosis

Vascularised limbal keratitis (VLK)

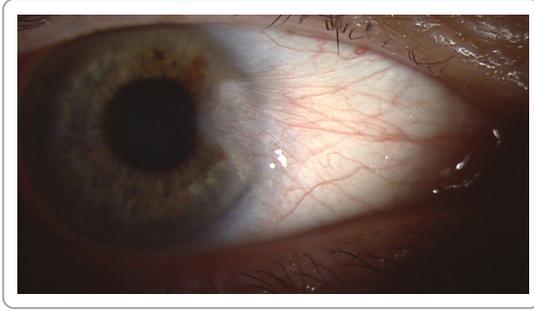


Figure 1: Nasal pterygia extending to pupil margin

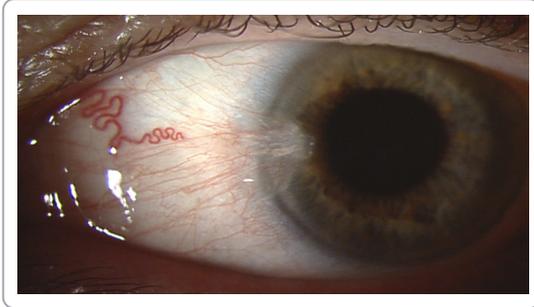


Figure 2: Medium magnification view of nasal pterygia



Figure 3: Nasal pterygia viewed under higher magnification

## Describing clinical appearance

Position: nasal/temporal and extent of encroachment onto cornea

42. Janson BJ, Sikder S. Surgical management of pterygium. *Ocul Surf* 2014; 12: 112-9.
43. Jaros P, DeLuise V. Pinguecula and pterygia. *Surv Ophthalmol* 1988; 33: 41-49
44. Linaburg T et al. Systematic Review: Effects of Pterygium and Pingueculum on the Ocular Surface and Efficacy of Surgical Excision. *Cornea* 2021; 40: 258-267.
45. Martins T et al. Mitomycin C in pterygium treatment. *Int J Ophthalmol* 2016; 9: 465-8.
46. Nouredin G, Yeung S. The use of dry amniotic membrane in pterygium surgery. *Clin Ophthalmol* 2016; 10: 705-12.
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# Bulbar Conjunctiva

## Bulbar Conjunctival Staining (General)

### Slit lamp viewing

Diffuse beam, direct illumination, medium/high magnification (16-30x), fluorescein with cobalt blue filter and yellow barrier filter over the viewing system, along with lissamine green with white light and optional red filter over viewing system.

### Incidence

>95% CL wearers show some level of conjunctival staining

### Aetiology

- Poor blinking
- Dry eye not related to CLs.

### Symptoms

- Often asymptomatic
- Can be combined with dryness and CL intolerance

### Signs

- Staining bulbar conjunctiva with fluorescein, rose bengal or lissamine green, especially nasal (lissamine green greater specificity for symptomatic patients)
- Curved linear staining parallel to limbus (furrow staining)

### Management

- Manage if  $\geq$  grade 2 or  $\geq$  1 grading interval increase or if symptoms
- Often asymptomatic with no management required
- Blinking exercises if poor blinking evident
- General dry eye management options to improve condition of tear film and ocular surface: lubrication (re-wetting drops), lid margin therapy (warm compresses, lid wipes, meibum expression), dietary and environmental advice

### Prognosis

Depends on cause — if minor, minimal impact, but prognosis can be poor if due to significant dry eye

### Differential diagnosis

Sjögren's syndrome, conjunctival oedema, bulbar conjunctival staining (CL related)

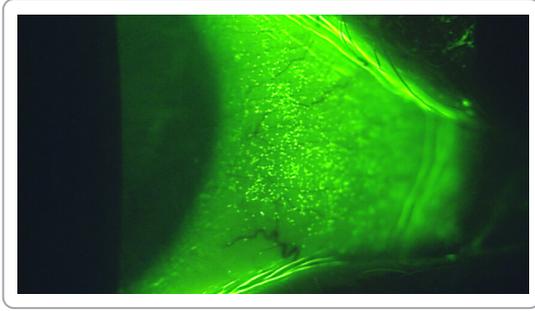


Figure 1: Punctate staining of conjunctiva viewed under blue light and Wratten filter

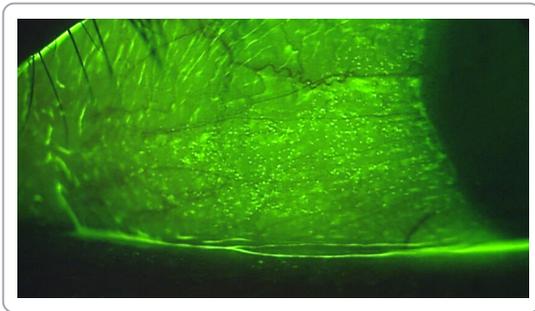


Figure 2: Punctate staining of conjunctiva viewed under blue light and Wratten filter

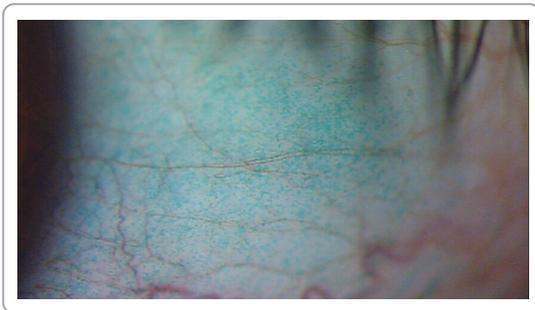


Figure 3: Punctate lissamine green staining of conjunctiva viewed with white light

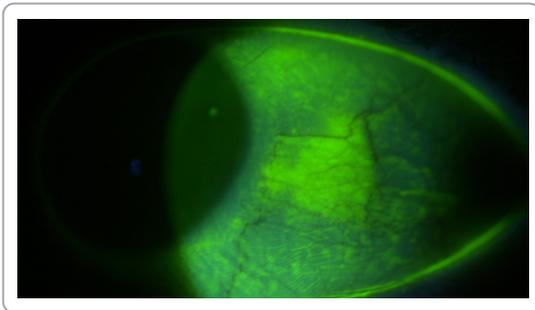


Figure 4: Confluent conjunctival stain

## Describing clinical appearance

Position: Superior, nasal, inferior, temporal

- 0: None
- 1: Minimal diffuse punctate
- 2: Coalescent, punctate
- 3: Confluent
- 4: Deep confluent

50. Bron A et al. Grading of corneal and conjunctival staining in the context of other dry eye tests. *Cornea* 2003; Oct 22(7): 640-50

51. Guillon M, Maissa C. Bulbar conjunctival staining in contact lens wearers and non lens wearers and its association with symptomatology. *Cont Lens Ant Eye* 2005; 28(2): 67-73

52. Keir N et al. The conjunctival response to soft contact lens wear. *Optometry in practice* 2010; 11(3): 123-134

53. Korb DR et al. An evaluation of the efficacy of fluorescein, rose bengal, lissamine green, and a new dye mixture for ocular surface staining. *Eye Contact Lens* 2008; 34(1): 61-4

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56. Schwallie J et al. Day to day variations in ocular surface staining of the bulbar conjunctiva. *Optom Vis Sci* 1998; 75(1): 55-61

57. Young G, Coleman S. Poorly fitting soft lenses affect ocular integrity. *CLAO J* 2001; 27(2): 68-74

# Bulbar Conjunctiva

## Bulbar Conjunctival Staining (CL Related)

### Slit lamp viewing

Diffuse beam, direct illumination, medium/ high magnification (16–30x), fluorescein with cobalt blue filter and yellow barrier filter over viewing system.

### Incidence

Some staining noted in majority of CL wearers and to a greater extent than non-CL wearers

### Aetiology

- Mechanical trauma of conjunctival epithelium due to lens manufacturing method, fit or design, tight fitting, lens edge design or decentration
- Dry eye symptomatology
- Poor blinking
- Solution sensitivity
- Scleral lens – steep lens edge pinching the conjunctiva causing circumferential or sectoral blood vessel impingement

### Symptoms

- Often asymptomatic; does not tend to affect clinical performance and CL acceptance remains high
- Can be combined with dryness and CL awareness

### Signs

- Staining of the bulbar conjunctiva with fluorescein, rose bengal or lissamine green (lissamine green greater specificity for symptomatic patients)

- Area often consistent with position of hydrogel lens edge

### Management

- Manage if  $\geq$  grade 2 or  $\geq$  1 grading interval increase or if symptoms
- Often asymptomatic with no management required and lens wear can continue
- Change lens design, fit or material
- Change care system
- Re-wetting agents to minimize dryness symptoms
- Scleral lenses – modify lens fit or change design to resolve conjunctival pinching and improve lens alignment with the conjunctiva

### Prognosis

- Depends on cause – good when refitting with different lens; poor if due to dry eye
- Does not tend to affect clinical performance unless due to dry eye
- Scleral lenses – important to manage as long-term lens impingement may result in corneal hypertrophy

### Differential diagnosis

Conjunctival oedema, bulbar conjunctival staining (general)

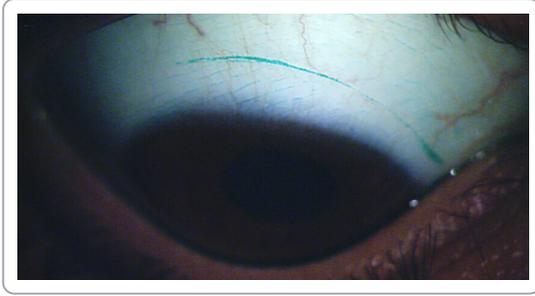


Figure 1: Superior lens edge lissamine green stain viewed under white light

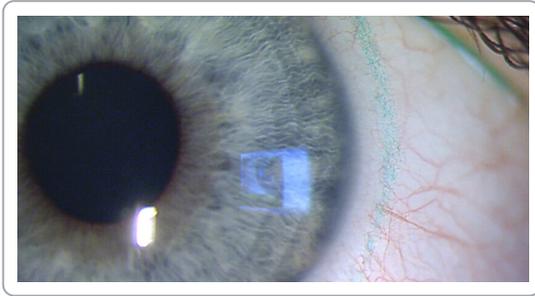


Figure 2: Temporal lens edge lissamine green stain viewed under white light

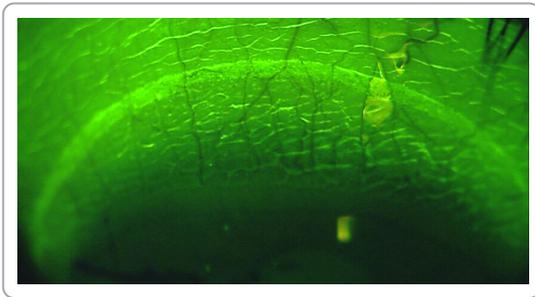


Figure 3: Superior lens edge stain viewed with fluorescein and blue light/yellow Wratten filter

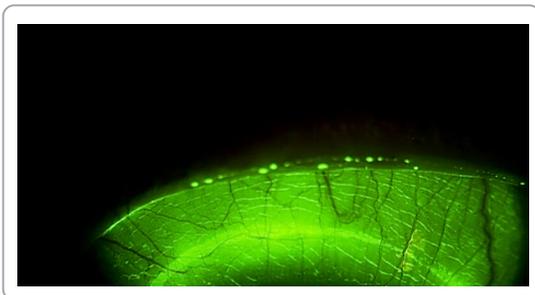


Figure 4: Superior lens edge stain viewed with fluorescein and blue light/yellow Wratten filter

## Describing clinical appearance

Position: Superior, nasal, inferior, temporal

- 0: None
- 1: Minimal diffuse punctate
- 2: Coalescent, punctate
- 3: Confluent
- 4: Deep confluent

50. Bron A et al. Grading of corneal and conjunctival staining in the context of other dry eye tests. *Cornea* 2003; Oct 22(7): 640-50

51. Guillon M, Maissa C. Bulbar conjunctival staining in contact lens wearers and non lens wearers and its association with symptomatology. *Cont Lens Ant Eye* 2005; 28(2): 67-73

52. Keir N et al. The conjunctival response to soft contact lens wear. *Optometry in practice* 2010; 11(3): 123-134

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54. Lakkis C, Brennan N. Bulbar conjunctival fluorescein staining in hydrogel CL wearers. *CLAO J* 1996; 22(93): 189-194

55. Maissa C et al. Contact lens-induced circumlimbal staining in silicone hydrogel contact lenses worn on a daily wear basis. *Eye Contact Lens* 2012; 38: 16-26.

56. Schwallie J et al. Day to day variations in ocular surface staining of the bulbar conjunctiva. *Optom Vis Sci* 1998; 75(1): 55-61

57. Young G, Coleman S. Poorly fitting soft lenses affect ocular integrity. *CLAO J* 2001; 27(2): 68-74

# Bulbar Conjunctiva

## Bulbar Conjunctival Oedema (Chemosis)

### Slit lamp viewing

Direct beam, medium magnification (16x), direct illumination

### Incidence

Unknown

### Aetiology

- Allergic response (solution)
- Hayfever
- Allergic reaction
- Excessive mechanical irritation (such as excessive eye-rubbing)

### Symptoms

- Watery eyes, excessive tearing.

### Signs

- Diffuse oedema of bulbar conjunctiva with or without hyperaemia

### Management

Remove cause

### Prognosis

Good – recovery within 24–48 hours

### Differential diagnosis

Pinguecula, post-surgical scarring

### Describing clinical appearance

Note position and describe extent

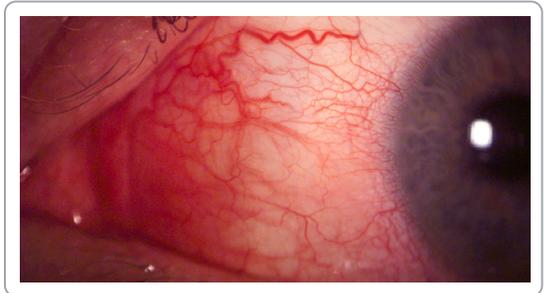


Figure 1: Acute allergic conjunctivitis with chemosis

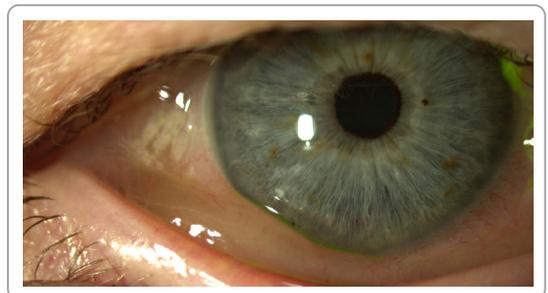


Figure 2: Acute conjunctival chemosis

# Bulbar Conjunctiva

## Bulbar Conjunctival Hyperaemia

### Slit lamp viewing

Diffuse beam, medium magnification (16x), direct illumination

### Incidence

- 15% – 20% CL wearers, with 20 – 35% clinically significant
- Approximately 15% non-CL wearers  $\geq$  grade 2

### Aetiology

- Solution sensitivity
- Dry eye symptomatology or pathological dry eye (KCS)
- Allergic reaction
- Infection – Microbial Keratitis (MK)
- Inflammation – Contact Lens Associated Red Eye (CLARE), Contact Lens Peripheral Ulcer (CLPU), Infiltrative Keratitis (IK) etc
- Mechanical – poor lens fit, trauma
- Metabolic – Corneal hypoxia, hypercapnia
- Poor general health (especially influenza, throat infection or substance abuse)
- Scleral lenses – blood vessel compression, tightly fitting lens, lens adhesion

### Symptoms

- May be asymptomatic – depends on cause
- Associated with CL intolerance, dryness, burning and itching

### Signs

- Bulbar conjunctival redness can be localized or diffuse
- Amount and pattern depend on lens type:
- rigid corneal lens wearers tend to exhibit this along the horizontal meridian (chronic drying)
- hydrogel lens wearers' hyperaemia tends to be diffuse
- scleral lens wearers hyperaemia usually localized to the area of lens compression / impingement – where circumferentiality can indicate hypoxia / lactic acid accumulation in the post lens tear film.

## Management

- Manage if  $\geq$  grade 2 or if  $\geq$  1 grading scale increase or if symptoms occur
- Lens wear may continue if symptoms allow
- Remove the cause
- Switch to DD if Aetiology is related to allergy
- Refit with higher oxygen performance materials (especially if hyperaemia is primarily located around the limbal region)
- Change lens material to reduce dryness
- Ocular lubricants
- Attend to any lid abnormalities (blepharitis; Demodex presence; MGD)
- Scleral lenses:
- Lens compression related - alter lens fit to reduce compression
- Toxicity related – increase lens material Dk/t and decrease central lens clearance where possible.

## Prognosis

- Good – although some wearers always exhibit some degree of bulbar hyperaemia

## Differential Diagnosis

- Subconjunctival hemorrhage, conjunctivitis, keratitis, uveitis, acute glaucoma



Figure 1: Example of low level conjunctival hyperaemia (actual grade dependent on grading scale used)



Figure 2: Magnified view of conjunctival hyperaemia

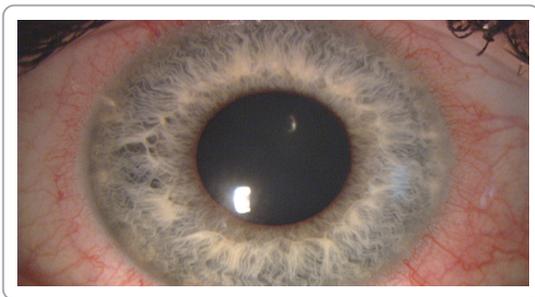


Figure 3: Low magnification diffuse light view of conjunctival hyperaemia

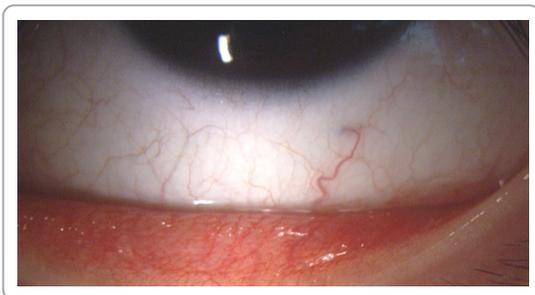


Figure 4: Example of inferior conjunctival hyperaemia with lower lid retracted

## Describing clinical appearance

Position: Superior, nasal, inferior, temporal

- 0: None
- 1: Slight injection of conjunctival vessels
- 2: Mild injection
- 3: Moderate injection
- 4: Severe injection

58. Efron N. Contact lens induced conjunctival hyperaemia. *Optician* 1997; 213(5591): 22-27

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63. Pult H et al. Limbal and bulbar hyperaemia in normal eyes. *Ophthalmic Physiol Opt* 2008; 28: 13-20.

64. Schulze M et al. The perceived bulbar redness of clinical grading scales. *Optom Vis Sci* 2009; 86: E1250-8.

# Bulbar Conjunctiva

## Lid Parallel Conjunctival Folds (LIPCOF)

### Slit lamp viewing

Diffuse beam, medium magnification (18 - 27x), direct white illumination with no CL and in primary gaze. No use of vital dyes (fluorescein, lissamine green or rose bengal). Evaluate in area perpendicular to temporal and nasal limbus (temporal LIPCOF and nasal LIPCOF) on bulbar conjunctiva above lower lid.

### Incidence

- In non-CL wearers, 57% LIPCOF Sum of 1 or less (nasal LIPCOF grade plus temporal LIPCOF grade)
- Higher in symptomatic compare to non-symptomatic CL wearers
- In CL wear correlates with CL discomfort, lid wiper epitheliopathy, older age and lower mucin production

### Aetiology

Possible causes are conjunctival looseness, decrease of elastic fibers, aging or mechanical forces between lower lid and conjunctiva

### Symptoms

Dryness. Combine with non-invasive break-up time (NIBUT) and Ocular Surface Disease Index (OSDI) questionnaire as predictive assessment for dry eye symptoms in CL wearers

### Signs

Folds in temporal and nasal lower quadrants of bulbar conjunctiva, parallel to lower lid

### Management

- Manage  $\geq$  grade 2 or if symptoms
- Refit with more lubricious lens surface
- Change lens type (SiHy to hydrogel) or wearing schedule (increase replacement frequency, reduce wearing time)
- Maintain good lens cleaning including rub and rinse step
- Manage any tear quality issues
- Rewetting drops or liposomal sprays

### Prognosis

Generally good resolution of symptoms with appropriate management

### Differential Diagnosis

Parallel permanent conjunctival folds, disrupted microfolds or conjunctival flaps. LIPCOF disappears when lower lid is lifted



Figure 1: Example of LIPCOF



Figure 2: Higher magnification example of LIPCOF

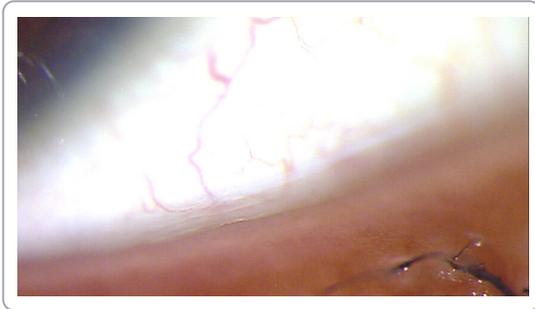


Figure 3: Higher magnification example of LIPCOF

## Describing clinical appearance

A combined LIPCOF score (LIPCOF Sum) can be calculated by adding together nasal LIPCOF grade and temporal LIPCOF grade

- 0: No conjunctival folds
- 1: One permanent and clear parallel fold
- 2: Two permanent and clear parallel folds (normally <0.2mm)
- 3: More than two permanent and clear parallel folds (normally >0.2mm)

65. Bandlitz S et al. Lid-parallel conjunctival fold (LIPCOF) morphology imaged by optical coherence tomography and its relationship to LIPCOF grade. *Cont Lens Ant Eye* 2019; 42(3): 299-303

66. Berry M et al. Mucins and ocular signs in symptomatic and asymptomatic contact lens wear. *Optom Vis Sci* 2008; 85(10): E930-938

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# Superior Tarsal Conjunctiva

## Contact Lens Associated Papillary Conjunctivitis (CLAPC)

Also known as **Contact Lens Induced Papillary Conjunctivitis (CLIPC)**, lid roughness, papillary hypertrophy

### Slit lamp viewing

Diffuse beam, medium magnification (16x), direct illumination, fluorescein to assess roughness

### Incidence

- Variable time of onset and severity; varies over years with different lenses and care regimens
- 2% rigid corneal lenses, 2 – 15% hydrogel DW, 2 – 19% hydrogel overnight, 2 – 7% SiHy
- Significantly reduced since the introduction of frequent replacement CLs
- Increased incidence with overnight wear, non-planned replacement and higher modulus materials (mechanical related)

### Aetiology

- Conjunctival inflammatory condition associated with CLs or trauma
- Immunological response – delayed hypersensitivity (denatured deposits, solution sensitivity)
- Mechanical response (lens design or material modulus, prostheses and sutures)
- Associated with atopy and MGD

### Symptoms

- Lens awareness, CL intolerance, foreign body sensation and itching
- Mucus formation, visual disturbance (lens dislocation, deposition and mucus)

### Signs

- Papillae (>0.3mm) on upper tarsal conjunctiva with central vascular tuft, mucus discharge, tarsal conjunctival hyperaemia and oedema
- Displaced CL and excessive movement with poor surface wetting and deposits
- Location of papillae tends to be confined to lesser area tarsal conjunctiva with SiHys (localized)

## Management

- Manage if  $\geq$  grade 2 or  $\geq$  1 grading interval increase or if symptoms
- Lens wear can continue if symptoms allow
- Improve lens hygiene (ensure rub and rinse step with MPS, change care system to preservative free)
- Increase lens replacement frequency (ideally daily disposable), replace existing lenses
- Alter lens design or material (reduce edge clearance and thickness of rigid corneal lenses, fit lower modulus soft lens material)
- Introduce lid hygiene if signs of lid margin disease
- If severe, temporary cessation of lens wear with topical mast cell stabilizers or steroids in cases where lens wear is essential (eg keratoconus)

## Prognosis

- Good, especially with frequent replacement lenses, although papillae can remain for months or years
- Better with early detection – resolves within 2 – 3 weeks if mechanical, longer if inflammatory cause
- Up to 60% recurrence with SiHy EW

## Differential Diagnosis

- Follicles – vessels on the outside of the enlarged area of tissue.
- Inferior tarsal conjunctivitis – not CL related
- Seasonal allergic conjunctivitis
- Vernal conjunctivitis – no vessels (young males, both inferior and upper tarsus)
- Giant papillary conjunctivitis caused by other reasons, for example mechanical irritation from protruding corneal sutures

## Describing clinical appearance

- 0: Few vessels on surface; smooth, even conjunctiva
- 1: Small papillae, few vessels on surface, uneven slit lamp surface reflection
- 2: Loss of transparency, small papillae, uneven slit lamp surface reflection
- 3: Papillae with vessels visible
- 4: Papillae with staining and vessels



Figure 1: Low grade hyperaemia and papillae



Figure 2: Light reflection showing small papillae in centre of tarsal conjunctiva



Figure 3: Visible moderate level of papillae

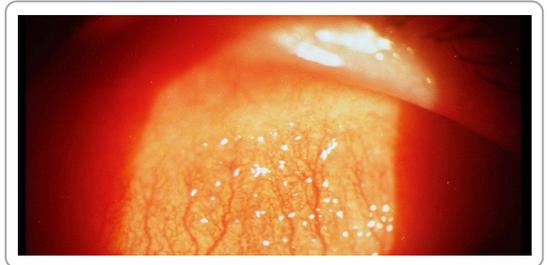


Figure 4: Moderate papillae and increased hyperaemia

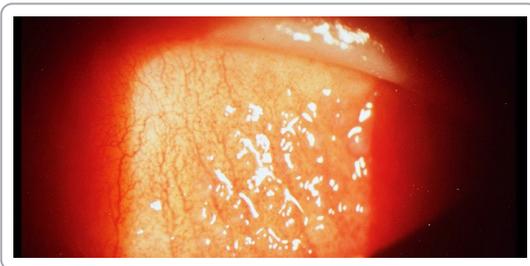


Figure 5: Marked giant papillae and hyperaemia

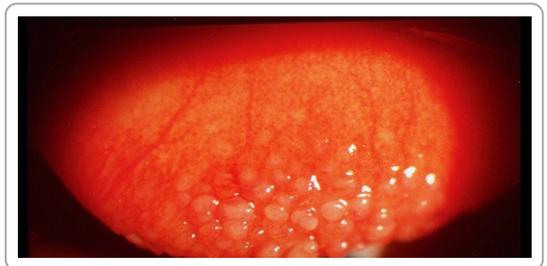


Figure 6: Marked giant papillae and hyperaemia

72. Dumbleton K. Adverse events with silicone hydrogel continuous wear. *Cont Lens Ant Eye* 2002; Sep 25(3): 137-46

73. Dumbleton K. Noninflammatory silicone hydrogel contact lens complications. *Eye & contact lens* 2003; 29(1 Suppl): S186-S194

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# Limbal Area

## Limbal Hyperaemia

### Slit lamp viewing

Diffuse beam, direct illumination, medium magnification (16x)

### Incidence

True incidence unknown although common in most lens types. Occurs to some degree with all hydrogel lenses, although may be mild with thin, mid-water hydrogels.

### Aetiology

- Short-term clinical sign of corneal hypoxia – related to oxygen performance of lens
- Inflammation (tight lens syndrome)
- Mechanical irritation (trauma, poor lens fit)
- Atopic/allergic reaction
- Solution sensitivity
- Infection

### Symptoms

- Often none; depends on cause
- Possible pain

### Signs

- Engorged limbal blood vessels with possible subsequent neovascularisation
- May be localized or full coverage, depending on lens type or Aetiology, with conjunctival vessel involvement

### Management

- Manage if  $\geq$  grade 2 or if  $\geq 1$  grading scale interval increase or if symptoms occur
- Cease lens wear until resolution. Refit with higher oxygen performance lens materials (SiHy, rigid corneal lenses RCL))
- Reduce wearing time or change to DD
- Optimize lens fit
- Remove allergen
- Change care system

### Prognosis

- Good – depends on cause
- Reversible
- Noticeable “white-eye” difference between SiHy and traditional hydrogels

### Differential Diagnosis

Neovascularisation, super limbic keratoconjunctivitis (SLK), keratitis, CLARE, uveitis, acute glaucoma, intra-ocular infection



Figure 1: Example of limbal hyperaemia

## Describing clinical appearance

Position: Superior, nasal, inferior, temporal

### Depth

- 0: None
- 1: Slight injection of limbal vessels
- 2: Mild injection
- 3: Moderate injection
- 4: Severe injection

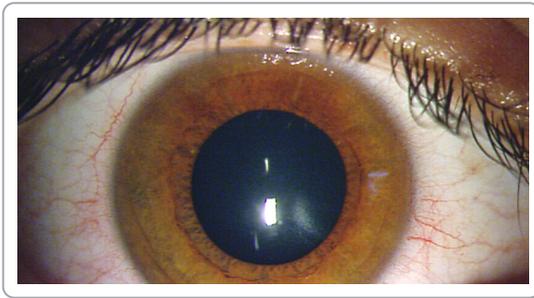


Figure 2: Low magnification, diffuse illumination view of limbal hyperaemia



Figure 3: Example of limbal hyperaemia

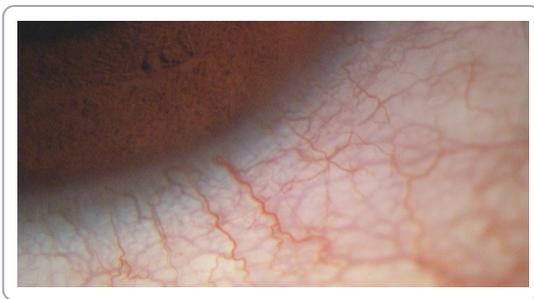


Figure 4: Higher magnification view of limbal hyperaemia



Figure 5: Differential diagnosis: limbal and conjunctival hyperaemia secondary to corneal foreign body

80. du Toit R, et al. Recovery from hyperaemia after overnight wear of low and high transmissibility hydrogel lenses. *Curr Eye Res* 2001; 22: 68-73.
81. Papas E et al. High oxygen-transmissibility soft contact lenses do not induce limbal hyperaemia. *Curr Eye Res* 1997; 16 (9): 942-948
- Papas E. On the relationship between soft contact lens oxygen transmissibility and induced limbal hyperaemia. *Exp Eye Res* 1998; 67: 125-31.
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# Limbal Area

## Limbal Staining

### Slit lamp viewing

Direct illumination, medium/high magnification (16–30x) with fluorescein with blue cobalt filter and yellow barrier filter

### Incidence

Occurs commonly in all lens types

### Aetiology

- Hypoxia
- Tight lens
- Mechanical irritation e.g. poor lens fit
- Atopic / allergic reaction
- Toxic solution reactions

### Symptoms

- Often asymptomatic depending on severity
- As severity increases, some CL intolerance may occur

### Signs

Staining around limbal area over conjunctiva and cornea

### Management

- Manage if  $\geq$  grade 2 or if  $\geq$  1 grading scale increase or if symptoms occur
- Lens wear can be continued if symptoms allow
- Increase oxygenation, reduce wearing time, lens refit
- Alter lens care regimen

### Prognosis

Good

### Differential Diagnosis

Limbal oedema, conjunctival staining

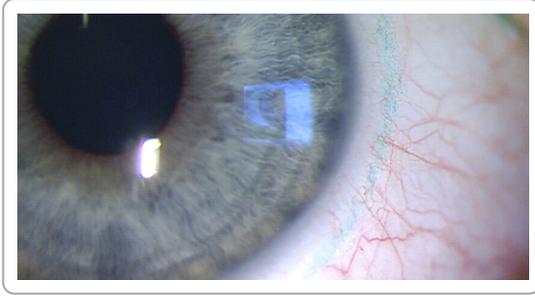


Figure 1: Lissamine green limbal stain from soft lens edge

## Describing clinical appearance

Position: Superior, nasal, inferior, temporal

- 0: None
- 1: Minimal diffuse punctate
- 2: Coalescent, punctate
- 3: Confluent
- 4: Deep confluent

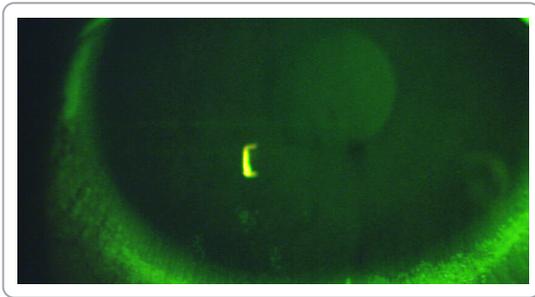


Figure 2: Wide band of fluorescein limbal stain resulting from soft lens edge

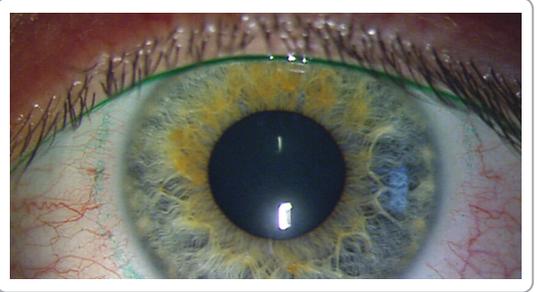


Figure 3: Low magnification view of circum-limbal lissamine green staining

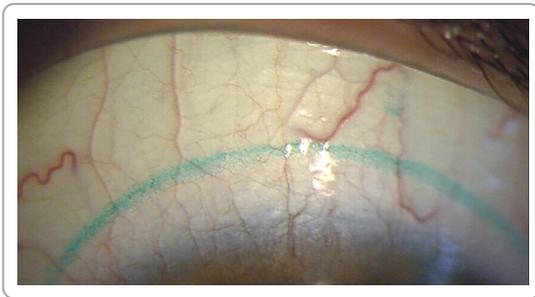


Figure 4: View of superior limbal lissamine green stain

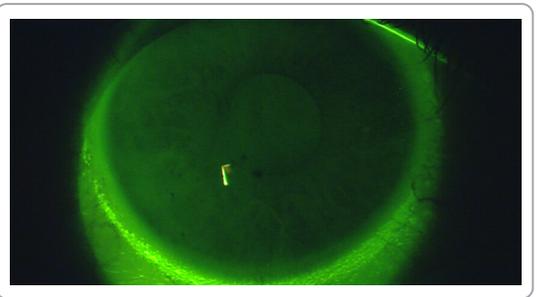


Figure 5: Fluorescein limbal stain viewed with blue light and Wratten filter

# Limbal Area

## Limbal Oedema

### Slit lamp viewing

Diffuse beam, medium magnification (16x), direct viewing with and without fluorescein

### Incidence

More common in soft CL wearers

### Aetiology

- Hypoxia
- Tight lens
- Mechanical irritation e.g. poor lens fit
- Atopic / allergic reaction
- Toxic solution reactions

### Symptoms

Generally asymptomatic depending on Aetiology

### Signs

Can see fluorescein staining in band close to limbus where dye pools in heaped-up areas of peri-limbal cornea — dye disappears when eye is flushed with saline

### Management

- Increase oxygen supply
- Change care regimen
- Modify fit — looser lens design

### Prognosis

Good

### Differential Diagnosis

- Limbal, corneal and conjunctival staining
- Limbal vernal conjunctivitis

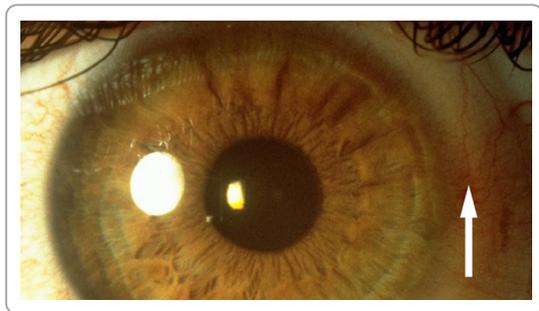


Figure 1: Limbal oedema viewed under diffuse white light

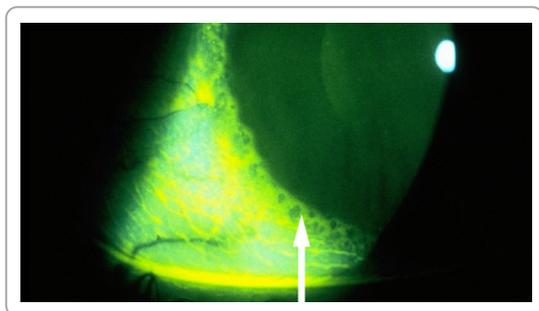


Figure 2: Limbal oedema viewed with fluorescein and blue light

## Describing clinical appearance

Record position and extent

# Limbal Area

## Limbal Bearing

### Slit lamp viewing

Diffuse beam, medium magnification (16x), direct viewing with and without fluorescein

### Incidence

Scleral lens wear

### Aetiology

Localized contact lens pressure interfering with limbal stem cells and their essential function in cornea epithelium regeneration

### Symptoms

None, some may report discomfort

### Signs

Limbal staining – if severe can lead to limbal stem cell deficiency and subsequent failure of the epithelium

### Management

Alter lens design to cause the landing zone of the scleral lens to land further from the limbus and thereby alleviate limbal bearing

### Prognosis

Good once lens fit is optimized

### Differential Diagnosis

- Limbal, corneal and conjunctival staining
- Limbal vernal conjunctivitis



Figure 1: Example of limbal bearing with a scleral lens in a very hyperemic eye

### Describing clinical appearance

Location of any limbal staining, limbal oedema, epithelial bullae and keratitis

# Limbal Area

## Superior Limbic Keratoconjunctivitis (SLK)

### Slit lamp viewing

Diffuse illumination, low/high magnification (10 – 16x), use of fluorescein with blue cobalt filter and yellow barrier filter

### Incidence

6.5% in symptomatic CL wearers (1992), although significantly lower since thimerosal and other such low molecular weight preservatives no longer used in modern care systems

### Aetiology

- Delayed hypersensitivity reaction to care regimen (thimerosal or other preservatives; Thimerosal Keratopathy), or deposits
- Hypoxia – contributing factor as typically under upper lid
- Mechanical irritation – excessive lens movement

### Symptoms

Lens awareness, burning, itching, redness, watering

### Signs

- Inflammatory response of superior limbus under top lid – bilateral epithelial keratinisation
- Limbal oedema and hyperaemia
- Superior bulbar conjunctival chemosis
- Epithelial haze
- Corneal and conjunctival staining under lid
- V-shaped fibrovascular pannus
- Infiltrates

### Management

- Reduce wearing time or temporary cessation of lens wear if severe
- Non-steroidal anti-inflammatory agents (NSAIDs) and mast cell stabilizers
- Ocular lubricants
- Replace lenses and change care system to non-preserved or change to DD

### Prognosis

Excellent – although can take many months

### Differential Diagnosis

Neovascularisation, conjunctivitis, infiltrative keratitis, Theodore's SLK (not CL related), SEALs

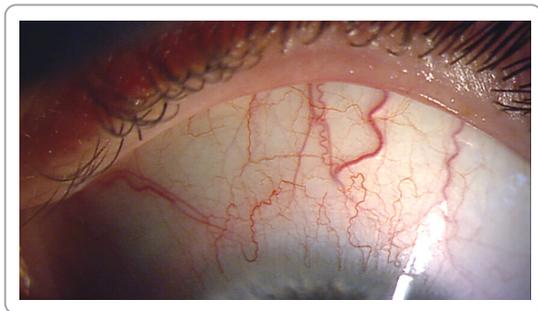


Figure 1: Example of superior limbic keratoconjunctivitis

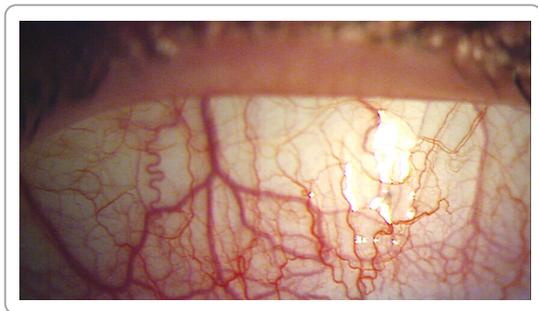


Figure 2: Higher magnification view of superior limbic keratoconjunctivitis

### Describing clinical appearance

Record position, extent and level of associated hyperaemia

- 86. Campbell R, Caroline P. Contact lens induced superior limbic keratoconjunctivitis. *Spectrum* 1996; 2: 56
- 87. Efron N. Contact lens induced superior limbic keratoconjunctivitis. *Optician* 1997; 213 (5599): 20-26
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# Limbal Area

## Neovascularisation

### Slit lamp viewing

Direct and indirect retro illumination, high magnification (30 – 80x)

### Incidence

- Approximately 10% CL wearers; related to oxygen performance of lens
- <1% (rigid corneal lenses (RCL)), 5 – 10% (hydrogel DW), 10 – 20% (hydrogel overnight wear), 0% (SiHy DW or overnight wear)

### Aetiology

- Hypoxia produces stromal oedema and softening with subsequent release of vaso-stimulatory agents with in-growth of vessels
- Corneal epithelial damage, mechanical, solution sensitivity, infection or pathology

### Symptoms

None (vision loss if extreme)

### Signs

- New blood vessels in cornea originating from limbal vessels
- Higher incidence in superior cornea due to presence of upper lid and reduction in oxygenation

### Management

- Manage if  $\geq 1$  grading scale interval increase; cease lens wear if Grade 3 or 4 or if vascularisation continues.
- Increase corneal oxygenation – high oxygen performance lens (rigid corneal or SiHy), reduce wearing time or cease overnight wear, and monitor closely
- Decrease mechanical stimulation
- Change care system
- Manage any associated pathology

### Prognosis

Good – irreversible but can stop progression; ghost vessels remain

### Differential Diagnosis

- Former corneal pathology with vessels growing towards site of damage
- Limbal hyperaemia
- Normal physiological blood vessel overlay
- Ghost vessels (relatively thick, start at limbus),
- Corneal nerves (any orientation)

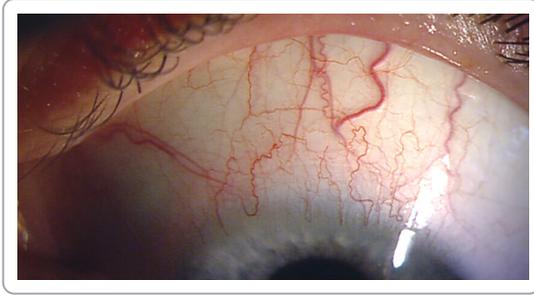


Figure 1: Example of neovascularisation viewed with direct illumination

## Describing clinical appearance

Position: Superior, nasal, inferior, temporal;  
descriptive and include depth

- 0: None
- 1: < 0.5mm
- 2: 0.5 – 1.0mm
- 3: 1.0 – 2.0mm
- 4: > 2.0mm

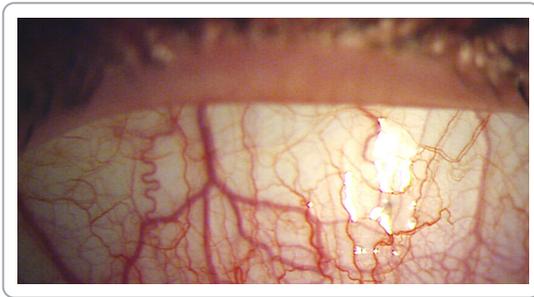


Figure 2: Neovascular changes in the superior limbal area

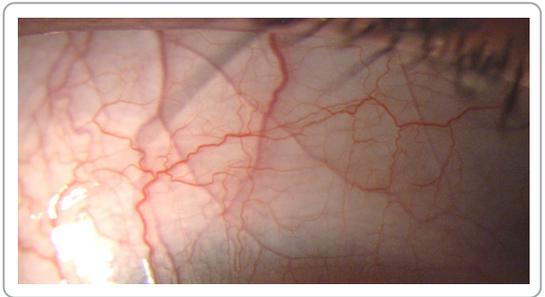


Figure 3: Superior neovascularisation

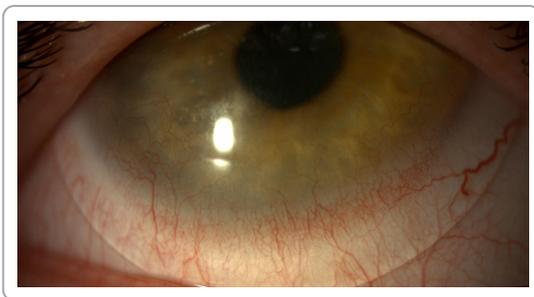


Figure 4: Extensive corneal vascularization visible under a scleral lens

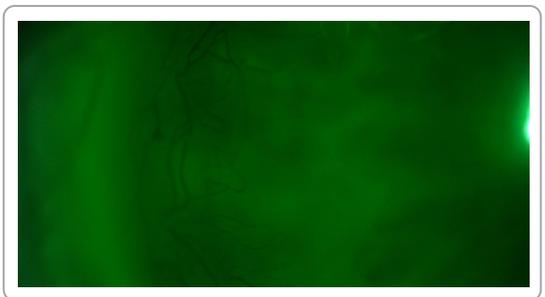


Figure 5: Extensive vascularisation visible with red-free filter (fluorescein also present in image)

90. Caffery B, Josephson J. Corneal vascularization. *Optom Clin* 1995; 4: 19-29.
91. Covey M et al. Hypoxic effects on the anterior eye of high-Dk soft contact lens wearers are negligible. *Optom Vis Sci* 2001; 78: 95-99.
92. Dumbleton K et al. Vascular response to extended wear of hydrogel lenses with high and low oxygen permeability. *Optom Vis Sci* 2001; 78: 147-51.
93. Dumbleton K et al. Objective and subjective responses in patients refitted to daily-wear silicone hydrogel contact lenses. *Optom Vis Sci* 2006; 83: 758-68.
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97. Sweeney DF. Clinical signs of hypoxia with high-Dk soft lens extended wear: is the cornea convinced? *Eye Contact Lens* 2003; 29: S22-5; discussion S26-9, S192-4.
98. Sweeney DF. Have silicone hydrogel lenses eliminated hypoxia? *Eye Contact Lens* 2013; 39: 53-60.
99. Wong AL et al. Bilateral corneal neovascularisation and opacification associated with unmonitored contact lens wear. *Am J Ophthalmol* 2003; 136: 957-8.

# Cornea

## Corneal Staining

### Slit lamp viewing

Direct illumination, parallelepiped or diffuse, medium/high magnification (16 – 30x) with fluorescein with blue cobalt filter and yellow barrier filter

### Incidence

- Common in all CL wearers (up to 60%) but often clinically insignificant
- More common in Ortho-K as the lens design is intended to produce compressive forces on the cornea
- Some degree also seen in non-CL wearers (35%)

### Aetiology

- Mechanical – trauma, foreign body, damaged lens, lens edge, material stiffness, thick lens design, Ortho-K lens wear, scleral lens wear
- Exposure – disruption of tear film and subsequent desiccation
- Metabolic – hypoxia, hypercapnia (tissue acidosis and desquamation of epithelial cells)
- Solution induced corneal staining (SICS) seen 2-4 hours after insertion with some SIHy and multi-purpose solution (MPS) combinations
- Toxic – care regimen hypersensitivity (1-10% hydrogel lens wearers)
- Allergic – delayed or immediate hypersensitivity reaction
- Infectious systemic disease, poor general health (e.g. influenza, throat infection)

### Symptoms

- Can be asymptomatic – depends on Aetiology and severity; for most cases of grade 3 or 4 there would be some discomfort or even pain
- Symptoms may include CL intolerance, reduced wearing time, dryness, itching
- Reduced vision if significant and located over visual axis
- Lacrimation

### Signs

- Superficial punctate epithelial erosions (extent, depth and location depends on Aetiology)
- Bulbar conjunctival hyperaemia
- Tarsal conjunctival changes
- Lacrimation
- Ortho-K:
  - apparent at lens removal and throughout the day
  - central staining more likely when correcting myopia; paracentral staining more likely when correcting hyperopia
  - exacerbated in new Ortho-K wearers

## Management

- Manage if  $\geq$  Grade 2 or if  $\geq 1$  grading scale increase
- Remove lenses until resolved
- Consider medical intervention/treatment for Grades 3 and 4 if related to trauma/mechanical causes or infection/inflammation
- Ocular lubricants to reduce symptoms
- Isolate cause and manage – change care system, refit/replace lens, improve oxygen performance, blinking, rewetting drops
- For SICS: ensure a rub & rinse step, alter combination of SiHy and MPS, switch to non-preserved solution or change to DD lens
- Ortho-K
  - Advise instilling aqueous ocular lubricant and gently nudging the lens with the eyelids to mobilize lens before removing
  - If new presentation in an existing Ortho-K wearer consider possible lens spoilage/damage/warpage – protein clean lens and if not resolved consider lens replacement
  - Grade 1 or 2: Acceptable if confident that staining is lens induced (mechanical) – reinforce advice on lens removal
  - Grade 3: Assess in the afternoon to verify staining resolves during the day.
  - Grade 4: Discontinue wear until resolved and consider prophylactic antibiotic if there is evidence of epithelial break
  - If mechanical-related staining is persistent consider altering lens parameters to reduce mechanical pressure
- Scleral lenses:
  - Assess whether the lens is bearing on the cornea
    - Central lens bearing or lens fenestrations causing abrasion – increase lens sagittal height
    - Para-central lens bearing – increase corneal, peripheral and limbal clearance alongside steepening the transition zone
- Hypoxia – increase lens material oxygen transmissibility
  - Toxic reaction to solutions – consider change to non-preserved solutions
  - Post lens fluid reservoir containing debris – verify compliance

## Prognosis

Good (unless Bowman's membrane penetrated and subsequent residual scarring)

## Differential Diagnosis

Dry eye or ocular surface disease, infectious keratitis, mechanical insult (eg: from fingers when applying or removing lens, or from excessive eye rubbing)

# Cornea

## Inferior Epithelial Arcuate Lesions (Smile Stain)

### Slit lamp viewing

Direct illumination, parallelepiped or diffuse, medium/high magnification (16 – 30x) with fluorescein with blue cobalt filter and yellow barrier filter

### Incidence

- Most common in thinner, higher water content hydrogels (3% disposable lens wearers)
- Incidence lower with mid water hydrogels and SiHy materials
- May also occur in non-CL wearers

### Aetiology

- Epithelial desiccation staining following lens dehydration and depletion post-lens tear film
- Poor tear film (rapid evaporation)
- Incomplete blinking
- Low humidity environments

### Symptoms

- Often none
- May have dryness, CL intolerance and reduced wearing time

### Signs

Superficial punctate epithelial erosions inferior in cornea, from 4 to 8 o'clock, 2 – 4 mm in from inferior limbus

### Management

- Manage if  $\geq$  grade 2 or if  $\geq$  1 grading scale increase
- Remove lenses for 24 hours with grade 2, 4 – 5 days with more intense staining
- May require medical treatment for grade 4
- Ocular lubricants
- Blinking exercises
- Change lens material or design (greater thickness, lower water content hydrogel, SiHy)

### Prognosis

Good – removal of lens resolves staining, although hard for complete resolution when due to incomplete blinking

### Differential Diagnosis

- Rigid corneal lens edge stain, soft lens back surface curve transition stain, trauma from insertion/removal
- May be associated with MGD

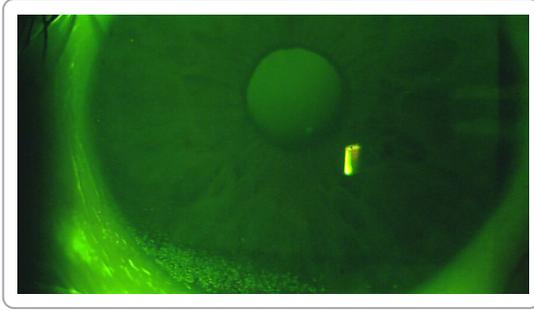


Figure 1: Example of LIPCOF



Figure 2: Higher magnification example of LIPCOF

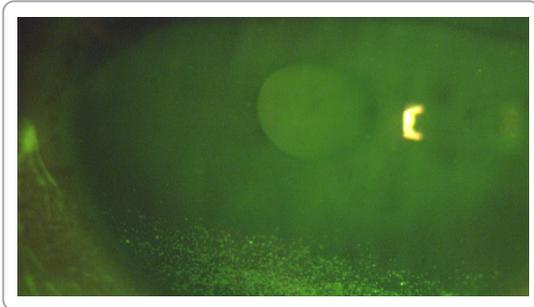


Figure 3: Higher magnification example of LIPCOF

## Describing clinical appearance

### Extent

- 0: None
- 1: 1 – 20 punctate diffuse spots
- 2: 21 – 40 punctate diffuse spots
- 3: > 40 diffuse spots and/or coalescing patches 4: Dense confluent patches

### Depth

- A: No stromal diffusion
- B: Stromal diffusion delayed (30 – 60 seconds)
- C: Stromal diffusion immediate but moderate
- D: Stromal diffusion immediate and widespread

110. Jones L, Jones D. Smile staining and dehydration. *Optician* 1995; 210(5513): 24-25

111. Little S, Bruce A. Role of the post-lens tear film in the mechanism of inferior arcuate staining with ultra-thin hydrogel lenses. *CLOA J* 1995; 21(3): 175-181

112. McNally J et al. Corneal desiccation staining with thin high water contact lenses. *Clin Exp Optom* 1987; 70: 106-111

113. Watanabe K, Hamano H. The typical pattern of superficial punctate keratopathy in lens wearers of extended wear disposable lenses. *CLAO J* 1997; 23: 134-137

114. Zadnik K, Mutti D. Inferior arcuate corneal staining in soft contact lens wearers. *Int Contact Lens Clin* 1985; 12: 110-114

# Cornea

## Foreign Body Staining (Mechanical)

### Slit lamp viewing

Direct illumination, parallelepiped or diffuse, medium/high magnification (16 – 30x) with fluorescein with blue cobalt filter and yellow barrier filter

### Incidence

- More common in rigid corneal lenses (RCL)
- Can occur in non-CL wearers

### Aetiology

Mechanical – foreign body under lens, damaged lens, poor blending on rigid corneal lens, make-up brush, clumsy application or removal, excessive eye rubbing

### Symptoms

Moderate to severe pain

### Signs

Often seen as vertical linear, foreign body track – can indicate the path the FB has taken under lens

### Management

- Manage if  $\geq$  grade 2 or if  $\geq$  1 grading scale increase
- Remove lenses for 24 hours with grade 2, 4 – 5 days with more intense staining
- Medical treatment for grade 4
- Replace damaged lenses
- Re-teach application and removal
- If staining reoccurs, remove causative agent

### Prognosis

Good unless Bowman's membrane is penetrated, which likely results in residual scarring

### Differential Diagnosis

Infectious keratitis

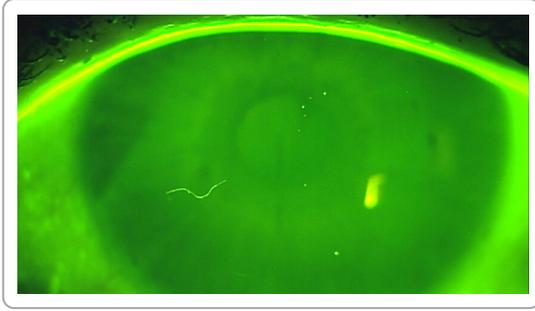


Figure 1: Example of staining from hair fibre trapped under lens

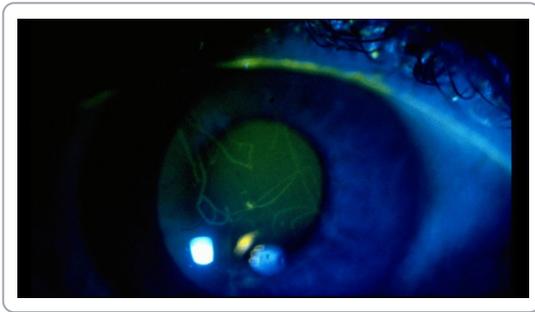


Figure 2: Foreign body track staining

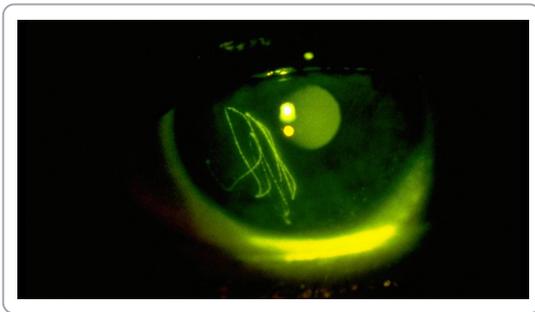


Figure 3: Foreign body track staining

## Describing clinical appearance

### Extent

Describe position, shape (e.g. branch like, parallel tracks, single track etc), and size

### Depth

- A: No stromal diffusion
- B: Stromal diffusion delayed (30 – 60 seconds)
- C: Stromal diffusion immediate but moderate
- D: Stromal diffusion immediate and widespread

# Cornea

## 3 and 9 O'clock Staining

### Slit lamp viewing

Direct illumination, medium/high magnification (16 – 30x) with fluorescein with blue cobalt filter and yellow barrier filter

### Incidence

Up to 80% DW rigid corneal lens (RCL) wearers, with approximately 15% clinically significant levels

### Aetiology

- Exposure keratitis – non-wetting of peripheral cornea as rigid corneal lens (RCL) bridges lid away from ocular surface due to lens design (poor or thick edge, edge clearance inadequate, total diameter too large or small)
- Poor lens wettability
- Incomplete blinking

### Symptoms

- May be associated with lens intolerance, reduced wearing time, dryness
- Conjunctival redness adjacent to affected area of cornea

### Signs

- Superficial punctate epithelial erosions in triangular area at 3 and 9 o'clock positions peripheral to cornea
- Conjunctival hyperaemia along the horizontal meridian
- If extreme, can lead to vascularised limbal keratitis (VLK) or dellen

### Management

- Manage if  $\geq$  grade 2 or if  $\geq$  1 grading scale increase
- Remove lenses for 24 hours with grade 2, 4 – 5 days with more intense staining
- Change lens design – optimize edge design and clearance, increase total diameter, may need toric back surface design to improve fit
- May need to refit with soft lens
- Ocular lubricants
- Blinking exercises

### Prognosis

- Good
- Rapid resolution following lens removal
- If severe, total resolution may be impossible

### Differential Diagnosis

Infectious keratitis, vascularised limbal keratitis



Figure 1: 3 & 9 staining viewed with blue light and yellow filter; rigid contact lens in place

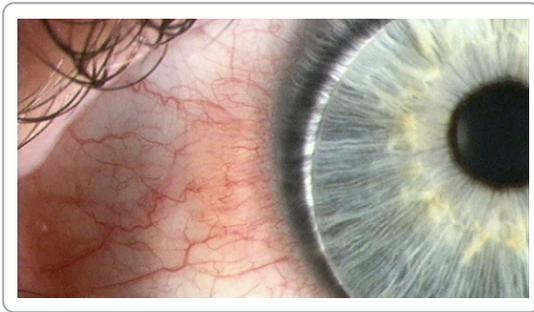


Figure 2: Associated bulbar conjunctival hyperaemia along the horizontal meridian

## Describing clinical appearance

Position: describe peripheral location

### Extent

- 0: None
- 1: 1 – 20 punctate diffuse spots
- 2: 21 – 40 punctate diffuse spots
- 3: > 40 diffuse spots and/or coalescing patches
- 4: Dense confluent patches

### Depth

- A: No stromal diffusion
- B: Stromal diffusion delayed (30 – 60 seconds)
- C: Stromal diffusion immediate but moderate
- D: Stromal diffusion immediate and widespread

116. Andrasko G. Peripheral corneal staining: incidence and time course. *Contact Lens Spectrum* 1990; 5 (7): 59-62
117. Bursinger U et al. The Aetiology and management of 3 and 9 o'clock staining. *Int Contact Lens Clin* 1989; 16 (5): 136-139
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119. Schnider C et al. Effect of patient and lens performance characteristics on peripheral corneal desiccation. *J Am Optom Assoc* 1996; 67 (3): 144-150
120. Schnider C et al. Effect of lens design on peripheral corneal desiccation. *J Am Optom Assoc* 1997; 68 (3): 163-170
121. van der Worp E et al. Corneal desiccation in rigid contact lens wear: 3- and 9-o'clock staining. *Optom Vis Sci* 2003; 80: 280-90.
122. van der Worp E et al. Eyeblink frequency and type in relation to 3- and 9-o'clock staining and gas permeable contact lens variables. *Optom Vis Sci* 2008; 85: E857-66.
123. van der Worp E et al. Evaluation of signs and symptoms in 3- and 9-o'clock staining. *Optom Vis Sci* 2009; 86: 260-5

# Cornea

## Solution Induced Corneal Staining (SICs)

### Slit lamp viewing

Direct illumination parallelized or diffuse, low/medium/high magnification (8 – 30x) with fluorescein with blue cobalt filter and yellow barrier filter.

### Incidence

- Staining maximal with certain combinations of SiHy or FDA group II soft lens materials with multi-purpose solutions (MPS) containing PHMB preservative or Tetricon 1107 surfactant.
- Incidence reported between 23% and 100%, depending on wear time.

### Aetiology

Two hypotheses:

1. SICS is a sign of corneal damage: a component in the lens care preservative leaches out of the CL and causes temporary damage to the corneal epithelial cells which leads to them to stain with fluorescein.
2. SICS is a harmless phenomenon: preservative/surfactant leaches out of the CL and binds to the epithelial cell membrane creating a new complex molecule that attracts fluorescein to bind harmlessly to it, giving the appearance of corneal staining/damage.

### Symptoms

Most commonly asymptomatic, although some reports have described stinging upon lens insertion or removal

### Signs

- Defined as punctate staining of at least 1% of the surface area of at least four of the five corneal zones: central (C), temporal (T), superior (S), nasal (N) and inferior (I).
- Superficial punctate epithelial staining in four out of the five corneal zones
- Typically scattered across the entire cornea but often reported to be less dense centrally, thus often appearing as a 'doughnut' staining pattern.
- Staining is maximal after 1-4hrs lens wear, with punctate stain typically covering 20-70% of the entire corneal area.

## Management

- Change either the lens material or the lens care product
- Switch to a non-preserved care product
- Ensure a rub/rinse step with MPS prior to lens insertion.

## Prognosis

- Good – rapid and complete resolution. Staining often resolved after about 2 hours, with either continued lens wear or following lens removal.
- Consequences of long term SICS are unknown; has been associated with increased corneal infiltrates and tear film inflammatory markers.

## Differential Diagnosis

- Infectious keratitis
- Smile stain (if restricted to inferior zones)

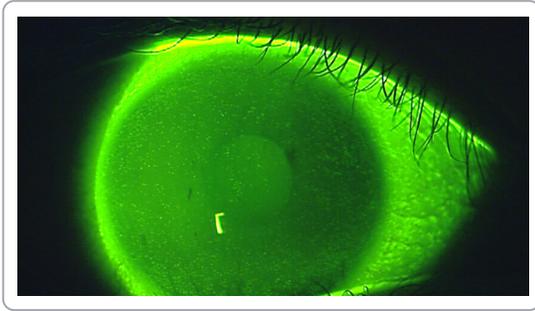


Figure 1: Example of SICS

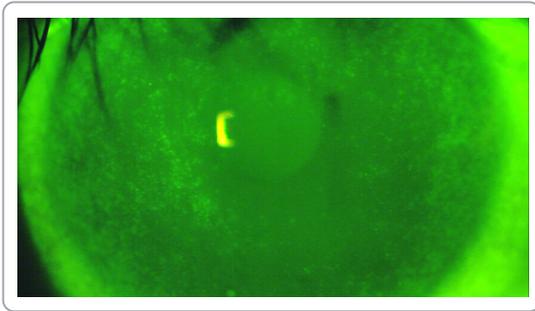


Figure 2 Example of SICS

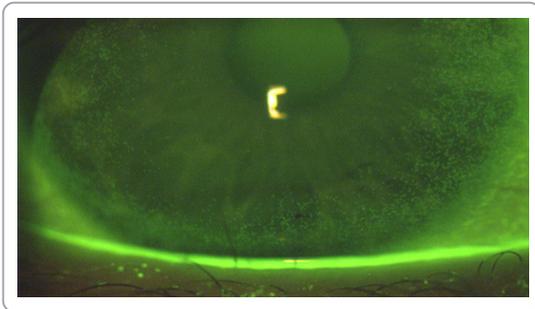


Figure 3: Example of SICs donut staining pattern

## Describing clinical appearance

No specific grading scheme for SICS, but can grade each corneal zone with the % affected by staining

124. Andraso G, Ryan K. Corneal staining and comfort observed with traditional and silicone hydrogel lenses and multipurpose solution combinations. *Optometry* 2008; 79: 444-454

125. Bandamwar K et al. Onset time course of solution induced corneal staining. *Cont Lens Anterior Eye* 2010; 33: 199-201.

126. Bright FV et al. A preservative-and-fluorescein interaction model for benign multipurpose solution-associated transient corneal hyperfluorescence. *Cornea* 2012; 31: 1480-1488

127. Carnt N et al. Solution toxicity in soft contact lens daily wear is associated with corneal inflammation. *Optom Vis Sci* 2007; 84: 309-15.

128. Carnt N et al. Corneal staining: The IER matrix study. *Contact Lens Spectrum* 2007; 22: 38-43

129. Garofalo RJ et al. Corneal staining and subjective symptoms with multipurpose solutions as a function of time. *Eye Contact Lens* 2005; 31: 166-174

130. Jones L et al. Asymptomatic corneal staining associated with the use of balaafilcon silicone-hydrogel contact lenses disinfected with a polyaminopropyl biguanide-preserved care regimen. *Optom Vis Sci* 2002; 79: 753-761

131. Khan TF et al. Cellular fluorescein hyperfluorescence is dynamin-dependent and increased by Tetronic 1107 treatment. *Int J Biochem Cell Biol* 2018; 101: 54-63

132. Papas EB et al. Complications associated with care product use during silicone daily wear of hydrogel contact lens. *Eye Contact Lens* 2007; 33: 392-3; discussion 399-400.

133. Peterson R et al. Impact of a rub and rinse on solution-induced corneal staining. *Optom Vis Sci* 2010; 87: 1030-6.

# Cornea

## Superior Epithelial Arcuate Lesions (SEALs)

### Slit lamp viewing

Direct illumination, parallelepiped or diffuse, medium/high magnification (16 – 30x) with fluorescein with blue cobalt filter and yellow barrier filter

### Incidence

Higher incidence in early higher modulus SiHy materials

### Aetiology

- Mechanical trauma due to inflexible nature (high modulus, thick design) of some lenses (especially early SiHy materials) and misalignment between lens and ocular surface in superior cornea due to pressure of top lid on corneal epithelium
- Hypoxia may play a role as under the top lid

### Symptoms

Asymptomatic when lenses worn; foreign body sensation may occur on lens removal

### Signs

- Arcuate staining 1mm from superior limbus between 10 and 2 o'clock; parallel to limbus, 0.1 – 0.3mm wide; often unilateral and asymmetric
- Diffuse or focal infiltration

### Management

- Manage if  $\geq$  grade 2 or if  $\geq$  1 grading scale increase
- Remove lens for 2 – 4 days or until totally healed
- Ocular lubricants may help foreign body symptoms
- Change lens design or material (thinner, more flexible lens material, lower modulus, thinner periphery, flatter BOZR) or try rigid corneal lens

### Prognosis

Good – rapid and complete resolution with no scarring or vision loss

### Differential Diagnosis

- Infectious keratitis
- Edge pressure from tight rigid corneal lens fit (symptomatic)
- Epithelial split due to mechanical action of soft lens (symptomatic)

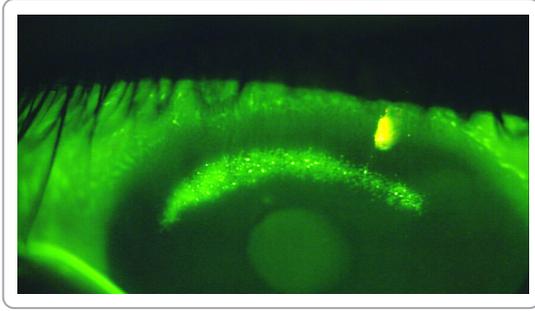


Figure 1: Superior epithelial arcuate lesion (SEAL) viewed with fluorescein

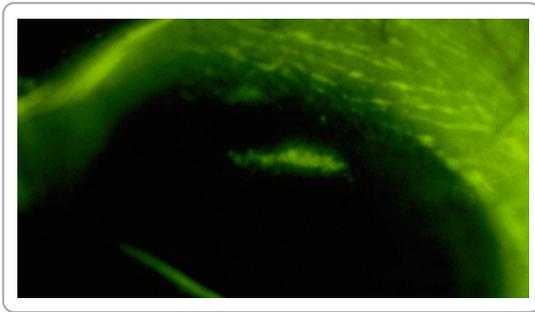


Figure 2: Superior epithelial arcuate lesion (SEAL) viewed with fluorescein

## Describing clinical appearance

### Extent

Describe position, shape and size

### Depth

- A: No stromal diffusion
- B: Stromal diffusion delayed (30 – 60 seconds)
- C: Stromal diffusion immediate but moderate
- D: Stromal diffusion immediate and widespread

134. Dumbleton K. Adverse events with silicone hydrogel continuous wear. *Cont Lens Ant Eye* 2002; Sep 25 (3): 137-46
135. Dumbleton K. Noninflammatory silicone hydrogel contact lens complications. *Eye Contact Lens* 2003; Jan 29 (1 Suppl): S186-189
136. Holden BA et al. Superior Epithelial Arcuate Lesions with Soft Contact Lens Wear. *Optom Vis Sci* 2001; 78(1): 9-12
137. Jalbert I et al. The characteristics of corneal staining in successful daily and extended disposable contact lens wearers. *Clin Exp Optom* 1999; 82: 4-10
138. Jalbert I et al. Epithelial split associated with wear of a silicone hydrogel contact lens. *CLAO J* 2001; 27: 231-3.
139. Lin M, & Yeh T. Mechanical complications induced by silicone hydrogel contact lenses. *Eye & CL* 2013; 39(1): 115-124
140. Young G, Mirejovsky D. A hypothesis for the Aetiology of soft contact lens induced arcuate keratopathy. *Int Contact Lens Clin* 1993; 20 (9): 177-179

# Cornea

## Mucin Balls

### Slit lamp viewing

Direct and indirect retro illumination, medium/high magnification (16 – 32x), fluorescein with blue cobalt filter and yellow barrier filter after lens removal

### Incidence

- Unknown — individual susceptibility varies substantially
- Higher with overnight wear, increasing with number of nights lenses slept in
- Seen more with higher modulus materials

### Aetiology

- Seen with overnight wear and higher modulus SiHys; material derived from pre-ocular tear film which collapses and is formed into spheroids (approx 50µm) due to relative motion between the CL and corneal surface
- Association with corneal infiltrative events (CIEs):
- Early formation after fitting associated with increased risk of CIEs
- Repeated presence of mucin balls over an extended period of time mildly protective against CIEs

### Symptoms

None; vision may be affected in extreme cases

### Signs

- Lens in place: translucent spheres of trapped debris behind lens — range in size 20 to 200µm, pre-epithelial location and display un-reversed illumination
- Lens removed: usually disappear within a few blinks after lens removal leaving superficial punctate pooling of fluorescein (appearance similar to dimple veil) resulting from indentation of epithelium by mucin balls

## Management

- Often not required as vision and comfort unaffected
- If noted early during the fitting process they are a potential marker for CIE - consider avoiding overnight wear
- Rapid resolution of indentations after lens removal
- Optimize lens fit with steeper base curve and closer match of lens shape to ocular surface
- Ocular lubricants before and after sleep for planned overnight wear
- Reduce number of consecutive nights without lens removal (e.g. from 30 to 6 nights)
- Refit with lower modulus lens or steeper base curve or alternative design

## Prognosis

Excellent – Epithelial filled indentations disappear within hours following lens removal

## Differential Diagnosis

- Microcysts (reversed illumination), vacuoles (both intra-epithelial and persist when lens removed)
- Dimple veil, epithelial bullae, lens deposit



Figure 1: Mucin balls visible with medium-high magnification and diffuse white light

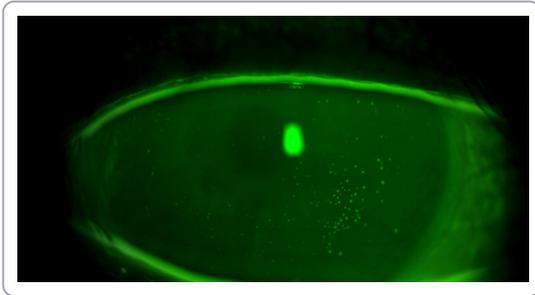


Figure 2: Mucin ball indents filled with fluorescein following lens removal

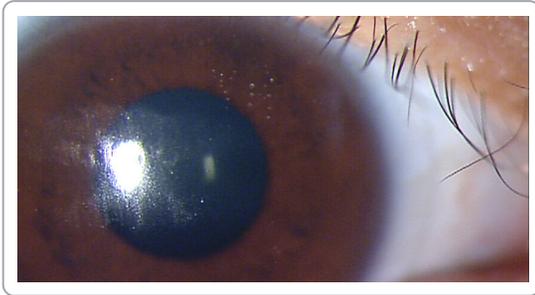


Figure 3: Mucin balls visible under soft lens

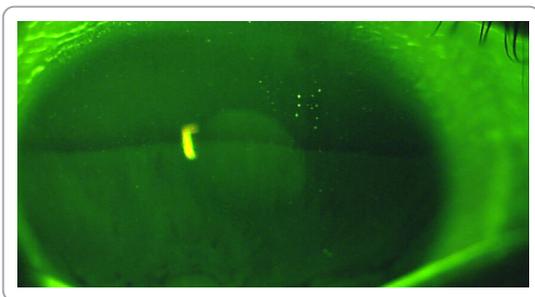


Figure 4: Mucin ball indents filled with fluorescein following lens removal (same eye as Figure 3)

## Describing clinical appearance

- 0: None
- 1: 0 – 20
- 2: 20 – 50
- 3: 50 – 100
- 4: >100

141. Dumbleton K et al. Clinical characterization of spherical post-lens debris associated with lotrafilcon high-Dk silicone lenses. *CLAO J* 2000; 26: 186-92.

142. Dumbleton K. Noninflammatory silicone hydrogel contact lens complications. *Eye Contact Lens* 2003 Jan; 29 (1 Suppl): S186-189

143. Lin M, & Yeh T (2013) Mechanical complications induced by silicone hydrogel contact lenses. *Eye & CL* 2013; 39(1): 115-124

144. Miller TJ et al. Clinical Appearance and Microscopic Analysis of Mucin Balls Associated with Contact Lens Wear. *Cornea* 2003; 22: 740-745

145. Pritchard N et al. Epithelial inclusions in association with mucin ball development in high-oxygen permeability hydrogel lenses. *Optom Vis Sci* 2000; 77: 68-72.

146. Szczotka-Flynn L et al. The association between mucin balls and corneal infiltrative events during extended contact lens wear. *Cornea* 2011; 30(5): 535-542

147. Szczotka-Flynn L et al. Mucin Balls Influence Corneal Infiltrative Events. *Optom Vis Sci* 2017; 94(4): 448-457

148. Tan J et al. Mucin balls with wear of conventional and silicone hydrogel contact lenses. *Optom Vis Sci* 2003; 80 (4): 291-297

# Cornea

## Dimple Veil

### Slit lamp viewing

Direct illumination paralleliped, indirect retro illumination, medium/high magnification (16 – 30x), fluorescein with blue cobalt filter and yellow barrier filter

### Incidence

- Exact prevalence unknown
- Could occur in all lenses; most prevalent in rigid lenses and Ortho-K
- an occur after sub-aqua diving and flying at high altitudes

### Aetiology

- Indentations in epithelium due to air bubbles trapped under lens
- Due to poor lens fit relationship between cornea and posterior lens surface (steep lenses, periphery of high riding lenses, edge stand off)
- Ortho-K – air bubbles being trapped behind the lens

### Symptoms

Normally asymptomatic

### Signs

- Small (10-200µm) spherical indentations in epithelium fill with aqueous phase tear film that stains; appearance similar to the surface of a golf ball
- Display un-reversed illumination

### Management

- Manage if  $\geq$  grade 2 or if  $\geq$  1 grading scale increase
- Remove lenses until staining resolved if severe
- Modify lens fit:
  - Central position: fit flatter central BOZR
  - Peripheral position: reduce edge clearance, total diameter and lens thickness; change to toric back surface
  - Scleral lenses: Alter lens specifications to reduce quantity of tear in the post lens tear film reservoir
- Ortho-K: advise on applying a viscous ocular lubricant to the back of the lens before inserting. Careful application that does not introduce new bubbles of its own can help reduce the chances of air bubbles forming

### Prognosis

- Good – immediate resolution once fitting relationship improved
- Ortho-K – not a concern if attempts to rectify are unsuccessful in resolving

### Differential Diagnosis

Corneal staining

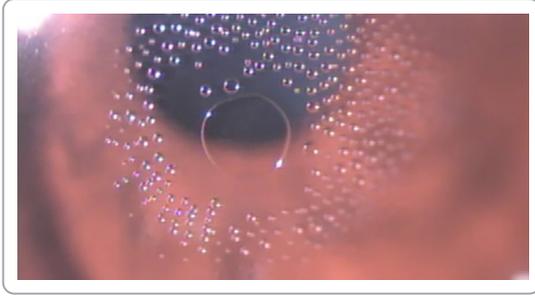


Figure 1: Dimple veil visible with diffuse white light view

### Describing clinical appearance

Note not 'true' staining but pooling of stain to record. Record area and approximate number seen.

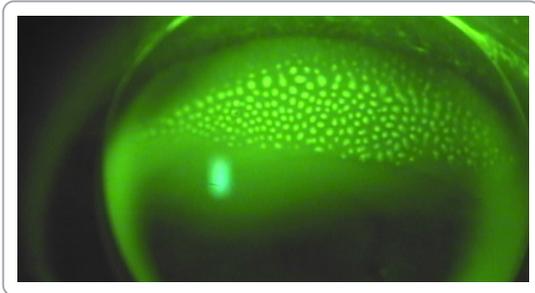


Figure 2: Dimple veil visible with fluorescein and blue light illumination

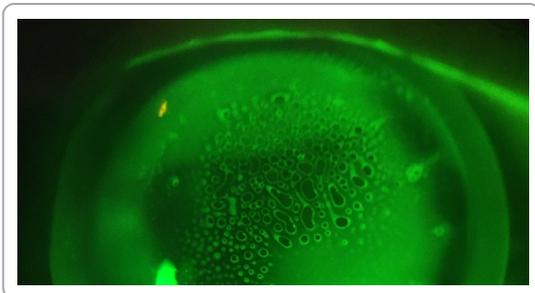


Figure 3: Dimple veil visible with fluorescein and blue light illumination

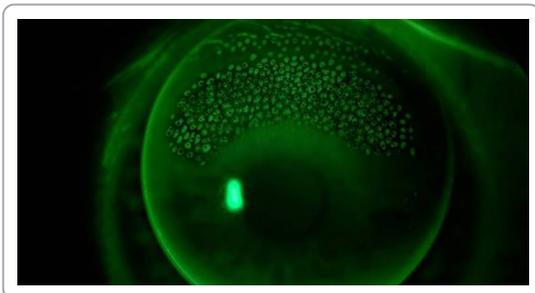


Figure 4: Dimple veil visible with fluorescein and blue light illumination

149. Fadel D. The influence of limbal and scleral shape on scleral lens design. *Cont Lens Anterior Eye* 2018; 41: 321-328.

150. Flynn W et al. Contact lens wear at altitude: subcontact lens bubble formation. *Aviat Space Environ Med* 1987; 58: 1115-8.

151. Jones L, Jones D. Dimple-veil staining. *Optician* 1995; 210 (5509): 32

152. Stillitano I et al. Bubble and corneal dimple formation after the first overnight wear of an orthokeratology lens: a case series. *Eye Contact Lens* 2007; 33: 253-8.

153. Strath R et al. Tear film bubble formation after decompression. *Optom Vis Sci* 1992; 69 (12): 973-975

# Cornea

## Epithelial Microcysts

### Slit lamp viewing

Indirect retro illumination, high magnification (25 – 40x)

### Incidence

- Almost always associated with soft lenses
- Hydrogel DW 10 – 20%, hydrogel overnight wear 100%, not caused with SiHy wear other than rebound phenomenon when refitting from lower Dk/t into SiHy
- Up to 50% population non-CL wearers minimal numbers (4% population have >10 microcysts)

### Aetiology

Chronic hypoxia leading to corneal oedema and epithelial microcysts

### Symptoms

None or mild hazing of vision in very severe cases

### Signs

- 10 – 50µm epithelial vesicles in superficial epithelium seen 2 – 3 months after overnight wear; if break through surface, see fine punctate staining
- Display reversed illumination (refractive index higher than surrounding tissue)

### Management

- If  $\leq$  grade 2, monitor but no action necessarily required. However, their presence is a sign of chronic hypoxia and can be eliminated with refitting with high Dk rigid corneal lenses or SiHy
- $\geq$  grade 3 – consider temporary cessation lens wear, although cornea can rehabilitate with refit straight into SiHy lenses
- Increase oxygen supply to cornea – higher oxygen performance lenses, convert from overnight wear to DW, reduce wearing time

### Prognosis

- Numbers may increase immediately after lens removal or refit with SiHy lenses and then gradually decrease over 3-4 weeks
- Excellent – complete resolution after a few months (do not reoccur with SiHys)

### Differential Diagnosis

- Vacuoles (un-reversed illumination)
- Bubbles in tear film, tear film debris
- Mucin balls (un-reversed illumination)
- Dimple veil (pooling of fluorescein in epithelium)
- Punctate corneal staining
- Endothelial bedewing

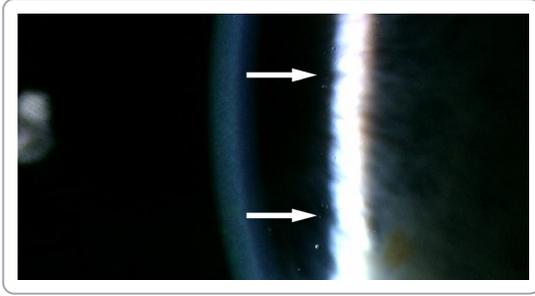


Figure 1: Microcysts, reversed illumination

### Describing clinical appearance

- 0: None
- 1: 1 – 10
- 2: 11 – 30
- 3: 31 – 70
- 4: >70

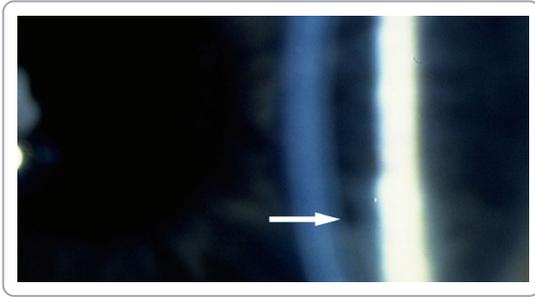


Figure 2: Vacuoles, unreversed illumination

154. Dumbleton K. Adverse events with silicone hydrogel continuous wear. *Cont Lens Anterior Eye* 2002; 25: 137-46.
155. Efron N. Contact lens induced epithelial microcysts. *Optician* 1996; 211 (5549): 24-29
156. Holden B, Sweeney D. The significance of the microcystic response: a review. *Optom Vis Sci* 1991; 68(9): 703-707
157. Jones L, Jones D. Non-inflammatory corneal complications of contact lens wear. *Cont Lens Anterior Eye* 2001; 24: 73-9.
158. Keay L et al. Microcyst response to high Dk/t silicone hydrogel contact lenses. *Optom Vis Sci* 2000; 77: 582-5.
159. Keay L et al. Microcysts: clinical significance and differential diagnosis. *Optometry* 2001; 72: 452-60.
160. Sweeney DF. Clinical signs of hypoxia with high-Dk soft lens extended wear: is the cornea convinced? *Eye Contact Lens* 2003; 29: S22-5; discussion S26-9, S192-4.
161. Zantos SG (1983) Cystic formation in the corneal epithelium during extended wear of CLs. *Int Contact Lens Clin* 1983; 10(3): 128-146
162. Efron N. Contact lens induced corneal oedema. *Optician* 1996; 211 (5540): 18-27

# Cornea

## Striae

### Slit lamp viewing

Direct illumination, parallelepiped, high magnification view of stroma (25 – 40x)

### Incidence

More prevalent in soft hydrogel lenses, especially low Dk/t or overnight wear

### Aetiology

- Hypoxia – lactic acid accumulation in cornea, subsequent osmotic shift in stroma and corneal oedema
- Fluid separation of collagen fibrils seen with  $\geq 5\%$  oedema
- Striae often appear vertical in slit lamp viewing, although different directions present when viewed with confocal microscopy

### Symptoms

Normally asymptomatic

### Signs

- Fine, white, vertically oriented lines in posterior stroma
- Number increase proportionally with increasing oedema and striae become gray and thicker

### Management

- Switch from overnight wear to DW
- Increase oxygen supply to cornea with high oxygen performance lens (SiHy, high Dk rigid corneal lens) that meet Dk/t thresholds to avoid swelling in all corneal regions
- Scleral lens – increase lens Dk/t, reduce lens clearance
- Minimum reported average Dk/t thresholds to avoid swelling:-
- DW 24 x 10<sup>-9</sup>, overnight wear 87 x 10<sup>-9</sup> (Holden & Mertz, 1984)
- DW 35 x 10<sup>-9</sup>, overnight wear 125 x 10<sup>-9</sup> (Harvitt & Bonnano, 1999)
- DW (central) 19.8 x 10<sup>-9</sup>, DW (peripheral) 32.6 x 10<sup>-9</sup> (Morgan and Brennan, 2010)

### Prognosis

Good

### Differential Diagnosis

Corneal nerves, ghost vessels, folds, herpes simplex ulcer, dendritic scarring, keratoconus, other corneal pathologies/ dystrophies

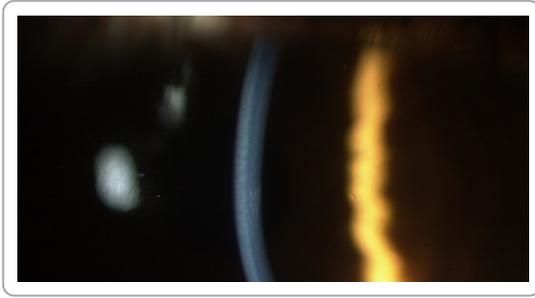


Figure 1: Striae, visible in optic section

### Describing clinical appearance

Count and record number of striae 1 striae = 5% oedema; each additional striae approximately 1% increase incremental oedema



Figure 2: Differential diagnosis: Vogt's striae

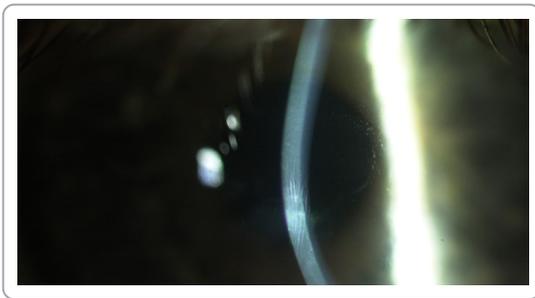


Figure 3: Differential diagnosis: Vogt's striae

162. Efron N. Contact lens induced corneal oedema. *Optician* 1996; 211 (5540): 18-27

163. Efron N et al. Confocal microscopic observations of the human cornea following overnight contact lens wear. *Clin Exp Optom* 2002; 85(3): 149-155

164. Harvitt D, Bonnano J. Re-evaluation of the oxygen diffusion model for predicting minimum contact lens Dk/t values needed to avoid corneal anoxia. *Optom Vis Sci* 1999; 76 (10): 712-719

165. Holden B, Mertz G. Critical oxygen levels to avoid corneal oedema for daily and extended wear contact lenses. *Invest Ophthalmol Vis Sci* 1984; 25: 1161-1167

166. Jalbert I, Stapleton F. The corneal stroma during contact lens wear. *Cont Lens Anterior Eye* 2005; 28: 3-12.

167. La Hood D, Grant T. Striae and folds as indicators of corneal oedema. *Optom Vis Sci* 1990; 2183 67(S): 196

168. La Hood D. Daytime oedema levels with plus powered low and high water content hydrogel contact lenses. *Optom Vis Sci* 1991; 68: 877-80.

169. Morgan PB et al. Central and peripheral oxygen transmissibility thresholds to avoid corneal swelling during open eye soft contact lens wear. *J Biomed Mater Res B Appl Biomater* 2010; 92(2): 361-365

# Cornea

## Folds

### Slit lamp viewing

Direct illumination, high magnification view of posterior stroma (25 – 40x), specular reflection (view endothelial topography)

### Incidence

- More prevalent in soft hydrogels worn for overnight wear, although very unlikely to see now with new, higher oxygen performance lens materials
- May also occur in diabetes and certain corneal pathologies (e.g. keratoconus)

### Aetiology

Hypoxia and high levels of corneal oedema leading to physical buckling of posterior stroma; seen with  $\geq 8\%$  oedema

### Symptoms

- Hazy vision
- May be some reported discomfort

### Signs

- Long, straight, dark lines seen in endothelial mosaic or as buckling in posterior stroma with higher levels oedema (hydrogel lens wear overnight – 8%)
- $> 4$  folds =  $> 10\%$  oedema and increase in number as level of oedema increases

### Management

- Switch from overnight wear to DW
- Increase oxygen supply to cornea with high oxygen performance lens (SiHy, high Dk rigid corneal lens (RCL)) that meet Dk/t thresholds to avoid swelling in all corneal regions
- Scleral lens – increase lens Dk/t, reduce lens clearance
- Minimum reported Dk/t thresholds to avoid swell:
  - DW 24 x 10<sup>-9</sup>, overnight wear 87 x 10<sup>-9</sup> (Holden & Mertz, 1984)
  - DW 35 x 10<sup>-9</sup>, overnight wear 125 x 10<sup>-9</sup> (Harvitt & Bonnano, 1999)
  - DW (central) 19.8 x 10<sup>-9</sup>, DW (peripheral) 32.6 x 10<sup>-9</sup> (Morgan & Brennan, 2010)

### Prognosis

Good

### Differential Diagnosis

Corneal pathology (dystrophies or disease), increased IOP, corneal nerves, normal aging change, anterior corneal folds from hypotony

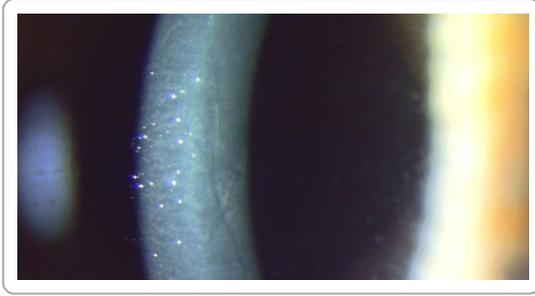


Figure 1: Corneal folds

## Describing clinical appearance

Count and record number of folds:

1 fold = 8% oedema

170. Abualhasan H et al. Anterior Corneal Folds Correlate With Low Intraocular Pressure and May Serve as a Marker for Ocular Hypotony. *J Glaucoma* 2019; 28(2): 178-180

171. Efron N. Contact lens induced corneal oedema. *Optician* 1996; 211 (5540): 18-27

172. Efron N et al. Confocal microscopic observations of the human cornea following overnight contact lens wear. *Clin Exp Optom* 2002; 85: 149-55.

173. Efron N. Contact lens-induced changes in the anterior eye as observed in vivo with the confocal microscope. *Prog Retin Eye Res* 2007; 26: 398-436.

174. Harvitt D, Bonnano J. Re-evaluation of the oxygen diffusion model for predicting minimum contact lens Dk/t values needed to avoid corneal anoxia. *Optom Vis Sci* 1999; 76(10): 712-719

175. Holden B, Mertz G. Critical oxygen levels to avoid corneal oedema for daily and extended wear contact lenses. *Invest Ophthalmol Vis Sci* 1984; 25: 1161-1167

176. Morgan PB et al. Central and peripheral oxygen transmissibility thresholds to avoid corneal swelling during open eye soft contact lens wear. *J Biomed Mater Res B Appl Biomater* 2010; 92(2): 361-365

177. Polse KA et al. Clinical assessment of corneal hydration control in Fuchs' dystrophy. *Optom Vis Sci* 1991; 68: 831-41.

# Cornea

## Scarring

### Slit lamp viewing

Direct and indirect illumination, medium / high magnification (16 – 30x), sclerotic scatter or optic section to identify location

### Incidence

Can occur with and without CL wear

### Aetiology

- Old pathology (inflammation, infection)
- Corneal surgery
- Trauma

### Symptoms

- None; depends on location
- If central, vision may be affected

### Signs

Depends on Aetiology

### Management

- None – observation only and record for baseline
- Vision may be improved with rigid corneal lens or scleral lens if cornea is distorted

### Prognosis

Irreversible; may be some decrease in intensity with time

### Differential Diagnosis

Active pathology (MK, CLPU), corneal dystrophies, infiltrate

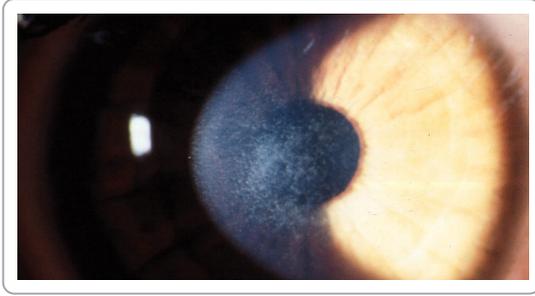


Figure 1: Post refractive surgery (PRK) scar

### Describing clinical appearance

Describe size, depth and location (superior, inferior, nasal, temporal, central)

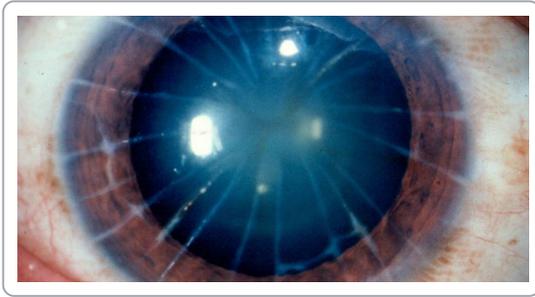


Figure 2: Marked radial keratotomy (RK) refractive surgery scars



Figure 3: Scarring, unknown cause

178. Hassan O et al. Management of Corneal Scarring Secondary to Herpes Zoster Keratitis. *Cornea* 2017; 36: 1018-1023.

179. Kwok S et al. Systematic Review on Therapeutic Strategies to Minimize Corneal Stromal Scarring After Injury. *Eye Contact Lens* 2019; 45: 347-355.

180. Parra A et al. Assessment of the Prosthetic Replacement of Ocular Surface Ecosystem (PROSE) scleral lens on visual acuity for corneal irregularity and ocular surface disease. *Ocul Surf* 2018; 16: 254-258.

181. Singh K et al. Rehabilitation of vision disabling corneal opacities: is there hope without corneal transplant? *Cont Lens Anterior Eye* 2013; 36: 74-9.

# Cornea

## Asymptomatic Infiltrative Keratitis (AIK)

**Asymptomatic Infiltrative Keratitis (AIK), also known as Asymptomatic Corneal Infiltrative Events (CIEs)**

### Slit lamp viewing

Direct focal illumination, medium/high magnification (16 – 25x) in white light and then with fluorescein with blue cobalt filter and yellow barrier filter

### Incidence

- 1 – 7% daily soft lens wear, with up to 20% in overnight wear of soft lenses in clinical trials (note asymptomatic nature means more seen in prospective trials than in routine practice)
- Occurs in non-lens wearers

### Aetiology

Sterile corneal infiltrates – inflammatory cells from limbal blood vessels form white spots in cornea in response to: hypoxia, closed eye environment, bacterial toxins, lens deposits, care systems, lid margin disease, poor hygiene, allergic reaction, adenoviral infection

### Symptoms

None (possibly mild bulbar conjunctival hyperaemia)

### Signs

- Small, focal non-staining infiltrates (up to 0.4mm) in cornea
- May be fine punctate staining
- Mild limbal and bulbar redness

### Management

- Temporary discontinuation lens wear if severe until infiltrates disappear
- Careful monitoring, ocular lubricants and lid hygiene
- No medication required in most cases
- Change to DW, frequent replacement, higher oxygen performance lens and alter care regimen to unpreserved system or switch to DD
- Resolution often within 14 days (longer with greater severity)

### Prognosis

- Very rarely scars so prognosis good as long as visual axis not involved – resolves within 2 weeks
- Certain subjects prone to recurrent inflammation – stop any EW

### Differential Diagnosis

Epidemic keratoconjunctivitis (adenoviral), IK, CLPU, CLARE, MK, corneal dystrophy, corneal nerves, corneal scar

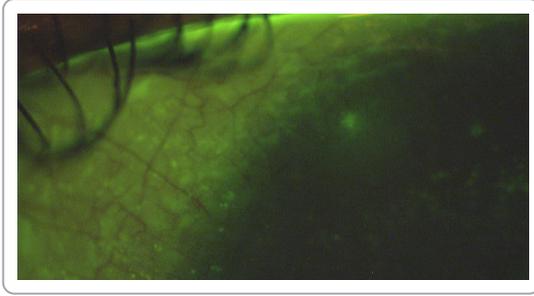


Figure 1: Asymptomatic infiltrative keratitis staining with fluorescein

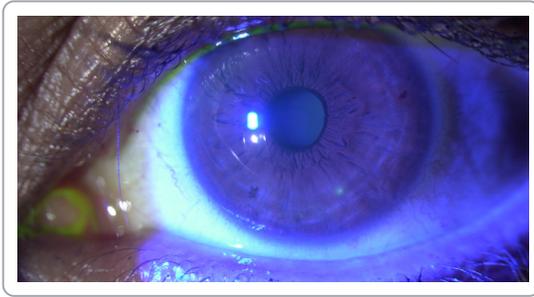


Figure 2: Asymptomatic infiltrative keratitis staining with fluorescein

## Describing clinical appearance

Describe number, size and location (superior, inferior, nasal, temporal; central or peripheral)

182. Cutter G et al. The clinical presentation, prevalence and risk factors of focal corneal infiltrates in soft contact lens wearers. *CLAO J* 1996; 22 (1): 30-37
183. Donshik P. Peripheral corneal infiltrates and contact lens wear. *CLAO J* 1998; 24 (3): 134-136
184. Hickson S, Papas E. Prevalence of idiopathic corneal anomalies in a non contact lens-wearing population. *Optom Vis Sci* 1997; 74: 293-7.
185. Szczotka-Flynn L, Chalmers R. Incidence and epidemiologic associations of corneal infiltrates with silicone hydrogel contact lenses. *Eye Contact Lens* 2013; 39: 49-52.
186. Szczotka-Flynn L et al. Corneal inflammatory events with daily silicone hydrogel lens wear. *Optom Vis Sci* 2014; 91: 3-12.
187. Steele KR, Szczotka-Flynn L (2017) Epidemiology of contact lens-induced infiltrates: an updated review. *Clin Exp Optom*; 100: 473-481
188. Sweeney DF et al. Clinical characterization of corneal infiltrative events observed with soft contact lens wear. *Cornea* 2003; 22: 435-42

# Cornea

## Infiltrative Keratitis (IK)

### Also known as Corneal Infiltrative Events (CIEs)

### Slit lamp viewing

Direct focal illumination, medium/high magnification (16 – 25x) in white light and then with fluorescein with blue cobalt filter and yellow barrier filter; white light optic section to assess depth

### Incidence

- 3% DW soft lens wear, 2.5-6% EW soft lens wear, 2x increased risk with reusable SiHys (DW or overnight wear), <0.5% DD wear
- Can occur in non-lens wearers

### Aetiology

- Infiltrative event other than those identified as CLPU, CLARE or MK
- Sterile corneal infiltrates – inflammatory reaction with anterior stromal infiltration due to: hypoxia, closed eye, tight lens, bacterial toxins, denatured lens deposits, solution sensitivity, lid margin disease, poor hygiene, allergic reaction, adenoviral infection, mechanical trauma
- Risk factors: high ametropia (>5D), younger age (15-25 years), older age (>50 years), males, lens case contamination, environmental influences, previous CIE, use of MPS, bacterial bioburden, solution induced corneal staining (SICS), smoking (current or past)

### Symptoms

- Lens intolerance and foreign body sensation
- Photophobia and lacrimation
- Episodes of acute red eye

### Signs

- Moderate bulbar redness, focal stromal infiltrates (described as marginal keratitis if at 4 and 8 o'clock and due to staphylococcal exotoxins from lid margins)
- With or without epithelial involvement (staining) and can be bilateral

## Management

- Temporary discontinuation of lens wear until infiltrates disappear and no other signs or symptoms
- Careful monitoring, ocular lubricants, lid hygiene
- No medication required (in most cases) topical antibiotic and anti-inflammatory may be considered if severe and/or on visual axis
- Resolution often within 14 days (longer with greater severity)
- Reduce lid margin bioburden and organism contamination
- Address case hygiene and replacement
- For SICS, alter combination of SiHy and MPS, switch to Hydrogen Peroxide system, or change to DD
- Once resolved, change to DW, increase lens oxygen performance, loosen lens fit, increase lens replacement frequency, change care system, review lens handling and hygiene

## Prognosis

- Rarely scars so prognosis good as long as visual axis not involved – resolves within 2 weeks
- Small residual scars may result depending on cause and depth of infiltration
- Certain subjects prone to recurrent inflammation – stop any overnight wear

## Differential Diagnosis

- MK
- epidemic keratoconjunctivitis
- corneal dystrophies
- corneal nerves
- herpes simplex
- old corneal scar



Figure 1: Infiltrative keratitis



Figure 2: Infiltrative keratitis

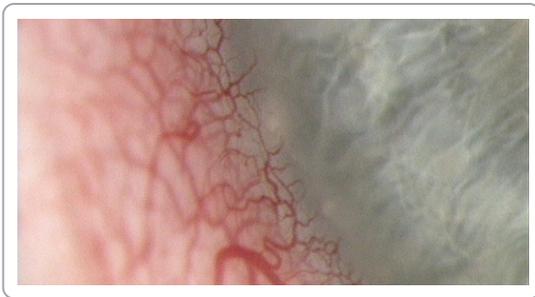


Figure 3: Marginal infiltrates with associated hyperaemia

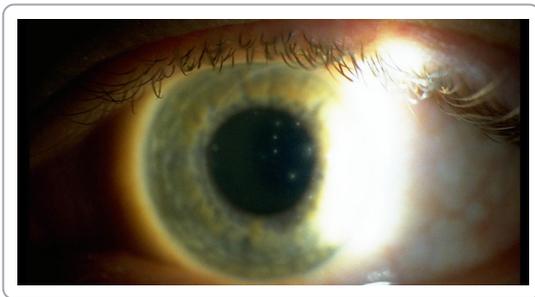


Figure 4: Infiltrative keratitis

## Describing clinical appearance

Describe size, location (superior, inferior, nasal, temporal; central or peripheral) and depth of lesion

189. Carnt N et al. Solution toxicity in soft contact lens daily wear is associated with corneal inflammation. *Optom Vis Sci* 2007; 84(4): 309-315
190. Chalmers R et al. Age and other risk factors for corneal infiltrative and inflammatory events in young soft contact lens wearers from the Contact Lens Assessment in Youth (CLAY) study. *Invest Ophthalmol Vis Sci* 2011; 52(9): 6690-6696
191. Chalmers R et al. Multicentre case-control study of the role of lens materials and care products on the development of corneal infiltrates. *Optom Vis Sci* 2012; 89 (3): 316-325
192. Chalmers R et al. Rates of adverse events with hydrogel and silicone hydrogel daily disposable lenses in a large postmarket surveillance registry: The TEMPO registry. *Invest Ophthalmol Vis Sci* 2015; 52(1): 654-653
193. Ozkan J et al. Risk factors for corneal inflammatory and mechanical events with extended wear silicone hydrogel contact lenses. *Optom Vis Sci* 2010; 87 (11): 847-853
194. Papas EB et al. Complications associated with care product use during silicone daily wear of hydrogel contact lens. *Eye Contact Lens* 2007; Nov 33(6 Pt 2): 392-393; discussion 399-400
195. Radford C et al. Risk factors for nonulcerative contact lens complications in an ophthalmic accident and emergency department: a case-control study. *Ophthalmology* 2009; 16: 385-392
196. Steele KR, Szczotka-Flynn L. Epidemiology of contact lens-induced infiltrates: an updated review. *Clin Exp Optom* 2017; 100: 473-481
197. Sweeney D et al. Clinical characterization of corneal infiltrative events observed with soft contact lens wear. *Cornea* 2003; 22: 435-42.
198. 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; 84: 247-56

# Cornea

## Contact Lens Peripheral Ulcer (CLPU)

**Also known as CNPU, sterile corneal ulcer, staining infiltrate, sterile keratitis, sterile infiltrate**

### Slit lamp viewing

Direct focal illumination, medium/high magnification (16 – 25x) in white light and then fluorescein with blue cobalt filter and yellow barrier filter; white light optic section to assess depth

### Incidence

- DW 1 – 5% (SiHys > hydrogels)
- Overnight wear 1 – 13% (hydrogel), 3.3 – 5.4% (SiHys)

### Aetiology

- Inflammatory reaction with focal excavation epithelium, infiltration and necrosis of the anterior stroma (Bowman's layer intact)
- Inflammatory response to exotoxins from gram positive bacteria (esp. staphylococcus spp)
- Due to: bacterial contamination, closed eye, tight lens, poor hygiene, lid margin disease
- Risk factors: high ametropia (>5D), younger age (15-25 years), case contamination, environmental influences

### Symptoms

- Up to 50% asymptomatic and unaware of complication
- Lens intolerance, foreign body sensation, photophobia, lacrimation, episodes acute red eye
- Symptoms reduce after lens removal

### Signs

- Moderate, localized hyperaemia
- Sterile infiltrate – peripheral / mid- peripheral, small (0.2 – 2.0mm), single focal, circular, stains and diffuse infiltration (in acute phase) (can be multiple, although rare)
- Anterior chamber reaction if severe (rare)

## Management

- Self-limiting on removal but close monitoring for 24 hours to ensure differential diagnosis from infected MK, (if central, >1mm and pain, treat suspiciously)
- Follow up depends on severity
- Cease lens wear until epithelium intact over lesion (up to 14 days)
- Ocular lubricants to prevent lid rubbing over affected area and dilute bacterial toxins
- Oral analgesics to reduce discomfort (if severe)
- Severe cases, acute red eye or no resolution with lens removal — consider prophylactic antibiotic with/without anti-inflammatory (esp. if infiltrates on visual axis)
- Eliminate bacterial source — lid hygiene, and review hygiene practices
- Increase lens replacement frequency, change care system, refit into DD, or rigid corneal lens
- If in planned overnight wear, consider limiting to six nights at a time or DW
- Review case hygiene and replacement

## Prognosis

- Good if visual axis not involved — single, spherical scar often remains, but can fade with time
- Certain subjects prone to recurrent inflammation (10 – 25%)

## Differential Diagnosis

IK, MK, corneal dystrophies, herpes simplex, stromal scar

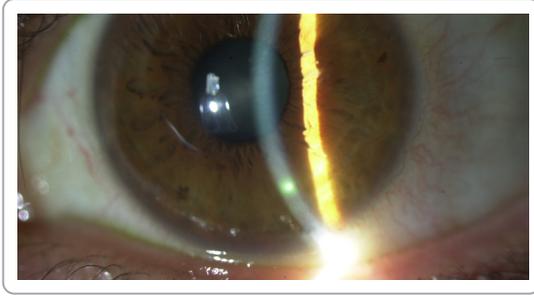


Figure 1: Active CLPU view in optic section

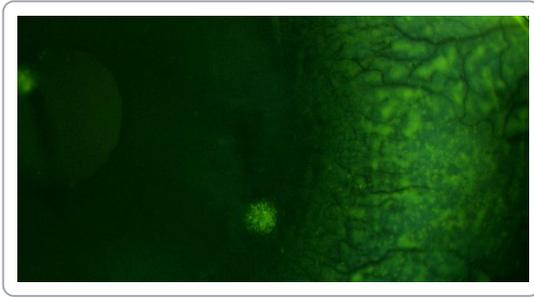


Figure 2: Active CLPU staining with fluorescein

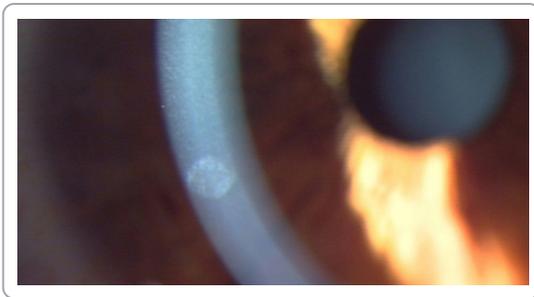


Figure 3: CLPU scar



Figure 4: CLPU scar

## Describing clinical appearance

Describe location (superior, inferior, nasal, temporal; central or peripheral) and size (digital image, or measure/estimate of diameter) and depth

199. Aasuri M et al. Differential diagnosis of microbial keratitis and contact lens-induced peripheral ulcer. *Eye Contact Lens* 2003 Jan; 29(1 Suppl): S60-62; discussion S83-84, S192-194
200. Chalmers RL et al. Risk factors for contact lens complications in US clinical practices. *Optom Vis Sci* 2010; 87(10): 725-735
201. Dumbleton K et al. Severity and management of contact lens related complications with continuous wear of high-Dk silicone hydrogel lenses. *Optom Vis Sci* 2000; 77(12s): 216
202. Dumbleton K. Adverse events with silicone hydrogel continuous wear. *Cont Lens Ant Eye* 2002; Sep 25(3): 137-46
203. Efron N, Morgan P. Can subtypes of contact lens-associated corneal infiltrative events be clinically differentiated? *Cornea* 2006 Jun; 25(5): 540-544
204. Grant T et al. Contact lens induced peripheral ulcers during hydrogel contact lens wear. *CLAO J* 1998; 24: 145-51.
205. Holden B 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: 538-43.
206. Ozkan J et al. Risk factors for corneal inflammatory and mechanical events with extended wear silicone hydrogel contact lenses. *Optom Vis Sci* 2010; 87(11): 847-853
207. Radford C et al. Risk factors for non-ulcerative contact lens complications in an ophthalmic accident and emergency department: a case-control study. *Ophthalmology* 2009; 116(3): 385-992
208. Wu P et al. The causes of and cures for contact lens-induced peripheral ulcer. *Eye Contact Lens* 2003; 29: S63-6; discussion S83-4, S192-4.

# Cornea

## Contact Lens Associated Red Eye (CLARE)

Also known as Acute Red Eye (ARE), Tight Lens Syndrome (TLS)

### Slit lamp viewing

Diffuse beam, direct illumination, medium magnification (16x)

### Incidence

Seen in overnight wear only – 1.4 – 12.3% (hydrogels), 1 – 3.8% with SiHys

### Aetiology

- Inflammatory reaction of cornea and conjunctiva subsequent to period of eye closure with CL wear due to endotoxins from gram negative bacteria (esp. *pseudomonas* spp)
- Seen with: closed eye (EW), tight lens, following upper respiratory tract infection

### Symptoms

- Can be woken in night with painful red eye
- Discomfort, photophobia, lacrimation

### Signs

- Acute unilateral circum-corneal and bulbar hyperaemia (> grade 2)
- Diffuse infiltrative keratitis – small focal, diffuse mid-peripheral corneal infiltrates (minimal staining)
- Anterior chamber reaction only if severe

### Management

- Self limiting on lens removal
- Temporary discontinuation of lens wear and ocular lubricants during active stage
- Careful monitoring for 12 – 24 hours to confirm diagnosis.
- Oral analgesics to reduce discomfort (if severe)
- If severe, topical prophylactic antibiotic or combo-drug (with steroid)
- hyperaemia resolves rapidly, infiltrates take longer
- Resume wear when infiltrates resolved and no more signs and symptoms
- Lid hygiene to reduce reoccurrence
- Refit with looser lens and/or switch to DW
- Avoid wearing ON when unwell

### Prognosis

Reoccurrence in 50% of patients reported with hydrogel overnight wear. Good – no scarring or vision loss

### Differential diagnosis

Conjunctivitis, keratoconjunctivitis, herpes simplex, CLPU, anterior uveitis, acute glaucoma

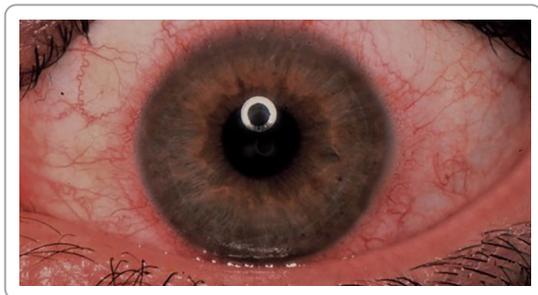


Figure 1: Contact lens associated red eye (CLARE)

### Describing clinical appearance

Describe affected areas of limbus, grade hyperaemia and note number, size and position of any infiltrates and corneal staining

209. Cole N et al. Different strains of *Pseudomonas aeruginosa* isolated from ocular infections or inflammation display distinct corneal pathologies in an animal model. *Curr Eye Res* 1998; 17: 730-5.
210. Dumbleton K. Adverse events with silicone hydrogel continuous wear. *Cont Lens Anterior Eye* 2002; Sep 25(3): 137-46
211. Efron N, Morgan P. Can subtypes of contact lens-associated corneal infiltrative events be clinically differentiated? *Cornea* 2006; 25: 540-4.
212. Holden B et al. Gram-negative bacteria can induce contact lens related acute red eye (CLARE) responses. *CLAO J* 1996; 24 (3): 145-151
213. Sankaridurg P et al. *Haemophilus influenzae* adherent to contact lenses associated with production of acute ocular inflammation. *J Clin Microbiol* 1996; 34: 2426-31.
214. Sweeney D. Recurrence of Acute Inflammatory Conditions with Hydrogel Extended Wear. *Invest Ophthal* 1993; 34(4): 1008
215. Willcox M, Hume E. Differences in the pathogenesis of bacteria isolated from contact-lens- induced infiltrative conditions. *Aust N Z J Ophthalmol* 1999; 27: 231-3

# Cornea

## Microbial Keratitis (MK)

**Also known as infected corneal ulcer, corneal abscess, suppurative keratitis, infectious keratitis, ulcerative keratitis**

### Slit lamp viewing

Direct illumination, medium/high magnification (16 – 25x) in white light and then fluorescein with blue cobalt filter and yellow barrier filter; white light optic section to assess depth

### Incidence

Rigid corneal lens (RCL) DW 0.01-0.04%, hydrogel DW 0.02-0.05%, SiHy DW 0.12%, hydrogel overnight wear 0.09-0.20%, SiHy overnight wear 0.18-0.25%, Ortho-K all ages 0.08%, Ortho-K kids 0.14%

### Aetiology

- Infection of compromised cornea (epithelial break, hypoxia) from invasion of bacteria (esp. epithelium, Bowman's layer and stroma with infiltration and necrosis of tissue)
- Risk factors: overnight wear, hypoxia, poor compliance and hygiene, swimming/showering in lenses, tap water, not storing CL case dry, male, smoking, trauma, poor general and ocular health (diabetes, respiratory disease), warm climates, socio-economic class, longer wearing periods, delay seeking treatment, high ametropia (>5D), younger age (15-25 years), lens case contamination, environmental influences

### Symptoms

- Severe pain with rapid onset, photophobia, epiphora, severe redness, reduced vision (depends on location), discharge, lid swelling
- No improvement after lens removal, pain usually increases

### Signs

- Examination can be difficult due to photophobia
- Full thickness epithelial defect with underlying infiltrate, Bowman's layer and stroma affected
- Generally central, large (>1mm), unilateral, irregular appearance
- Severe hyperaemia
- Anterior chamber activity (flare, hypopyon)
- Discharge and lid oedema

## Management

- Immediate discontinuation of lens wear – lenses and case not to be reused
- Ocular emergency – urgent management required (via Optometry or Ophthalmology depending on local scope of practice), corneal scrape, close monitoring and medical treatment
- No patching
- Intensive treatment (cycloplegic, analgesic, antimicrobial, NSAIDs)
- Advise about risk factors – improve hygiene, care regimen and avoid tap water
- Refit with higher oxygen performance lens (SiHy, rigid corneal lens (RCL)), change to DW, refit into DD
- Reduce bioburden and micro-organism contamination
- Review case hygiene (inc. rubbing & tissue wiping) and replacement

## Prognosis

- Variable, depends on causative organism and position of lesion – often resolves with scar and vascularisation
- Improved with rapid intervention
- 14% lose 2 lines or more best corrected VA;
- Better visual outcome with SiHy

## Differential diagnosis

CLPU, dense corneal staining, corneal abrasion



Figure 1: Large inferior central ulcer viewed under white light

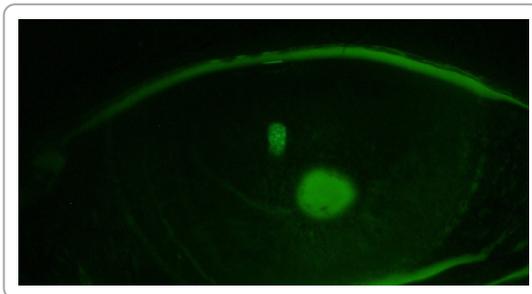


Figure 2: Large inferior central ulcer viewed with fluorescein

## Describing clinical appearance

Position: Superior, nasal, inferior, temporal, central

### Extent

- 0: None
- 1: 1 – 20 punctate diffuse spots
- 2: 21 – 40 punctate diffuse spots
- 3: > 40 diffuse spots and/or coalescing patches
- 4: Dense confluent patches

### Depth

- A: No stromal diffusion
- B: Stromal diffusion delayed (30 – 60 seconds)
- C: Stromal diffusion immediate but moderate
- D: Stromal diffusion immediate and widespread

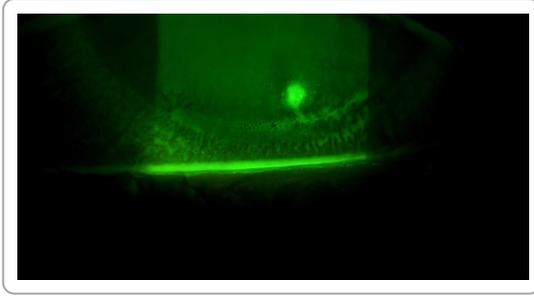


Figure 3: Inferior ulcer viewed with fluorescein

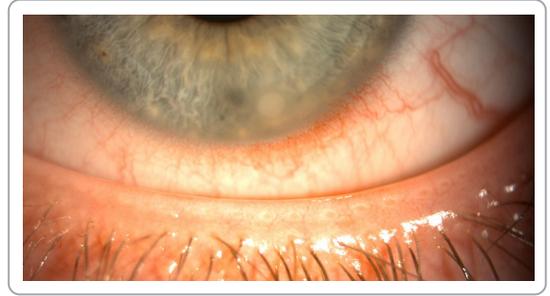


Figure 4: Microbial keratitis with associated limbal hyperaemia

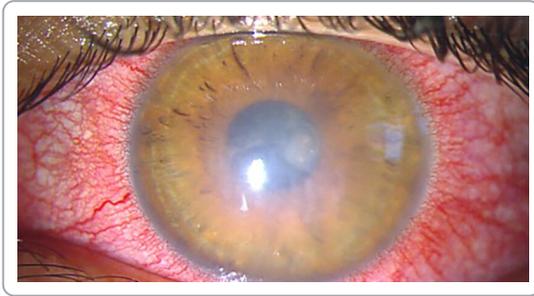


Figure 5: Microbial keratitis with associated conjunctival and limbal hyperaemia

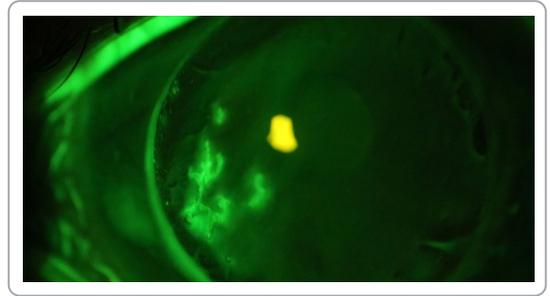


Figure 6: Herpes simplex keratitis viewed with fluorescein



Figure 7: Acanthamoeba keratitis



Figure 8: oedematous lids with active corneal ulcer visible

216. Bergmann J. Effects of contact lens wear on corneal ultrastructure. *Cont Lens Anterior Eye* 2001; 24: 115-20.
217. Bourne W. The effect of long-term contact lens wear on the cells of the cornea. *CLAO J* 2001 Oct; 27(4): 225-230
218. Efron N. Contact lens induced endothelial polymegathism. *Optician* 1996; 212(5575): 20-28
219. Doughty M, Fonn D. Pleomorphism and endothelial cell size in normal and polymegathous human corneal endothelium. *Int Contact Lens Clin* 1993; 20(5/6): 116-122
220. Holden B, Sweeney D. Corneal exhaustion syndrome (CES) in long-term contact lens wearers: a consequence of contact lens-induced polymegathism? *Am J Optom Physiol Opt* 1988; 65: 95.
221. McMahon T et al. Recovery from induced corneal oedema and endothelial morphology after long-term PMMA contact lens wear. *Optom Vis Sci* 1996; 73: 184-8.
222. Nieuwendaal C et al. Morphology and function of the corneal endothelium after long-term contact lens wear. *Invest Ophthalmol Vis Sci* 1994 Jun; 35(7): 3071-3077
223. Polse K et al. Hypoxic effects on corneal morphology and function. *Invest Ophthalmol Vis Sci* 1990; 31(8): 1542-1554
224. Sweeney D. Corneal exhaustion syndrome with long-term wear of contact lenses. *Optom Vis Sci* 1992; 69: 601-8

# Cornea

## Endothelial Changes - Polymegathism

### Slit lamp viewing

Endothelial cell mosaic viewed with specular reflection and high magnification (40x)

### Incidence

- Unknown — natural process of aging
- Condition accelerated by CLs of low oxygen transmission (Dk//t)

### Aetiology

- Long-term hypoxia causing long-term acidosis and structural damage to endothelial cells
- Factors other than CL wear (trauma, surgery, keratoconus, diabetes etc)

### Symptoms

- None
- Long-term — corneal exhaustion syndrome — discomfort, reduced wearing time and may develop sudden CL intolerance with reduced vision and photophobia

### Signs

- Differing cell sizes in endothelium
- Amount related to duration of lens wear and amount of hypoxia

### Management

- Refit with higher oxygen performance lenses (SiHy, rigid corneal lens (RCL))
- Change to DW
- Reduce wearing time

### Prognosis

- Poor — endothelium never reverts completely to normal endothelial mosaic, although grade of condition would be need be extremely severe to compromise the cornea and vision
- Long-term hypoxia and hypercapnia can lead to endothelial decompensation and corneal exhaustion syndrome

### Differential diagnosis

- Corneal dystrophies
- Blebs — transient endothelial cell oedema due to hypoxia/hypercapnia during adaptation to lens leading to black, non-reflecting areas; innocuous, asymptomatic and no management required

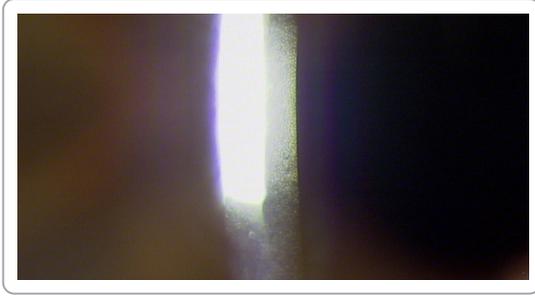


Figure 1: Corneal endothelial cells viewed in specular reflection

## Describing clinical appearance

Describe how much of visible area is affected: % of cells showing change, amount of variation in cell size and shape

225. Bergmanson J. Effects of contact lens wear on corneal ultrastructure. *Cont Lens Anterior Eye* 2001; 24: 115-20.
226. Bourne W. The effect of long-term contact lens wear on the cells of the cornea. *CLAO J* 2001 Oct; 27(4): 225-230
227. Efron N. Contact lens induced endothelial polymegathism. *Optician* 1996; 212(5575): 20-28
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230. McMahon T et al. Recovery from induced corneal oedema and endothelial morphology after long-term PMMA contact lens wear. *Optom Vis Sci* 1996; 73: 184-8.
231. Nieuwendaal C et al. Morphology and function of the corneal endothelium after long-term contact lens wear. *Invest Ophthalmol Vis Sci* 1994 Jun; 35(7): 3071-3077
232. Polse K et al. Hypoxic effects on corneal morphology and function. *Invest Ophthalmol Vis Sci* 1990; 31(8): 1542-1554
233. Sweeney D. Corneal exhaustion syndrome with long-term wear of contact lenses. *Optom Vis Sci* 1992; 69: 601-8.

# Cornea

## Lens Binding (Rigid Lenses)

### Slit lamp viewing

Diffuse illumination, low to medium magnification (10 – 16x), fluorescein with blue cobalt filter and yellow barrier filter

### Incidence

- Most common in rigid corneal lens (RCL) overnight wear (80% on eye opening, 25% persistent)
- Can occur with DW rigid corneal lenses (RCL) and scleral lenses

### Aetiology

- Eyelid pressure
- Corneal toricity
- Lens design – diameter, edge and periphery, thickness
- Lens material – flexibility
- Ortho-K – mechanical pressure
- Scleral lens – excessive lens flexure, low corneal clearance, tight landing zone

### Symptoms

- Often asymptomatic
- Comment on lens removal being difficult and spectacle blur on lens removal
- Scleral lens – discomfort, reduced wearing time

### Signs

- Immobile, decentered lens – indentation ring that stains on removal

- Post-lens debris
- Ortho-K: corneal staining, most likely central in myopia correction and para-central in hyperopia correction
- Scleral lens – complete or near completion lens adhesion minimizes tear exchange resulting in accumulation of toxic metabolic waste and subsequent oedema / inflammation.

### Management

- Temporary cessation of lens wear with clinically significant staining
- Alter lens fit – increase lens mobility (reduce TD, increase edge clearance, flatter BOZR)
- Ortho-K: see corneal staining management
- Scleral lens – alter lens design specifications to improve lens fit / reduce lens flexure

### Prognosis

- Fair – some patients continually have adherence – change to DW rigid corneal lens, low modulus SiHy materials or soft lenses
- Scleral lens – good once lens fit improved

### Differential diagnosis

- Record if found, describe area of adhesion and any associated staining



Figure 1: Rigid corneal lens bound to cornea

### Describing clinical appearance

Record if found, describe area of adhesion and any associated staining

234. Cho P et al.: Do fenestrations affect the performance of orthokeratology lenses? *Optom Vis Sci* 2012; 89: 401-10.

235. Chui W, Cho P. Recurrent lens binding and central island formations in a fast-responding orthokeratology lens wearer. *Optom Vis Sci* 2003; 80: 490-4.

236. Swarbrick H, Holden B. Ocular characteristics associated with rigid gas permeable lens adherence. *Optom Vis Sci* 1996; 73(7): 473-481

237. Swarbrick H, Holden B. Effects of lens parameter variation on rigid gas-permeable lens adherence. *Optom Vis Sci* 1996; 73: 144-55

# Cornea

## Corneal Distortion (Warpage)

### Slit lamp viewing

Not visible with slit lamp — seen with keratometry, retinoscopy and corneal topography

### Incidence

30% long-term PMMA wearers, also reported in soft CL wearers, including SiHy lenses

### Aetiology

- Chronic hypoxia
- Poor lens fit (often seen with high corneal astigmatism)
- Associated with corneal binding

### Symptoms

- Spectacle blur on lens removal
- With long term, may get discomfort, reduced wearing time and develop sudden CL intolerance (corneal exhaustion syndrome)

### Signs

- Distortion of keratometry mires or corneal topography
- Steepering corneal curvature
- Distorted retinoscopy reflex
- Changes in refractive error
- Reduced visual acuity

### Management

- Alleviate hypoxia — refit with higher oxygen performance lens (rigid corneal lens or SiHy)
- Prevent rigid lens bearing — alter lens fit/ design
- Refit with a soft lens
- If occurs with SiHy lenses then refit with lower modulus material
- Temporary cessation of lens wear prior to refit unnecessary
- Ortho-K:
  - Advise instilling aqueous ocular lubricant and gently nudging the lens with the eyelids to ensure the lens is mobile before lens removal
  - If new presentation in an existing Ortho-K wearer consider possible lens spoilage/ damage/warping — consider lens replacement
  - Alter lens design if persistent

### Prognosis

Good if managed — although can take many months

### Differential diagnosis

- Keratoconus
- Intentional corneal molding (orthokeratology)
- Unintentional corneal molding (and subsequent refractive changes with higher modulus SiHys, especially if worn inside out)

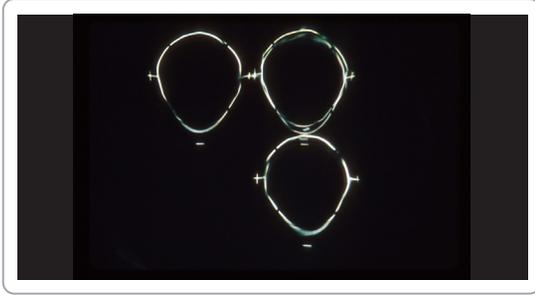


Figure 1: Corneal distortion illustrated by distorted keratometer mires

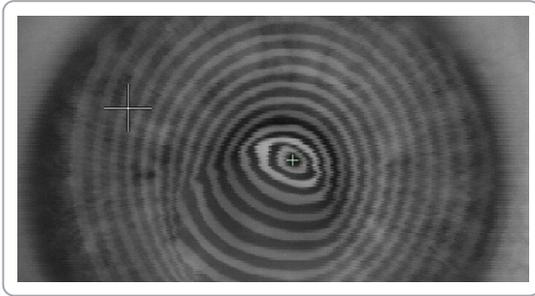


Figure 2: Corneal distortion illustrated by distorted placido disc reflection

### Describing clinical appearance

- 0: None — clear mires
- 1: Slight distortion mires
- 2: Distorted mires
- 3: Very distorted mires
- 4: Extremely distorted mires

238. Asbell P, Wasserman D. Contact lens-induced corneal warpage. *Int Ophthalmol Clin* 1991; 31: 121-6.
239. Calossi A et al. Corneal warpage resolution after refitting an RGP contact lens wearer into hydrophilic high water content material. *CLAO J* 1996; 22: 242-4.
240. Rayess Y et al. Influence of soft contact lens material on corneal warpage: prevalence and time to resolution. *Can J Ophthalmol* 2018; Apr 53(2): 135-138
241. Maeda N et al. Alteration of corneal asphericity in rigid gas permeable contact lens induced warpage. *CLAO J* 1994; 20: 27-31.
242. Rengstorff R. Contact lens induced corneal distortion. In *Anterior Segment Complications of Contact Lens Wear 1994*; Vol 1 (ed Silbert J), pp 351–365 Churchill Livingstone Inc, New York
243. Schornack M. Hydrogel contact lens induced warpage. *Cont Lens Ant Eye* 1993; 26(3): 153-159
244. Wang X et al. Time to resolution of contact lens-induced corneal warpage prior to refractive surgery. *CLAO J* 2002; 28: 169-71.
245. Wilson S et al. Topographic changes in contact lens-induced corneal warpage. *Ophthalmology* 1990; 97: 734-44

# Tear Film

## Tear Film Quality

### Slit lamp viewing

Direct focal illumination, high magnification (25 - 40x), low light intensity

### Assessment of the Tear Film

- General quality, such as frothing, foaming, particles – slit lamp
- Lipid layer presence – Interferometer (automated assessment or subjective grading)
- Tear stability through invasive (Fluorescein) and non-invasive tear film break-up time (NIBUT) – Keratometer, Topographer, Interferometer (automated or observer assessment)
- Tear osmolarity - osmometer, and conducted ahead of any invasive tests

### Aetiology

- Separation of tear film by CL into two layers (pre- and post-lens tear film) leads to thinner lipid layer, increased evaporation and destabilized tear film
- Poor meibum quality and expression
- Incomplete blinking, reduced blink rate affect tear exchange, spreading, drainage, meibum expression, ocular exposure
- Variation in individual tear chemistry (ocular physiology, medication, age, diet)
- CL material, design, wearing schedule, care regimen
- Cosmetics, soap contamination
- Environment (humidity, smoking, pollution)

### Symptoms

- Dryness symptoms including discomfort, grittiness and irritation - assess using questionnaire such as Ocular Surface Disease Index (OSDI), Contact Lens Dry Eye Questionnaires (CLDEQ-8)
- Blurred or variable vision

### Signs

- Low pre-ocular TBUT, poor tear mixing
- Thin or absent tear film lipid layer or excess lipid (debris in tear film),
- Reduced wettability, deposited CL
- Bulbar conjunctival hyperaemia
- Superficial punctate corneal epithelial erosions
- Bulbar conjunctival staining
- Lid margin staining
- High tear osmolarity (>308 mOsmol/L) or interocular difference >8 mOsmol/L

## Management

- Manage all grades if signs or symptoms exist—improve tear film quality
- Treat any lid margin disease – lid hygiene, warm compresses, device-assisted thermal pulsation & expression in office, debridement
- Consider CL type (design, material, modality, replacement frequency and care system)
- Maintain good lens cleaning including rub and rinse step
- Use of lipid containing drops or sprays, visco-elastic agents or overnight lubricants
- Alter diet, Omega-3 oral supplements, alter environment, change cosmetics, blink training
- Consider Ortho-K – circumnavigates the need for CL wear in the open eye and its associated symptoms

## Prognosis

- Good – unless fundamental imbalance of tear film (keratoconjunctivitis sicca)



Figure 1: Tear film lipid layer viewed with EasyTearView+

### Describing clinical appearance

Note: these are descriptions of the images not a grading scale:

- 0: Amorphous/marble
- 1: Polluted tear film
- 2: Lipid
- 3: Excessive lipid, foam in tears
- 4: No lipid layer

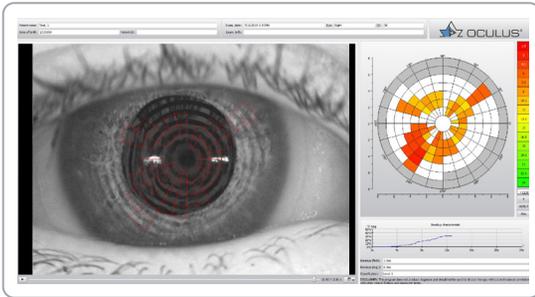


Figure 2: Image of non-invasive tear break up time viewed on Oculus K5

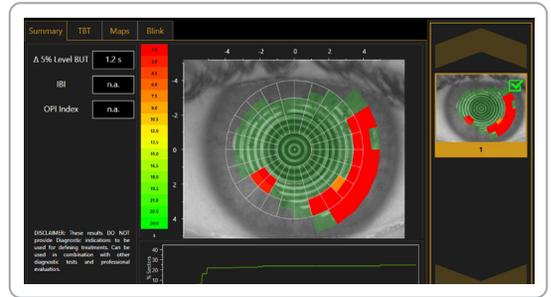


Figure 3: Image of non-invasive tear break up time

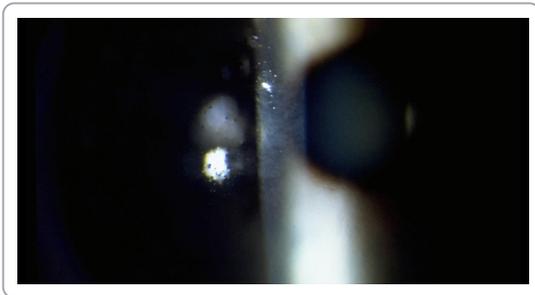


Figure 4: Example of poor tear film quality

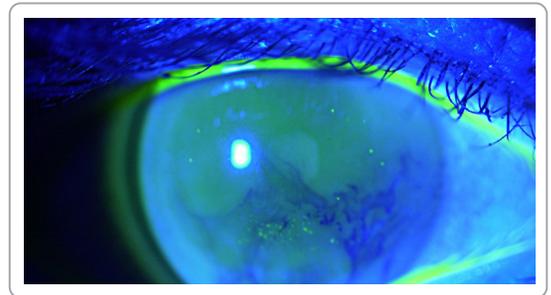


Figure 5: Fluorescein tear film break up

246. Chalmers R et al. Contact Lens Dry Eye Questionnaire-8 (CLDEQ-8) and opinion of contact lens performance. *Optom Vis Sci* 2012; 89(10): 1435-1442

247. Craig J et al. The TFOS International Workshop on Contact Lens Discomfort: Report of the Contact Lens Interactions with the Tear Film Subcommittee. *Invest Ophthalmol Vis Sci* 2013 Oct; 54(11): TFOS123-56

248. Downie L, Craig J. Tear film evaluation and management in soft contact lens wear: a systematic approach. *Clin Exp Optom* 2017 Sep; 100(5): 438-458

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250. Tomlinson A et al. Tear film osmolarity: determination of a referent for dry eye diagnosis. *Invest Ophthalmol Vis Sci* 2006; 47(10): 4309-4315

251. Wang M et al. Impact of blinking on ocular surface and tear film parameters. *Ocul Surf* 2018 Oct; 16(4): 424-429

# Tear Film

## Tear Film Quantity

### Slit lamp viewing

Direct focal illumination, high magnification (40x), narrow slit beam with low intensity to measure (with eye-piece graticule) or grade inferior tear meniscus height (lower lid margin to top of specular reflex) in primary gaze and with normal blinking.

### Assessment of the Tear Film

- Tear volume on eye through non-invasive tear meniscus height – slit lamp with or without graticule, tear interference, optical coherence tomography
- Tear production through invasive tests - Schirmer test, Phenol red thread test

### Aetiology

- Multifactorial, including age, medication, systemic or ocular conditions, environment
- Poor tear film exacerbated by presence of CL

### Symptoms

- Dryness, discomfort, grittiness, irritation, sensitivity to adverse environments- assess using questionnaires such as Ocular Surface Disease Index (OSDI), Contact Lens Dry Eye Questionnaires (CLDEQ-8, CLDEQ-5)
- Blurred or variable vision

### Signs

- Reduced tear meniscus height, irregular tear meniscus (notching or scalloped edge), concave tear profile

- Low Schirmer test scores (at 5 mins, normal >10mm, borderline 5-10mm, severe dry <5mm) or low Phenol red thread test scores (at 15 secs, dry eye <10mm)

### Management

- Address associated systemic or ocular conditions
- Manage any tear quality issues
- Assess prior to lens fit and during CL wear to manage appropriately
- Change lens type (design, material, modality, replacement frequency)
- Maintain good lens cleaning including rub and rinse step
- Change lens care solution
- Rewetting drop/ ocular lubricants
- Attention to nutrition or nutritional supplements (essential fatty acids)
- Environmental changes (humidifier)
- Tear retention measures (to reduce drainage and increase tear contact time) such as punctal plugs or surgery
- Consider Ortho-K – circumnavigates the need for CL wear in the open eye and its associated symptoms

### Prognosis

- Generally good resolution of symptoms with appropriate management unless intractable underlying systemic or ocular condition

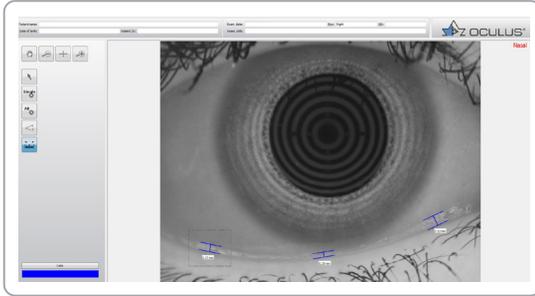


Figure 1: Tear meniscus height measurement on Oculus K5

### Describing clinical appearance

- Low:  $\leq 0.1\text{mm}$  or a difference of at least  $0.06\text{mm}$  between the eyes.
- Medium:  $0.1\text{mm}$  to  $0.25\text{mm}$
- High:  $\geq 0.25\text{mm}$  (indicates reflex tearing and / or deficiency in naso-lacrimal drainage)

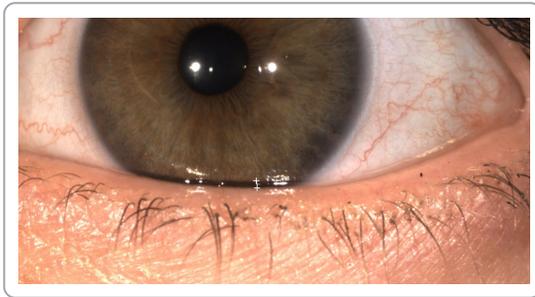


Figure 2 Tear meniscus height with graticule markings visible

252. Bitton E et al. Influence of the blink interval on tear meniscus height in soft contact lens and nonlens wearers. *Eye Contact Lens* 2010; 36: 156-63.
253. Chalmers R et al. Validation of the 5-Item Dry Eye Questionnaire (DEQ-5): Discrimination across self-assessed severity and aqueous tear deficient dry eye diagnoses. *Cont Lens Ant Eye* 2010; Apr 33(2): 55-60
254. Chalmers R et al. Contact Lens Dry Eye Questionnaire-8 (CLDEQ-8) and opinion of contact lens performance. *Optom Vis Sci* 2012; 89(10): 1435-1442
255. Craig J et al. The TFOS International Workshop on Contact Lens Discomfort: Report of the Contact Lens Interactions with the Tear Film Subcommittee. *Invest Ophthalmol Vis Sci* 2013 Oct; 54(11): TFOS123-156
256. Downie L, Craig J. Tear film evaluation and management in soft contact lens wear: a systematic approach. *Clin Exp Optom* 2017 Sep; 100(5): 438-458
257. Guillon J-P. Current clinical techniques to study the tear film and tear secretions. In *The Tear Film: Structure, Function and Clinical Examination*. Butterworth-Heinemann, Oxford, 2002
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259. Nagahara Y et al. Prominent Decrease of Tear Meniscus Height With Contact Lens Wear and Efficacy of Eye Drop Instillation. *Eye Contact Lens* 2015; 41: 318-22.
260. Papas E et al. The TFOS International Workshop on Contact Lens Discomfort: Report of the Management and Therapy Subcommittee. *Invest Ophthalmol Vis Sci* 2013 Oct; 54(11): TFOS183-203
261. Srinivasan S, et al. Apparent time-dependent differences in inferior tear meniscus height in human subjects with mild dry eye symptoms. *Clin Exp Optom* 2007; 90: 345-50.

# Contact Lens Abnormalities

## Reduced Lens Wettability

### Slit lamp viewing

Assessing the wettability by grading the optical quality of Purkinje image (reflection of light from corneal surface: internal and external diffuser on, high magnification (32x), light focused on the front surface of the tear film

### Incidence

- All CLs associated with deposition and disruption to tear film
- Seen more often with longer planned replacement

### Aetiology

- Poor tear film quality/quantity
- Lens material related
- Care regimen related
- External sources of contamination (eg: cosmetics, debris from handling)
- Poorly polished rigid corneal lens

### Symptoms

Reduced CL tolerance, reduced wearing time, dryness, variable vision

### Signs

- Reduced lens wettability
- Low pre-lens BUT <5-10 seconds
- May see lid margin disease, corneal staining, conjunctival injection, mucus and/or increased tear debris

### Management

Determine the cause and select appropriate management:

- Abnormalities on the lid margins or around the eyes (i.e. greasy skin, excessive makeup): educate about lid/skin hygiene and use of cosmetic products with CLs
- Signs and symptoms of DED or MGD: recommend treatment to improve tear film quality (i.e. thermal pulsation, rewetting drops)
- Signs of Incomplete blink patterns or dehydration: teach blink exercises to ensure proper tear film renewal or encourage patients to take break from extended screen time
- Review or switch:
  - Lens cleaning regimen (apply rub and rinse or add surface cleaners),
  - Lens material (Hydrogel to SiHy or vice versa)
  - Lens wear modality and replacement frequency (i.e. switch to DD)
- Repolish or replace rigid corneal lenses or switch lens material
- Advice on proper hand hygiene altering diet, dehydration, environment

### Prognosis

Good with appropriate management

### Differential Diagnosis

Visible deposits

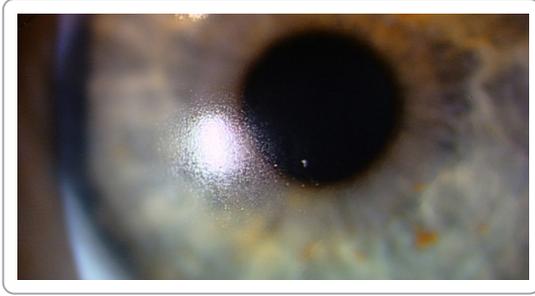


Figure 1: Lens surface drying between blinks

### Describing clinical appearance

- 0: Shiny and clear
- 1: Hydrophobic spots before blink
- 2: Hydrophobic spots just after blink
- 3: Increased area of hydrophobicity
- 4: Total hydrophobic surface

# Contact Lens Abnormalities

## Visible Deposition of Tear Film Components

**Note: ALL contact lens materials interact with, and deposit (adsorb to surface and/or absorb into lens matrix) tear film components (proteins and lipids). This occurs at a molecular level and is not visible on clinical examination. In some circumstances (absorption of active lysozyme to etafilcon A, or lipid to senofilcon A) evidence is available to suggest this can be beneficial to lens comfort.**

Over time, and more commonly seen with longer planned replacement schedules, visible deposits can occur. The clinical assessment and management of those visible deposits related to the tear film is described below, and those related to external sources of contamination in the following section:

### Slit lamp viewing

All types of illumination, low to medium magnification (10 – 16x)

### Incidence

Variable: depends on material type, age of lens, cleaning regimen, tear composition, external sources of deposits

### Aetiology

- Tear film components interact with lens material. Amount of interaction dependent on tear film component charge (positive, negative, no charge) and size, along with material charge (ionicity), pore size and surface treatment
- Build up of visible deposits can be affected by rub and rinse regimen

### Symptoms

- May be none
- Reduced vision and comfort possible with marked deposition
- Ortho-K: Blurred / distorted vision following lens removal after overnight wear

### Signs

- Reduced wetting of lens surface
- Exact appearance depends on type of deposition:
  - Protein: visible on surface when denatured and appears as a film, dries between blinks
  - Lipid: greasy appearance, in discrete spots or spread as a film on lens surface
  - Calculi (Jelly bumps): Raised spots strongly adhered to surface of lens (consist of minerals from tear film surrounded by lipid)
- Ortho-K:
  - Appearance of posterior lens surface deposits that may be more noticeable within the tear reservoir (reverse curve) zone
  - Unexplained loss of refractive effect

## Management

- Identify type of deposition and address
- Improve compliance with rub and rinse regimen
- Change care regimen/add specific cleaner
- Change lens material
- Increase lens replacement frequency, switch to DD if required
- Manage meibomian gland dysfunction if present and source of lipid deposition
- Replace lens if calculi present and switch to more frequent replacement
- Ortho-K: Carefully use cotton bud/Q-tip soaked in daily cleaner to remove persistent deposits within the tear reservoir (reverse curve) zone, refit if signs unresolved

## Prognosis

Good with appropriate management

## Differential Diagnosis

- Poor wetting
- External sources of contamination (see next section) eg: cosmetics

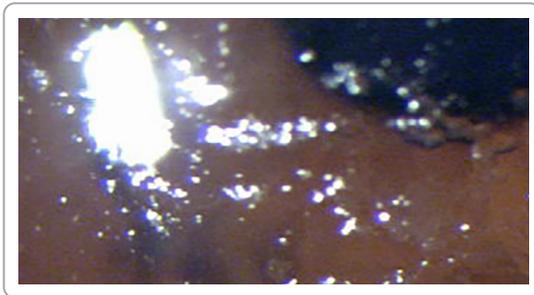


Figure 1: Calculi on lens

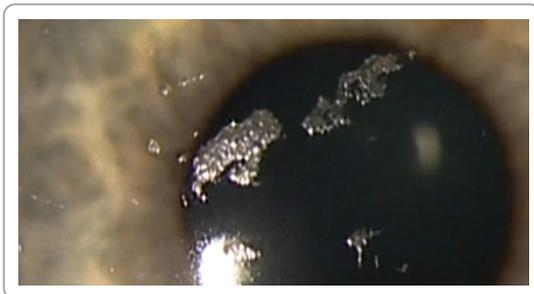


Figure 2: Calculi visible in centre of lens

## Describing clinical appearance

Extent: 1 – 100% coverage of lens and description of appearance

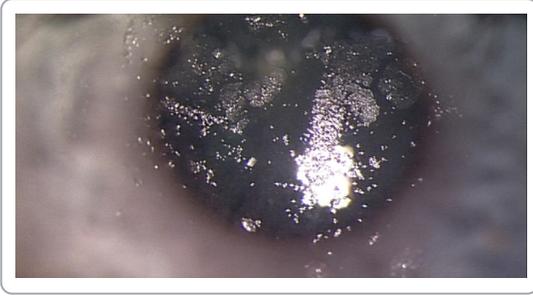


Figure 3: Heavily deposited lens



Figure 4: Deposits visible on a silicone hydrogel contact lens

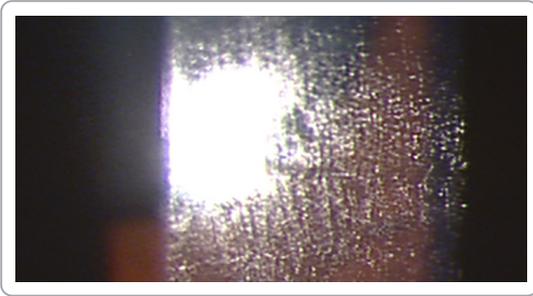


Figure 5: Lipid film on lens surface

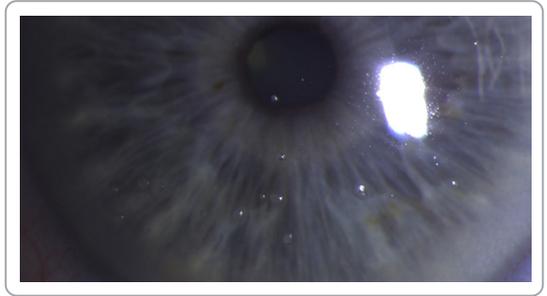


Figure 6: Discrete lipid type deposits on lens surface



Figure 7: Calculi (Jelly bumps)



Figure 8: Protein film

268. Lorentz H, Jones L. Lipid deposition on hydrogel contact lenses: how history can help us today. *Optom Vis Sci* 2007; 84(4): 286-295
269. Luensmann D, Jones L. Protein deposition on contact lenses: the past, the present, and the future. *Cont Lens Ant Eye* 2012; 35(2): 53-64
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275. Willcox M. Solutions for care of silicone hydrogel lenses. *Eye Contact Lens* 2013; 39(1): 24-28

# Contact Lens Abnormalities

## Visible Lens Spoilation From External Sources

### Slit lamp viewing

All types of illumination, low to medium magnification (10 – 16x)

### Incidence

Variable, patient and environment dependent

### Aetiology

External source of deposition or other lens spoliation

### Symptoms

- Many be none
- Reduced vision and comfort possible

### Signs

- Dependent on cause, for example:
- Cosmetics: shiny, greasy appearance
- Fungal: filamentary growth visible on and within the lens
- Rust: discrete orange-brown spots

### Management

- Identify cause
- Educate on rub and rinse requirement
- Replace frequent planned replacement lenses with fresh lens
- Review hygiene practices and hand-to-lens contamination
- Increase lens replacement frequency, refit with DD if required

### Prognosis

Good with cause addressed and managed

### Differential diagnosis

Other lens deposition originating from tear film



Figure 1: Low magnification view of make up on lens

### Describing clinical appearance

Extent: 1-100% coverage of lens and description



Figure 2: Higher magnification view of mascara on lens



Figure 3: Make up on lens

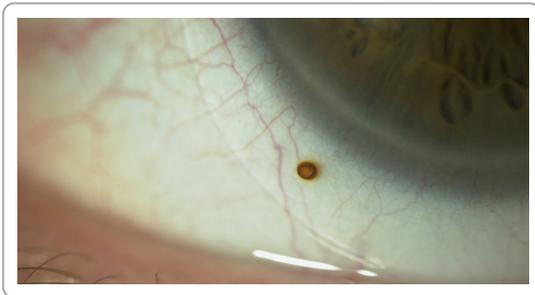


Figure 4: Rust spot

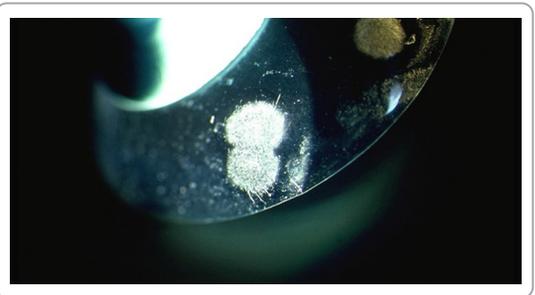


Figure 5: Fungal deposit

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# Contact Lens Abnormalities

## Lens Damage

### Slit lamp viewing

All types of illumination, low to medium magnification (10 – 16x)

### Incidence

Unknown — can occur in all types — may be higher with new wearers and hyperopes

### Aetiology

- May be related to poor lens handling
- CL might be trapped in lens case
- Ortho-K: lens warping

### Symptoms

- Often none or minor irritation and foreign body sensation which resolves on lens removal
  - Ortho-K: Blurred / distorted vision on removal of lens after overnight wear

### Signs

- Rigid corneal lens (RCL) — scratched, section missing, broken
- Soft — torn, segment missing, hole
- May see corneal or conjunctival staining adjacent to damaged area of lens
- Ortho-K: Unexplained loss of refractive effect

### Management

- Replace lens
- Re-evaluate handling procedures if damage due to poor handling
- Consider different material type and replacement frequency if reoccurs
- Ortho-K: replace lens

### Prognosis

Excellent

### Differential diagnosis

None

## Describing clinical appearance

Extent: 1-100% coverage of lens and description



Figure 1: Nick in soft lens edge

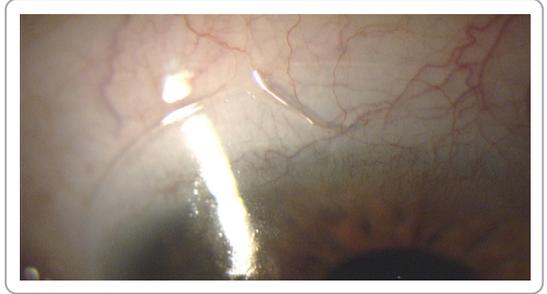


Figure 2: Tear in soft lens edge



Figure 3: Torn soft lens edge

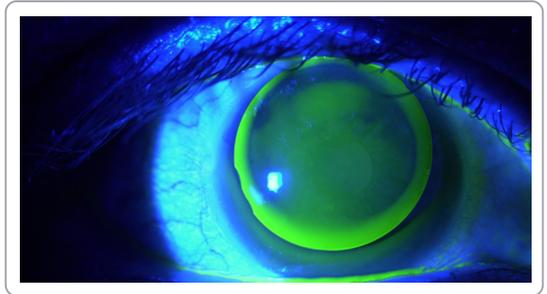


Figure 4: Chipped rigid corneal lens edge

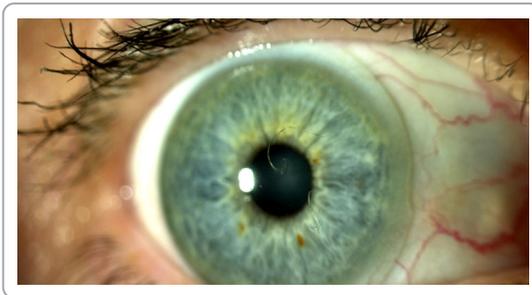


Figure 5: Crack in soft lens



Figure 6: Scratched rigid corneal lens

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