J&J
Institute

A Handbook of Contact Lens Management

4TH EDITION

Vision Made Possible™

Editors, advisory panel and contributing authors (editions 1-3, 1996-2011)

Jørgen Schou Anderson, Gudrun Bischoff, Jørgen Bruun-Jensen, Ian Davies, Alison Ewbank, Javier Gonzalez Cavada, Lyndon Jones, Jonathan Kersley, Allan Kruse, Tom Løfstrøm, Gerald Lowther, George Mertz, John Meyler, Heiko Pult, Lene Ringmann, Anna Sulley, Jane Veys.

Content editor's (edition 4, 2021):

Global Professional Education & Development, Johnson & Johnson Vision Care

Centre for Ocular Research & Education (CORE), University of Waterloo, Canada,

The editors of this resource have taken care to ensure that the recommendations and advice given are correct and compatible with the clinical information available at the time of publication. As new information becomes available, changes in treatment or advice may become necessary.

The guidelines are provided for educational purposes only. The final clinical judgement and management approach should be determined by the qualified eye care professional. Johnson & Johnson Vision Care or Centre for Ocular Education & Research (CORE) do not engage in the practice of prescribing or patient follow-up. This resource is not a substitute for appropriate education and training or for the exercise of independent professional judgement,

Content contributors' (edition 4, 2021)

Kristy Canavan, John Meyler, Karen Walsh, Lyndon Jones, Doerte Luensmann, Amir Moezzi, Marc Schulze, Ulli Stahl, Hendrik Walther, Jill Woods, and Mike Yang CORE, Canada, and Paul Gifford, Myopia Profile, Australia

Photography and videos

Kathy Dumbleton, Trusit Dave, Xanvier Dezard, Carsten Edmond, Andrew Gasson, John Hermen, Sheila Hickson-Curran, Saleel Jivraj, Richard Lindsay, Carole Maldonado Codina, Keyur Patel, Rachael Peterson, Heiko Pult, David Ruston, Cristina Schnider, Stroee Stenerson, Brian Tompkins, James Wolffsohn

The Johnson & Johnson Institute®

Acknowledgement

Two images are used with kind permission from Andrew Gasson and Judith Morris from the book: The Contact Lens Manual, 3rd edition; Butterworth-Heinemann, 2005

First published 1996 (Reprinted 2002). Second edition 2006 (Reprinted 2009). Third edition 2011. Fourth edition 2021.

Originally developed by Synoptik and Johnson and Johnson Vision Care, part of Johnson and Johnson Medical Ltd. Original Authors (1st edition): Jørgen Schou Anderson, Aut CL Optometrist. Ian Davies, MCOptom, DipCL, FAAO. Allan Kruse, Aut CL Optometrist. Tom Løfstrøm, MSc, Optom. Lene Ringmann, Aut CL Optometrist

A Handbook of Contact Lens Management

4TH EDITION

Contents

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[1]), 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 forth 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

1. Wolffsohn et al. Contact Lens Evidence-based Academic Reports (CLEAR). Cont Lens Ant Eye 2021; 12(4): 22-34

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.

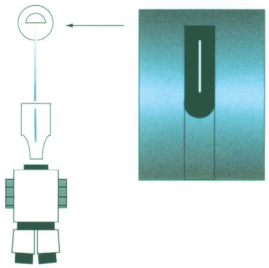


Fig 1

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)*.

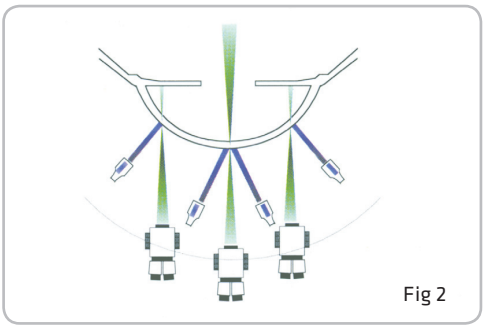


Fig 2

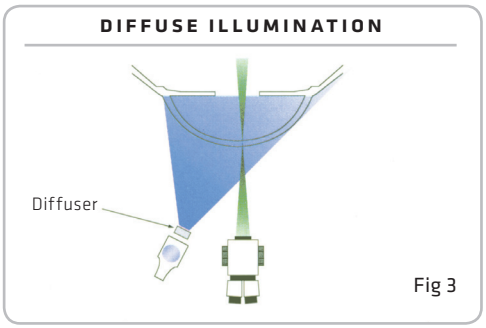


Fig 3

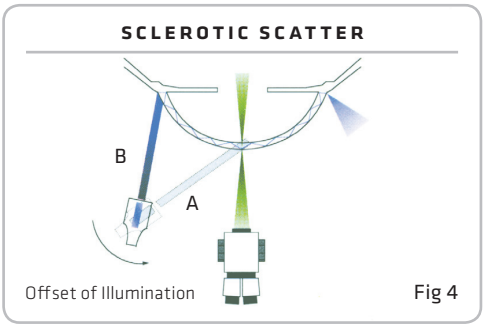


Fig 4

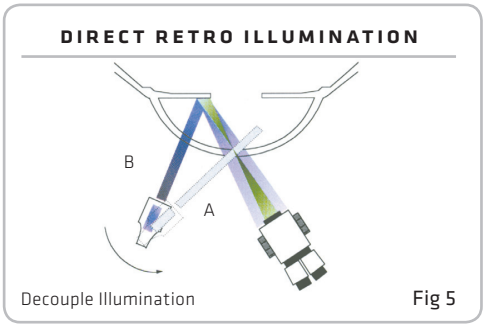


Fig 5

(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.

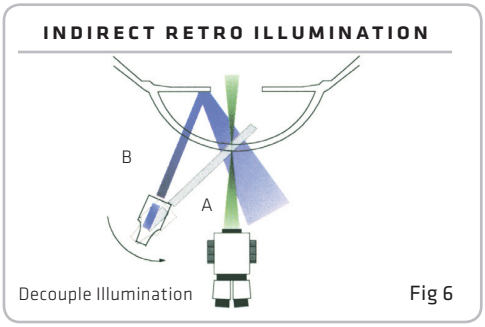
(iii) Focusing check

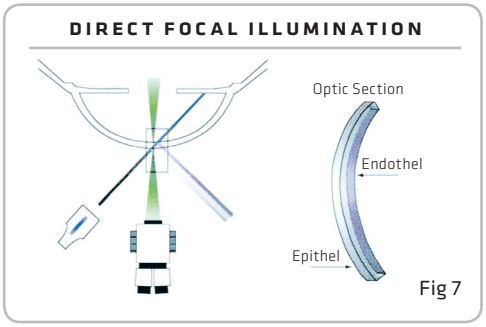
With the eyelids closed, the examiner should now focus the light on the lids and check its focus by rotating the illumination system round from side to side. As it rotates, the light should remain stationary on the lid. If it is showing relative movement, then the instrument is not in focus. With the 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.

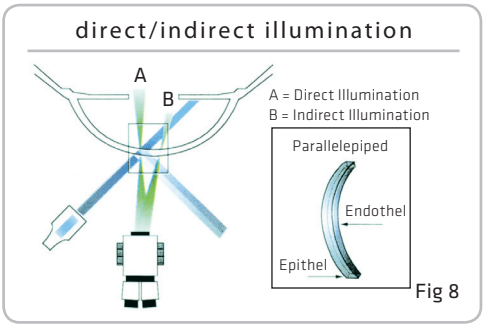
Slit Lamp Routine

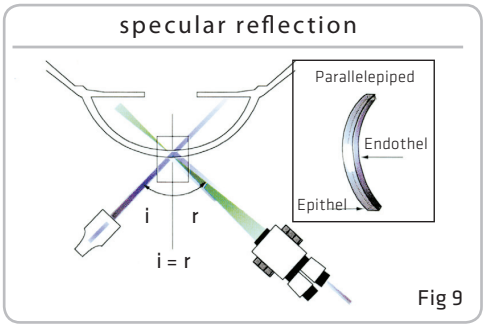
(i) Overall view: low magnification, wide diffuse beam *(Figure 3)*.

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)*, 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)*. 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)*, 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

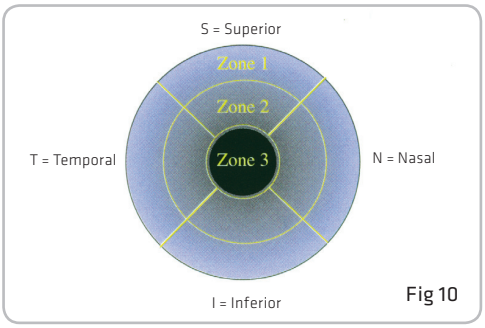


Fig 10

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)*. 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.

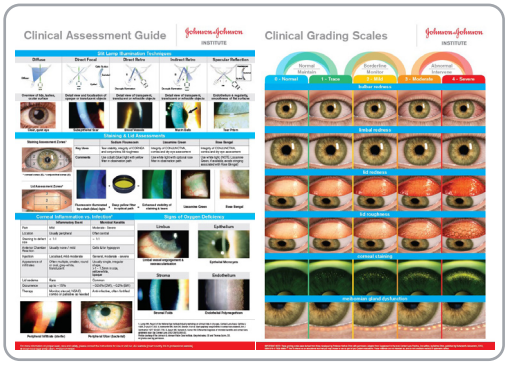
Recording The Results

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 been 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 VISION CARE 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)

Further Reading

2,3

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
- Seborrheic 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 sebhorrheal or as local immune reaction
- Seborrheic (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 ≥ 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

Further Reading

4, 5, 6, 7, 8, 9, 10, 11, 12

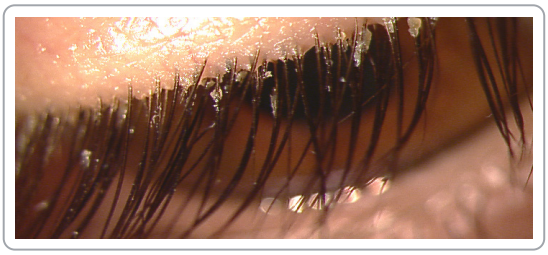


Figure 1: Primarily Staphylococcal Blepharitis, with evidence of some Demodex collarettes too

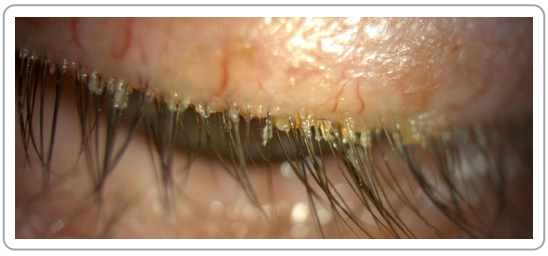


Figure 3: Cylindrical dandruff/collarettes of Demodex Blepharitis

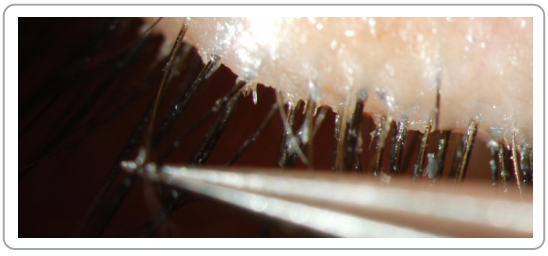


Figure 2: Exposed Demodex tail visible in centre of image

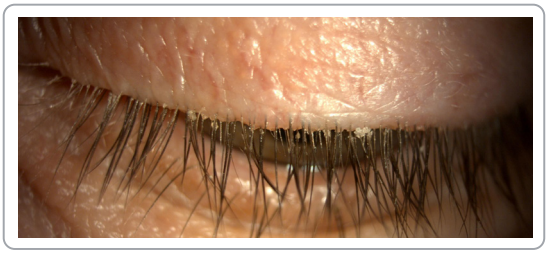


Figure 4: Seborrheic Blepharitis

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

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

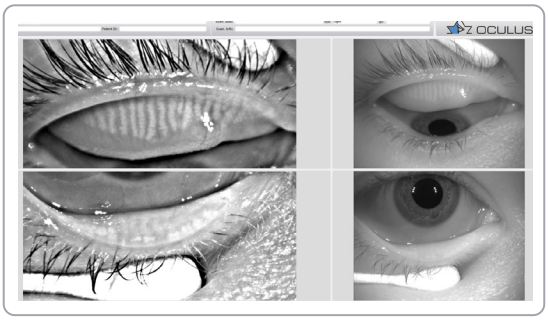


Figure 1: Meibography example showing gland drop out, more severe in lower than upper lid

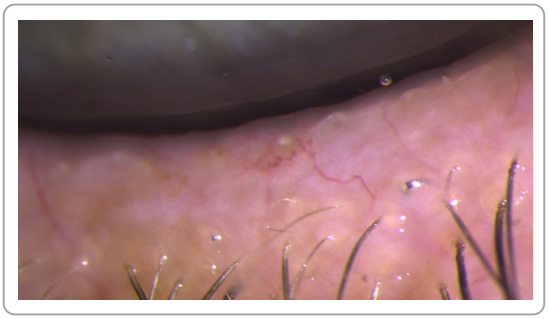


Figure 2: High magnification view of telangiectasia, capped meibomian glands and irregular lid margin

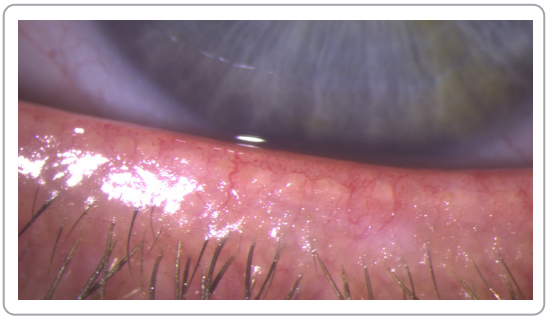


Figure 3: Telangiectasia, capped meibomian glands

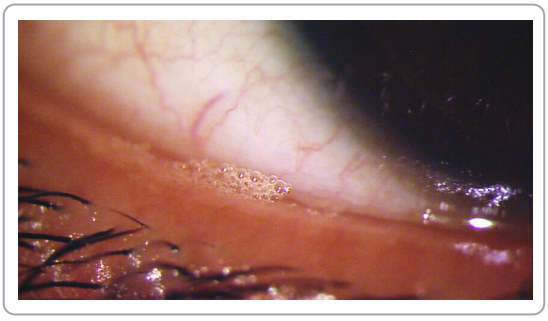


Figure 4: Example of frothy tears

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

Further Reading

13, 14, 15, 16, 17, 18, 19, 20, 21, 22

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% ≥ 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 (LIPCOF), 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 ≥ 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

Further Reading

23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37

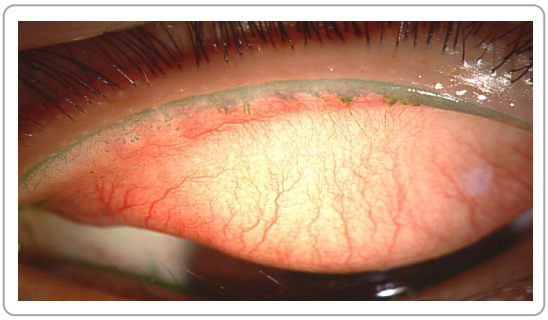


Figure 1: Upper lid wiper epitheliopathy with lissamine green

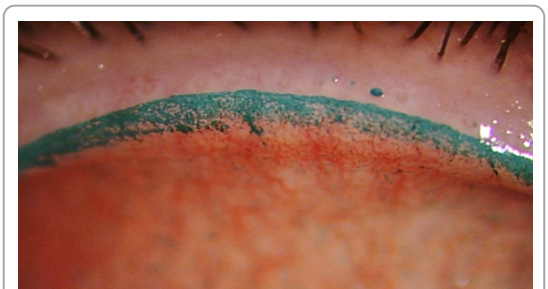


Figure 2: Higher magnification view of upper lid wiper epitheliopathy with lissamine green

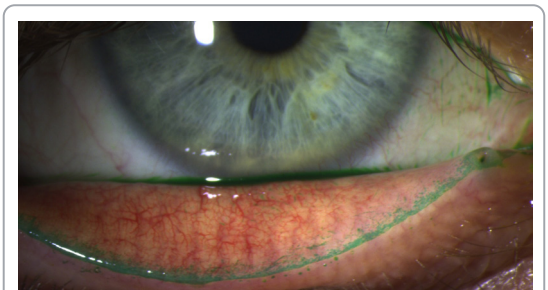


Figure 3: Lower lid wiper epitheliopathy with lissamine green

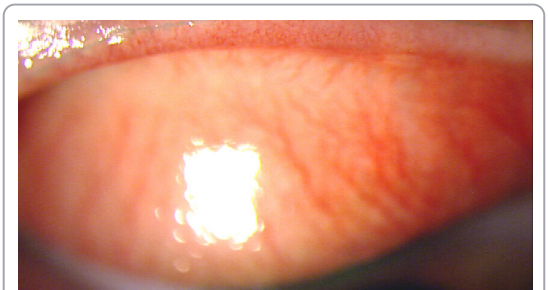


Figure 4: Differential diagnosis: Marx's line

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

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 contra-indication 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

Further Reading

38, 39, 40, 41

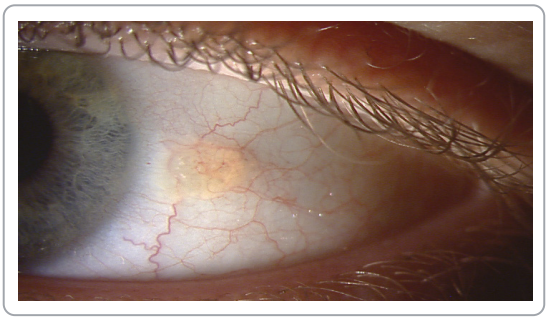


Figure 1: Prominent temporal Pinguecula

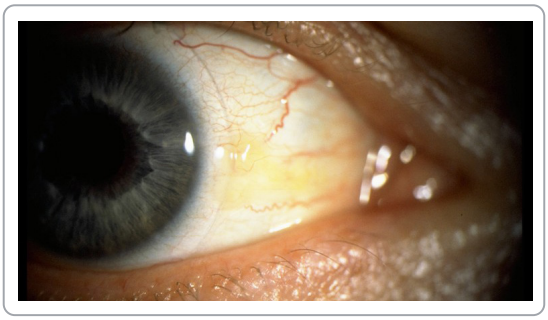


Figure 2: Pinguecula interacting with soft contact lens edge

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

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 contra-indication 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)

Further Reading

42, 43, 44, 45, 46, 47, 48, 49

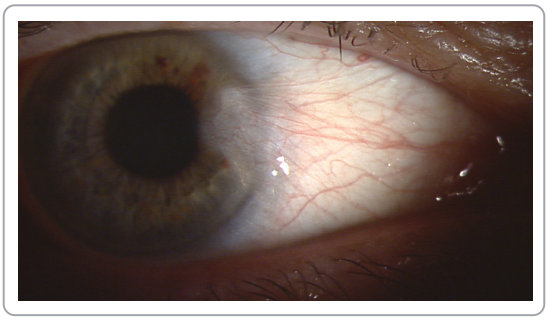


Figure 1: Nasal pterygia extending to pupil margin

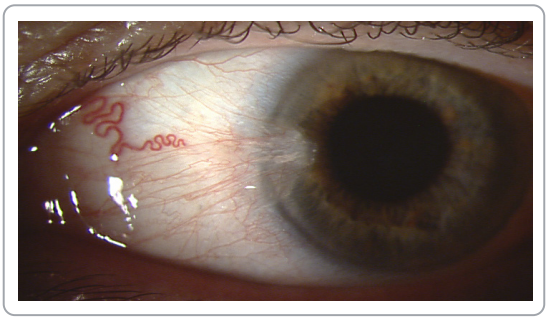


Figure 2: Medium magnification view of nasal pterygia

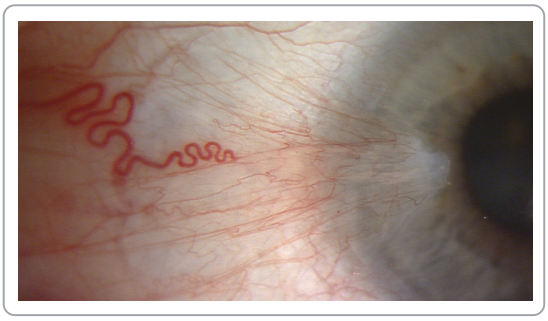


Figure 3: Nasal pterygia viewed under higher magnification

Describing clinical appearance

Position: nasal/temporal and extent of encroachment onto cornea

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 ≥ grade 2 or ≥ 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)

Further Reading

50, 51, 53, 54, 56, 285

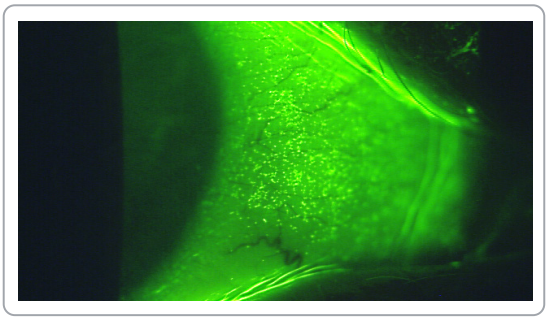


Figure 1: Punctate staining of conjunctiva viewed under blue light and Wratten filter

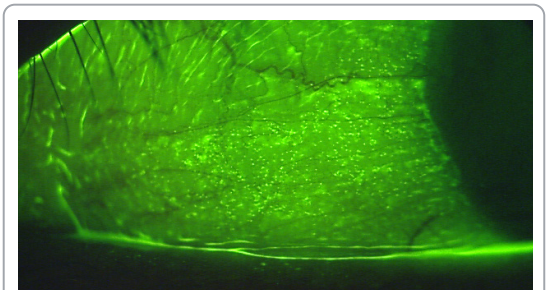


Figure 2: Punctate staining of conjunctiva viewed under blue light and Wratten filter

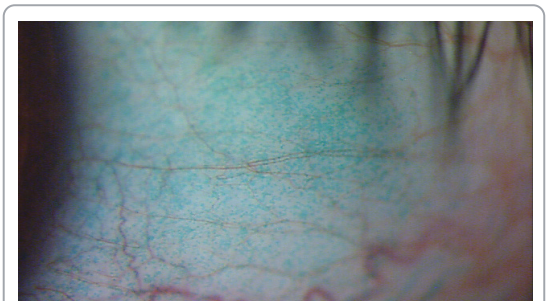


Figure 3: Punctate lissamine green staining of conjunctiva viewed with white light

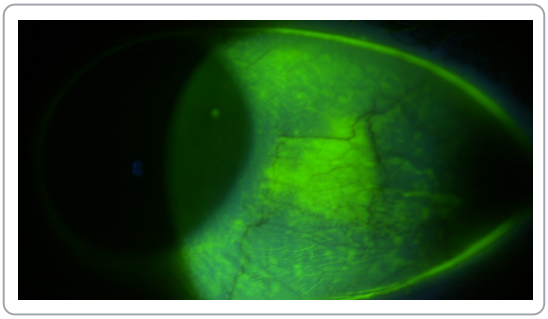


Figure 4: Confluent conjunctival stain

Describing clinical appearance

Position: Superior, nasal, inferior, temporal

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

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 ≥ grade 2 or ≥ 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)

Further Reading

50, 51, 52, 53, 54, 55, 56, 57

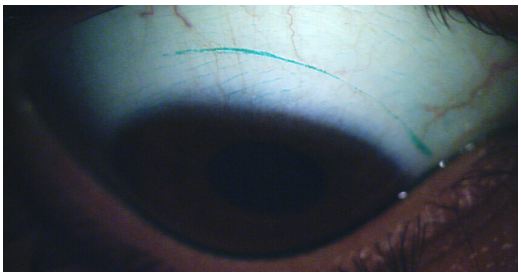


Figure 1: Superior lens edge lissamine green stain viewed under white light

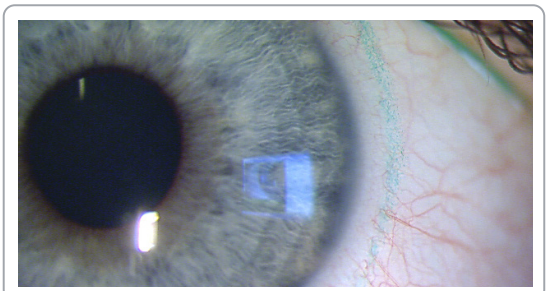


Figure 2: Temporal lens edge lissamine green stain viewed under white light

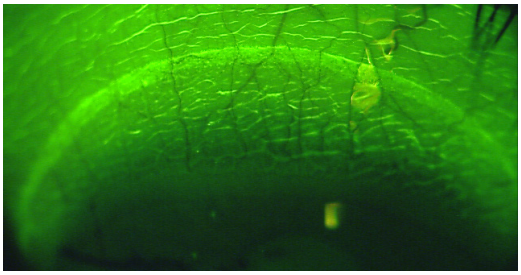


Figure 3: Superior lens edge stain viewed with fluorescein and blue light/yellow Wratten filter

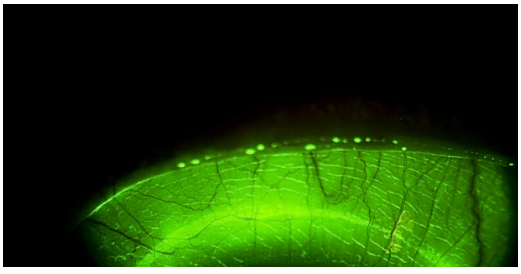


Figure 4: Superior lens edge stain viewed with fluorescein and blue light/yellow Wratten filter

Describing clinical appearance

Position: Superior, nasal, inferior, temporal

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

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

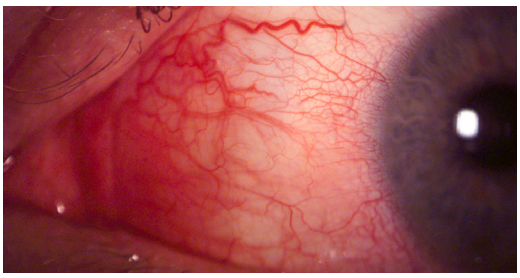


Figure 1: Acute allergic conjunctivitis with chemosis

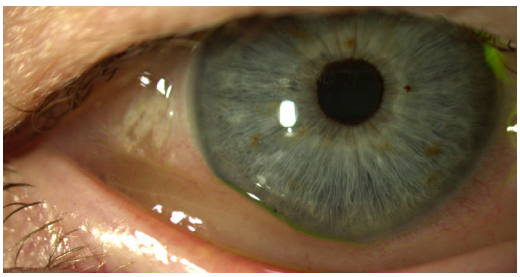


Figure 2: Acute conjunctival chemosis

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 ≥ 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 ≥ grade 2 or if ≥ 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

Further Reading

58, 59, 60, 61, 62, 63, 64

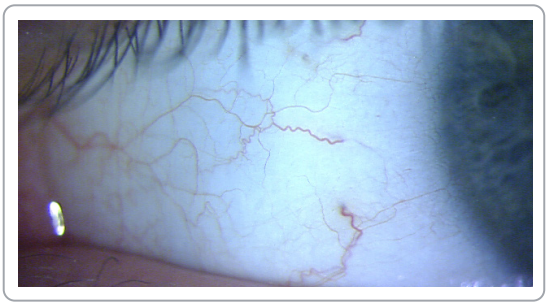


Figure 1: Example of low level conjunctival hyperaemia (actual grade dependent on grading scale used)

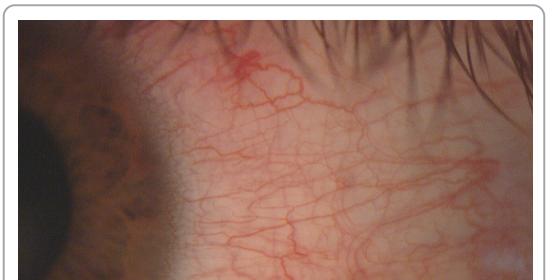


Figure 2: Magnified view of conjunctival hyperaemia

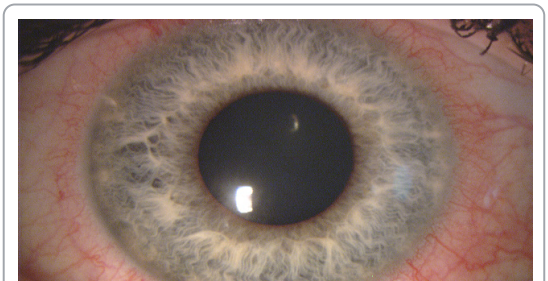


Figure 3: Low magnification diffuse light view of conjunctival hyperaemia

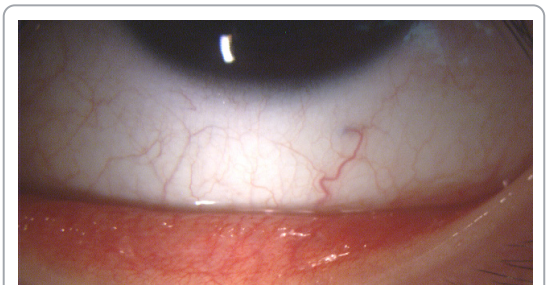


Figure 4: Example of inferior conjunctival hyperaemia with lower lid retracted

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

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 ≥ 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

Further Reading

65, 66, 67, 68, 69, 70, 71

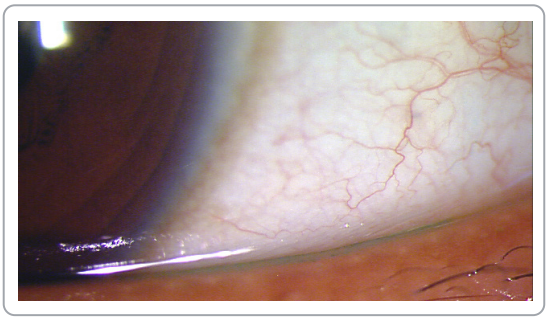


Figure 1: Example of LIPCOF

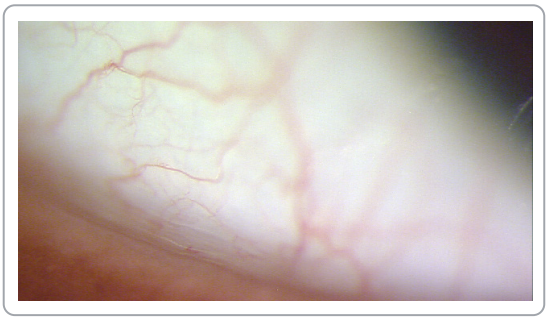


Figure 2: Higher magnification example of LIPCOF

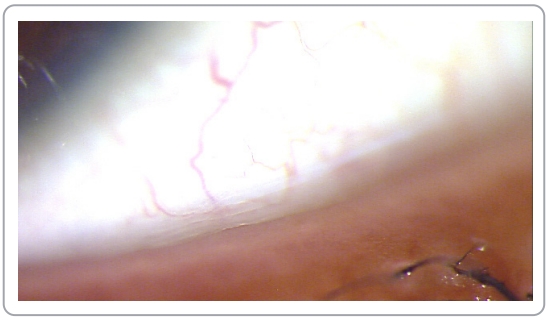


Figure 3: Higher magnification example of LIPCOF

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)

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 ≥ grade 2 or ≥ 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

Further Reading

72, 73, 74, 75, 76, 77, 78, 79

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

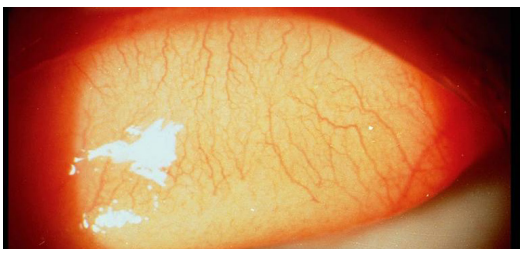


Figure 1: Low grade hyperaemia and papillae

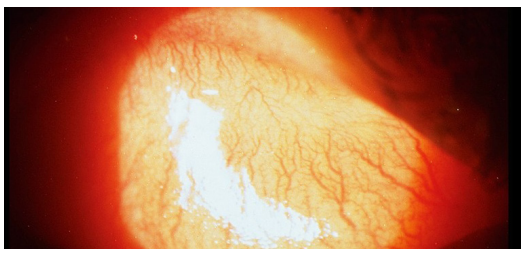


Figure 2: Light reflection showing small papillae in centre of tarsal conjunctiva

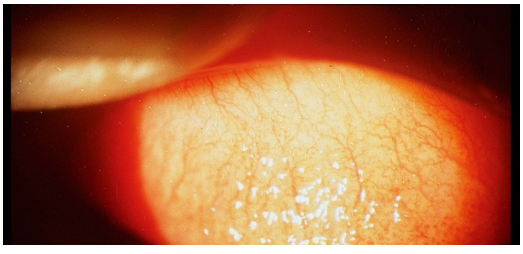


Figure 3: Visible moderate level of papillae

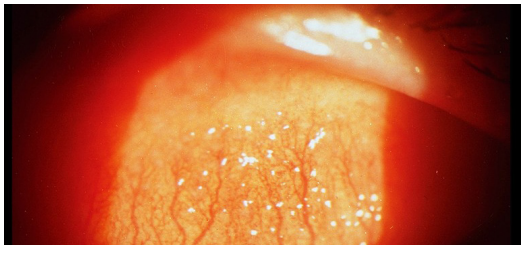


Figure 4: Moderate papillae and increased hyperaemia

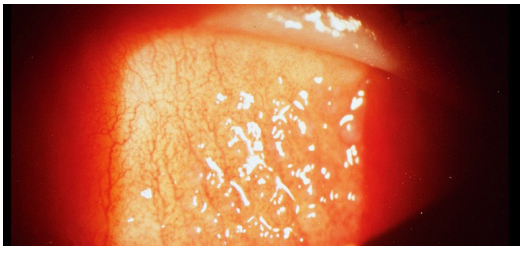


Figure 5: Marked giant papillae and hyperaemia

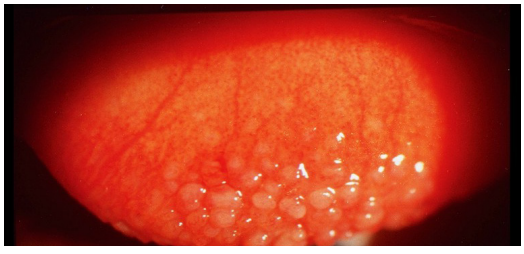


Figure 6: Marked giant papillae and hyperaemia

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 ≥ grade 2 or if ≥ 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

Further Reading

80, 81, 82, 83, 84, 85

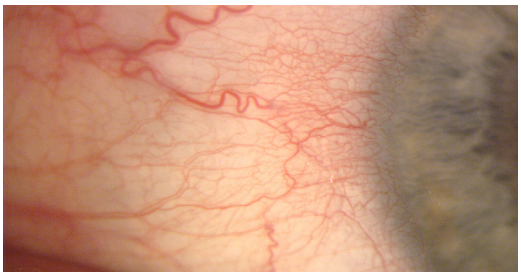


Figure 1: Example of limbal hyperaemia

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

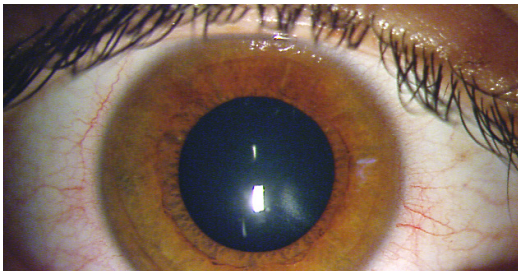


Figure 2: Low magnification, diffuse illumination view of limbal hyperaemia

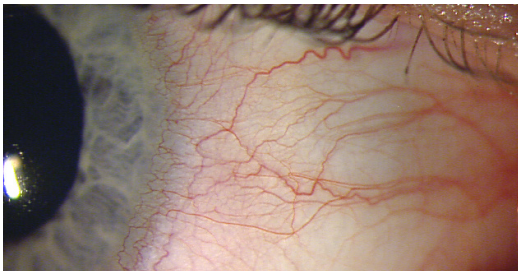


Figure 3: Example of limbal hyperaemia

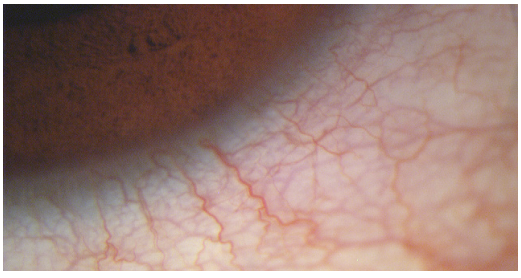


Figure 4: Higher magnification view of limbal hyperaemia

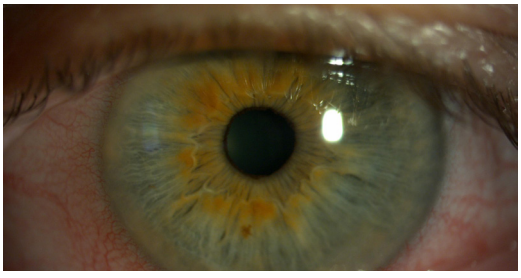


Figure 5: Differential diagnosis: limbal and conjunctival hyperaemia secondary to corneal foreign body

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 ≥ grade 2 or if ≥ 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

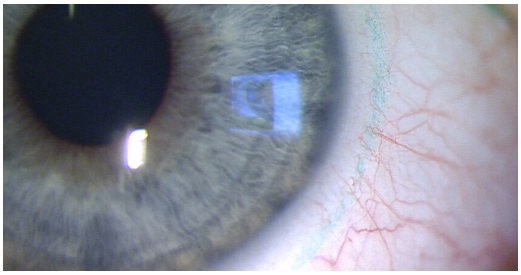


Figure 1: Lissamine green limbal stain from soft lens edge

Describing clinical appearance

Position: Superior, nasal, inferior, temporal

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

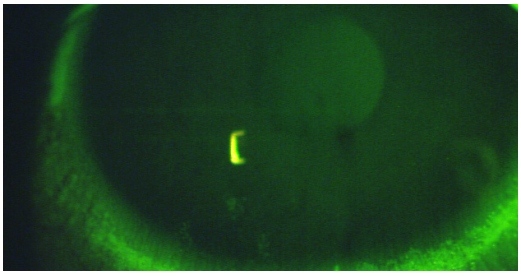


Figure 2: Wide band of fluorescein limbal stain resulting from soft lens edge

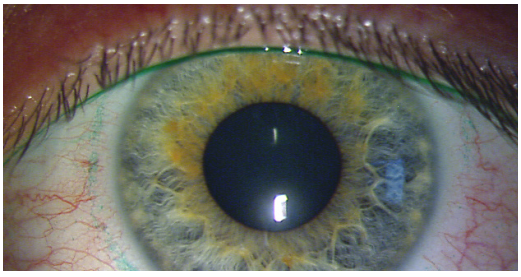


Figure 3: Low magnification view of circum-limbal lissamine green staining

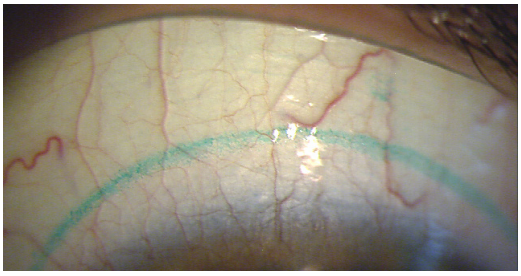


Figure 4: View of superior limbal lissamine green stain

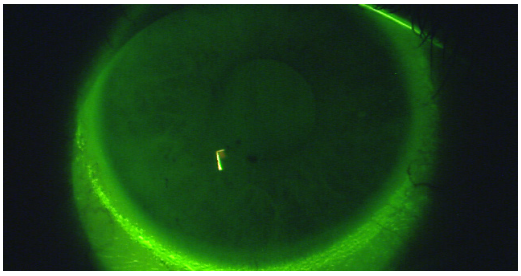


Figure 5: Fluorescein limbal stain viewed with blue light and Wratten filter

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

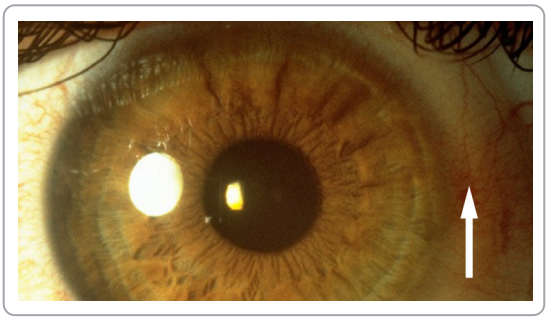


Figure 1: Limbal oedema viewed under diffuse white light

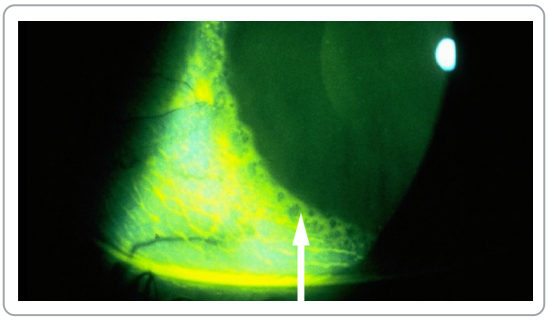


Figure 2: Limbal oedema viewed with fluorescein and blue light

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

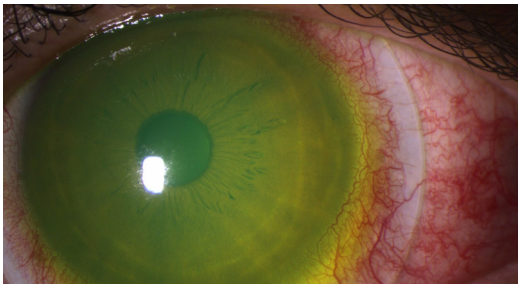


Figure 1: Example of limbal bearing with a scleral lens in a very hyperemic eye

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

Further Reading

80, 81, 82, 83, 84, 85, 86, 87, 88, 89

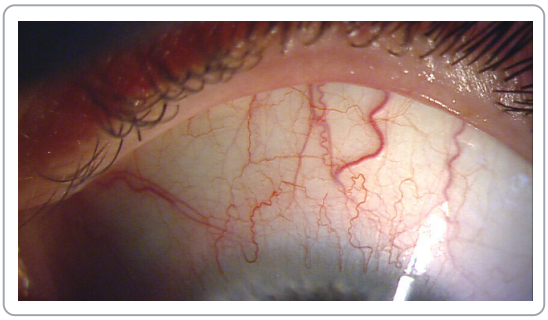


Figure 1: Example of superior limbic keratoconjunctivitis

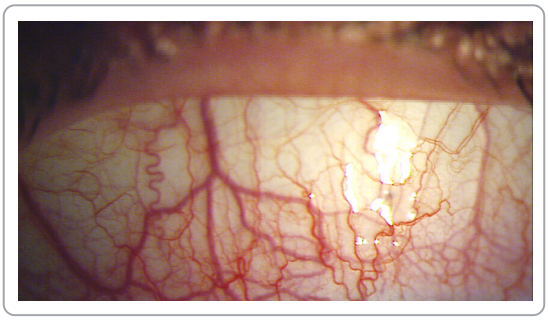


Figure 2: Higher magnification view of superior limbic keratoconjunctivitis

Describing clinical appearance

Record position, extent and level of associated hyperaemia

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 ≥ 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)

Further Reading

90, 91, 92, 93, 94, 95, 96, 97, 98, 99

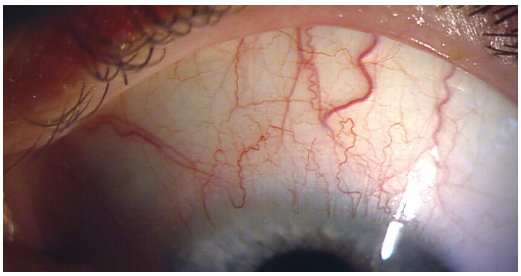


Figure 1: Example of neovascularisation viewed with direct illumination

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

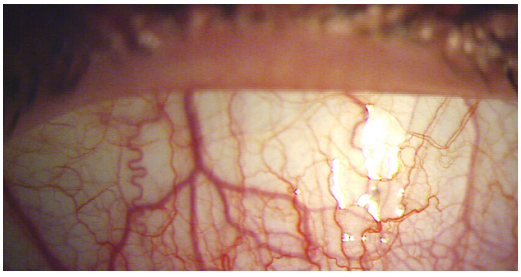


Figure 2: Neovascular changes in the superior limbal area

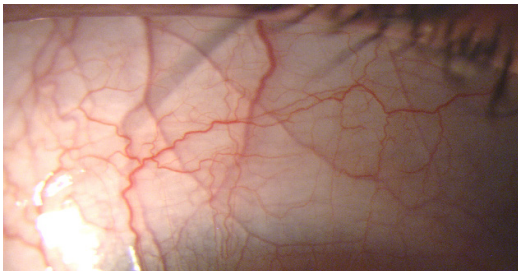


Figure 3: Superior neovascularisation

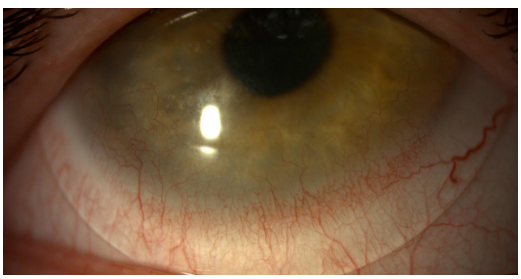


Figure 4: Extensive corneal vascularization visible under a scleral lens

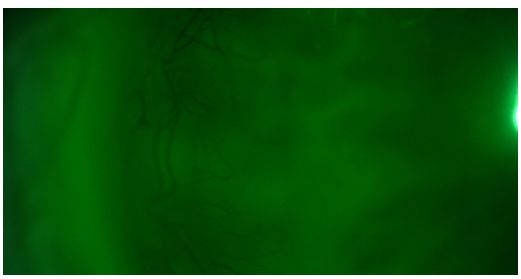


Figure 5: Extensive vascularisation visible with red-free filter (fluorescein also present in image)

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 ≥ Grade 2 or if ≥ 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)

Further Reading

100, 101, 102, 103, 104, 105, 106, 107, 108, 109

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

Position: Superior, nasal, inferior, temporal, central

Depth

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

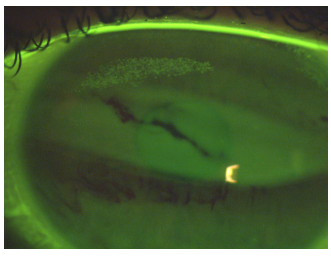


Figure 1: Example of punctate stain on superior cornea

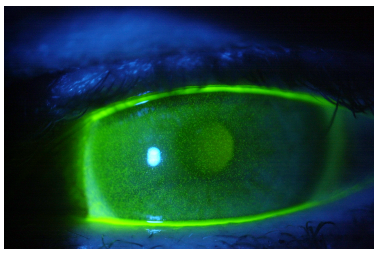


Figure 2: Extensive punctate stain

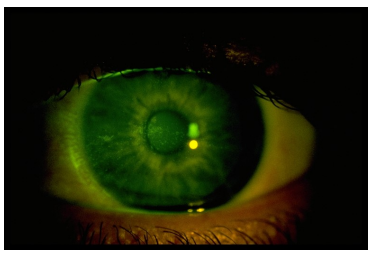


Figure 3: Central corneal stain

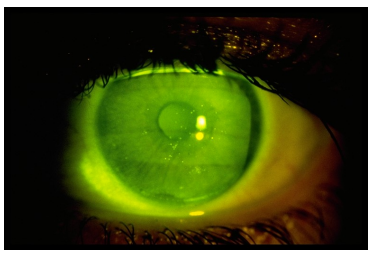


Figure 4: Discrete punctate corneal stain

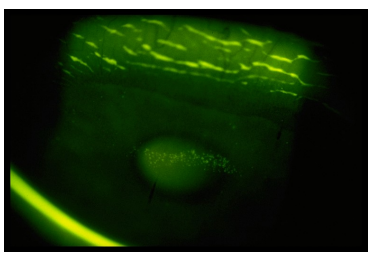


Figure 5: Superior punctate stain

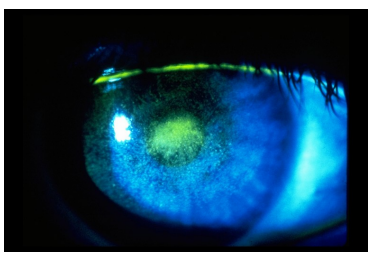


Figure 6: Confluent central corneal stain

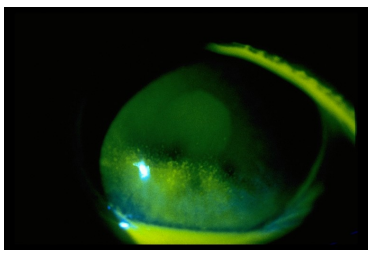


Figure 7: Inferior punctate and confluent stain

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 ≥ grade 2 or if ≥ 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

Further Reading

110, 111, 112, 113, 114

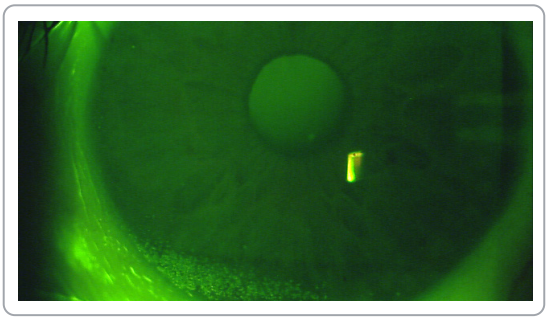


Figure 1: Example of LIPCOF

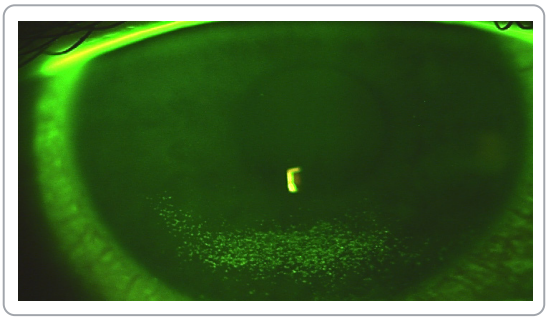


Figure 2: Higher magnification example of LIPCOF

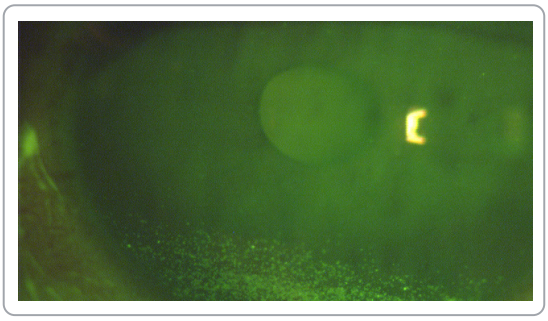


Figure 3: Higher magnification example of LIPCOF

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

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 ≥ grade 2 or if ≥ 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

Further Reading

115

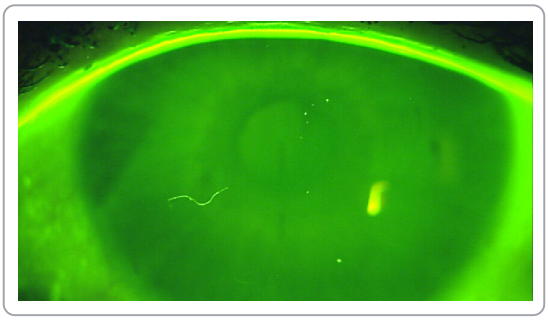


Figure 1: Example of staining from hair fibre trapped under lens

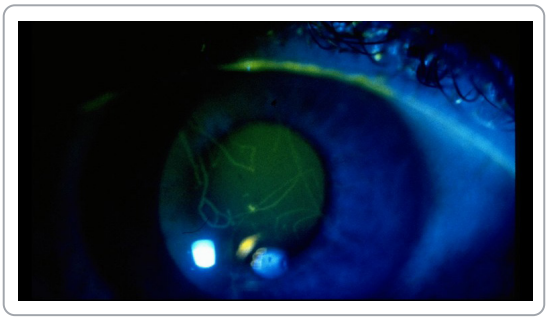


Figure 2: Foreign body track staining

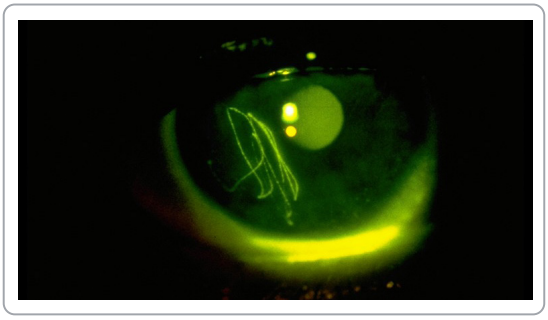


Figure 3: Foreign body track staining

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 ≥ grade 2 or if ≥ 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

Further Reading

116, 117, 118, 119, 120, 121, 122, 123

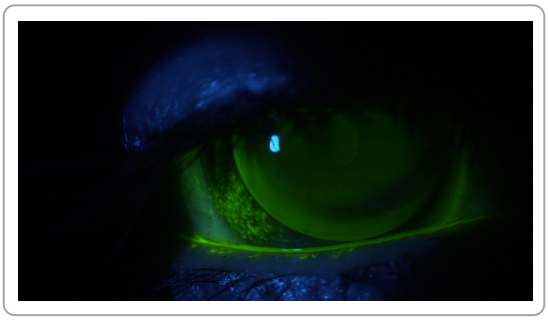


Figure 1: 3 & 9 staining viewed with blue light and yellow filter; rigid contact lens in place

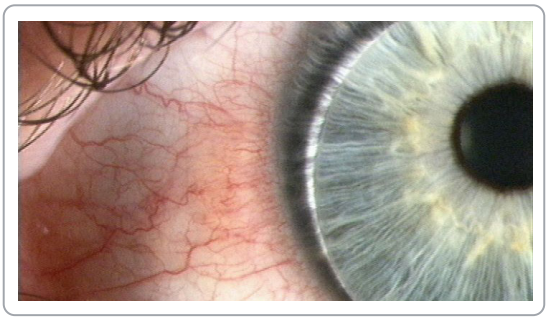


Figure 2: Associated bulbar conjunctival hyperaemia along the horizontal meridian

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

Cornea

Solution Induced Corneal Staining (SICs)

Slit lamp viewing

Direct illumination parallelepiped 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 Tetronic 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)

Further Reading

124, 125, 126, 127, 128, 129, 130, 131, 132, 133

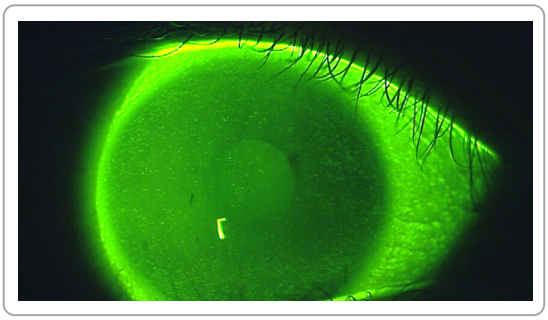


Figure 1: Example of SICS

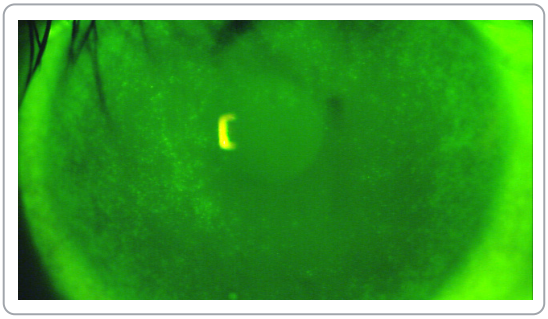


Figure 2 Example of SICS

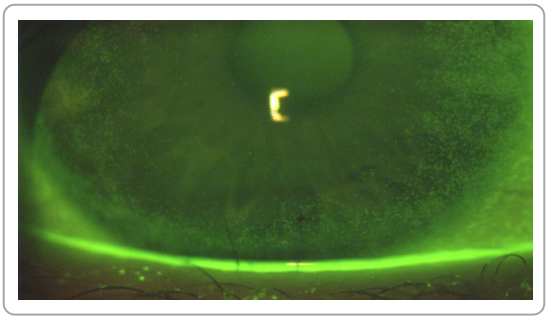


Figure 3: Example of SICs donut staining pattern

Describing clinical appearance

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

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 ≥ grade 2 or if ≥ 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)

Further Reading

134, 135, 136, 137, 138, 139, 140

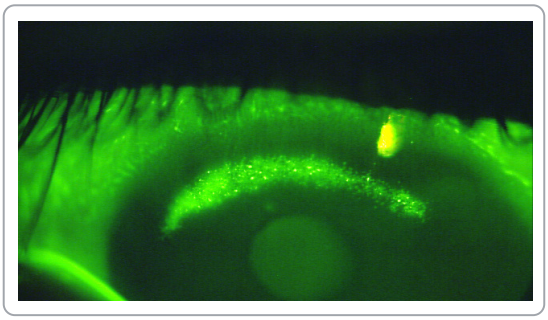


Figure 1: Superior epithelial arcuate lesion (SEAL) viewed with fluorescein

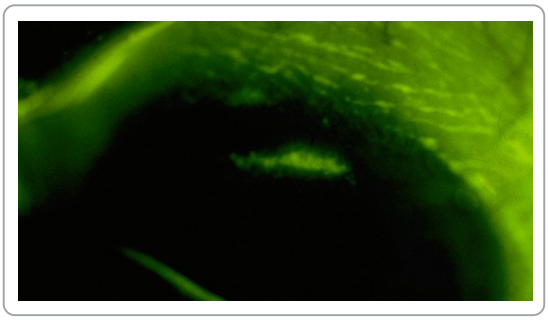


Figure 2: Superior epithelial arcuate lesion (SEAL) viewed with fluorescein

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

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

Further Reading

141, 142, 143, 144, 145, 146, 147, 148

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

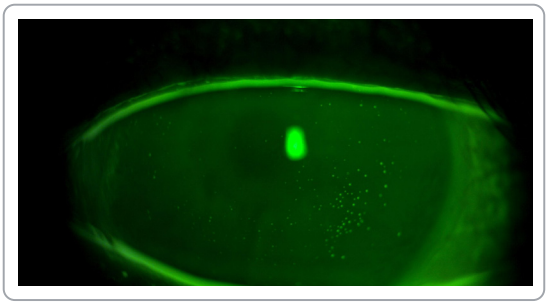


Figure 2: Mucin ball indents filled with fluorescein following lens removal

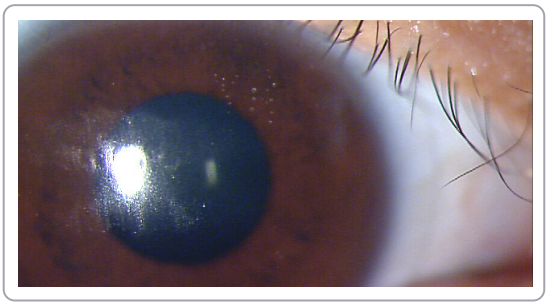


Figure 3: Mucin balls visible under soft lens

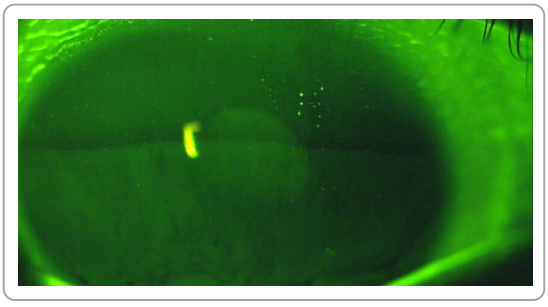


Figure 4: Mucin ball indents filled with fluorescein following lens removal (same eye as Figure 3)

Describing clinical appearance

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

Cornea

Dimple Veil

Slit lamp viewing

Direct illumination parallelepiped, 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 ≥ grade 2 or if ≥ 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

Further Reading

149, 150, 151, 152, 153

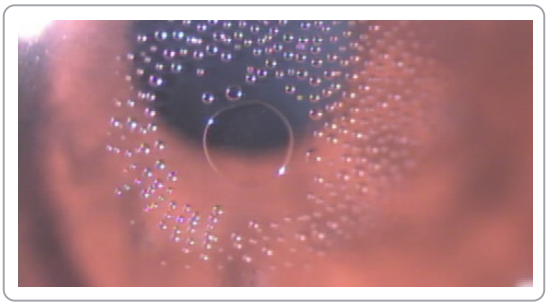


Figure 1: Dimple veil visible with diffuse white light view

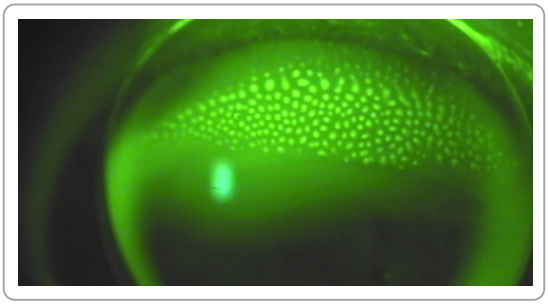


Figure 2: Dimple veil visible with fluorescein and blue light illumination

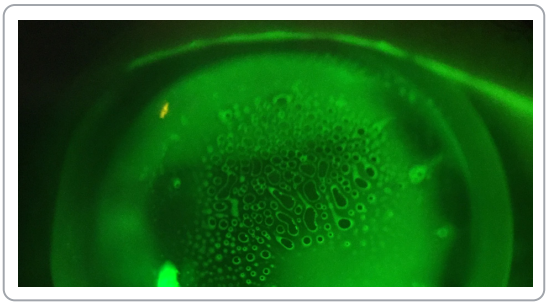


Figure 3: Dimple veil visible with fluorescein and blue light illumination

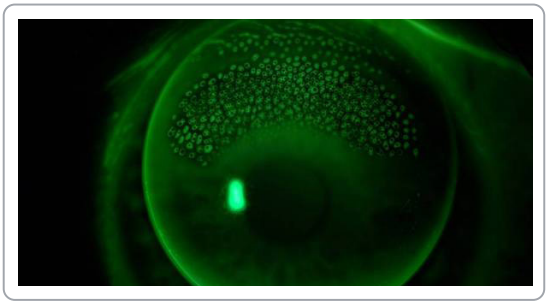


Figure 4: Dimple veil visible with fluorescein and blue light illumination

Describing clinical appearance

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

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 ≤ 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
- ≥ 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

Further Reading

154, 155, 156, 157, 158, 159, 160, 161, 162

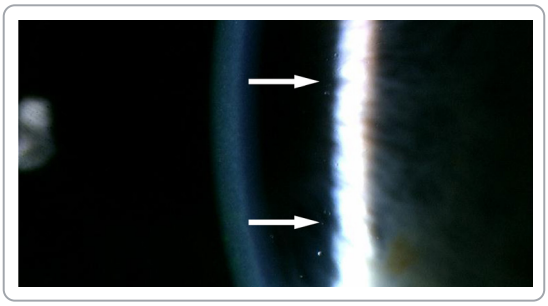


Figure 1: Microcysts, reversed illumination

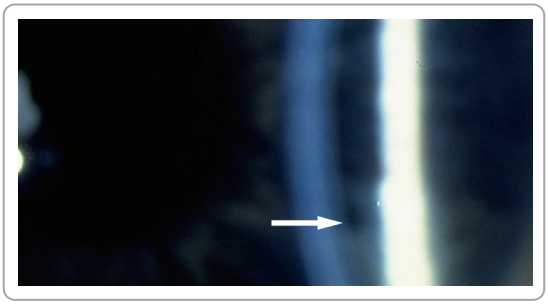


Figure 2: Vacuoles, unreversed illumination

Describing clinical appearance

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

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 ≥ 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 × 10-9, overnight wear 87 × 10-9 (Holden & Mertz, 1984)
- DW 35 × 10-9, overnight wear 125 × 10-9 (Harvitt & Bonnano, 1999)
- DW (central) 19.8 × 10-9, DW (peripheral) 32.6 x 10-9 (Morgan and Brennan, 2010)

Prognosis

Good

Differential Diagnosis

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

Further Reading

162, 163, 164, 165, 166, 167, 168, 169

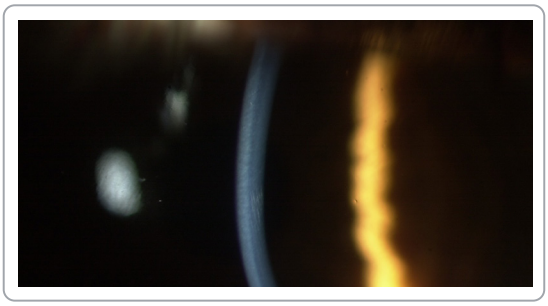


Figure 1: Striae, visible in optic section

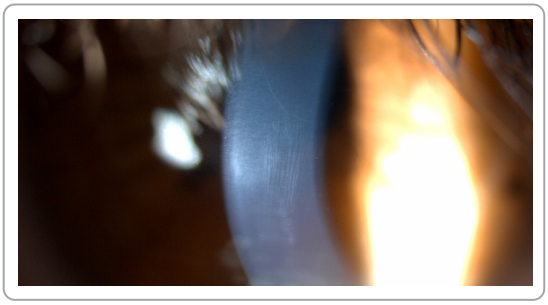


Figure 2: Differential diagnosis: Vogt's striae

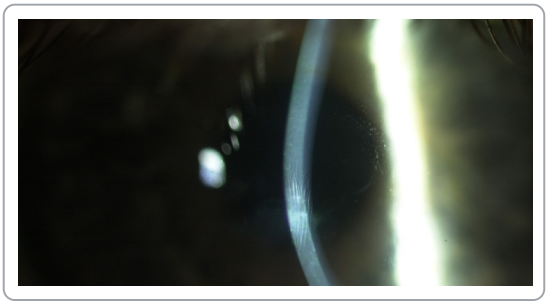


Figure 3: Differential diagnosis: Vogt's striae

Describing clinical appearance

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

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 ≥ 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 × 10-9, overnight wear 87 × 10-9 (Holden & Mertz, 1984)
  - DW 35 × 10-9, overnight wear 125 × 10-9 (Harvitt & Bonnano, 1999)
  - DW (central) 19.8 × 10-9, DW (peripheral) 32.6 x 10-9 (Morgan & Brennan, 2010)

Prognosis

Good

Differential Diagnosis

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

Further Reading

170, 171, 172, 173, 174, 175, 176, 177

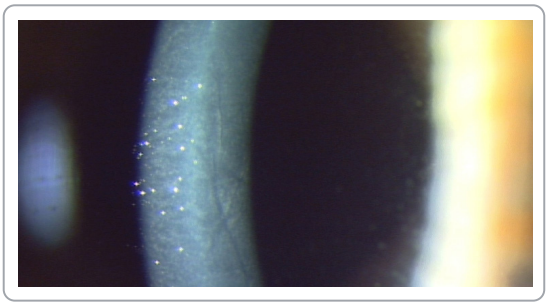


Figure 1: Corneal folds

Describing clinical appearance

Count and record number of folds:
1 fold = 8% oedema

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

Further Reading

178, 179, 180, 181

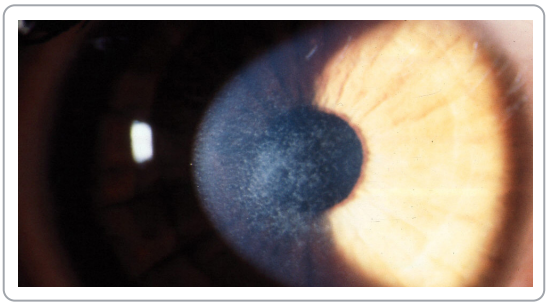


Figure 1: Post refractive surgery (PRK) scar

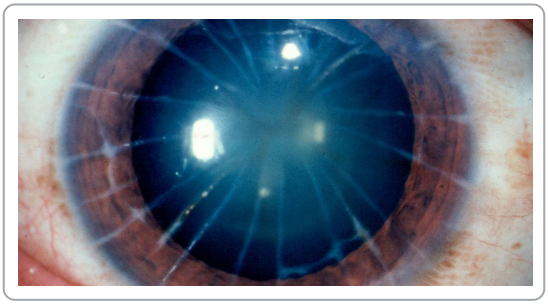


Figure 2: Marked radial keratotomy (RK) refractive surgery scars

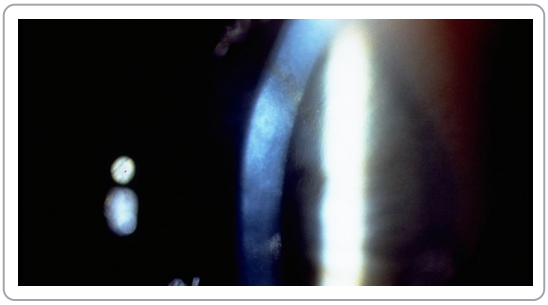


Figure 3: Scarring, unknown cause

Describing clinical appearance

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

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

Further Reading

182, 183, 184, 185, 186, 187, 188

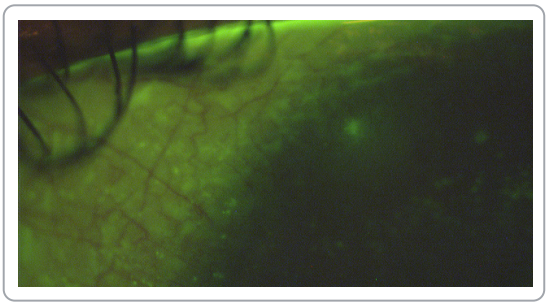


Figure 1: Asymptomatic infiltrative keratitis staining with fluorescein

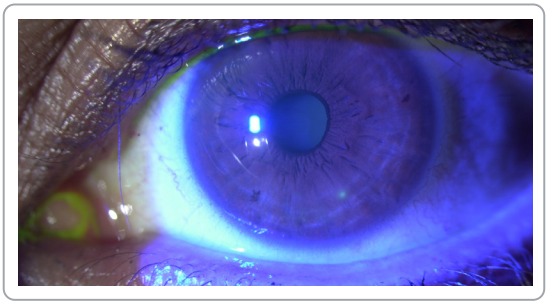


Figure 2: Asymptomatic infiltrative keratitis staining with fluorescein

Describing clinical appearance

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

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

Further Reading

189, 190, 191, 192, 193, 194, 195, 196, 197, 198

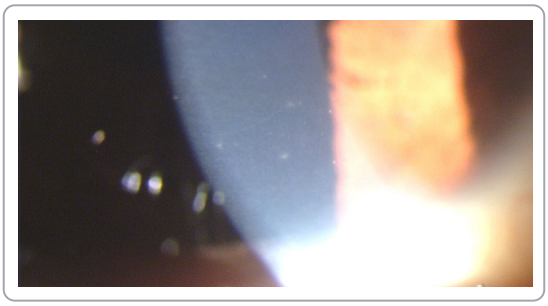


Figure 1: Infiltrative keratitis

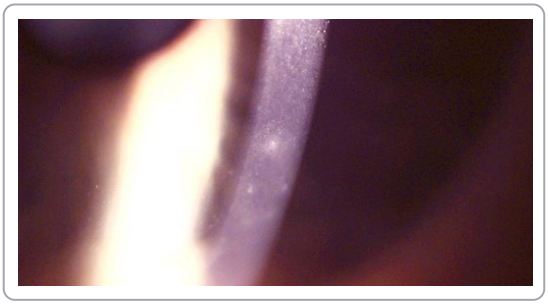


Figure 2: Infiltrative keratitis

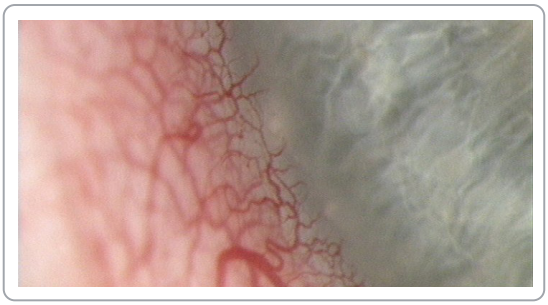


Figure 3: Marginal infiltrates with associated hyperaemia

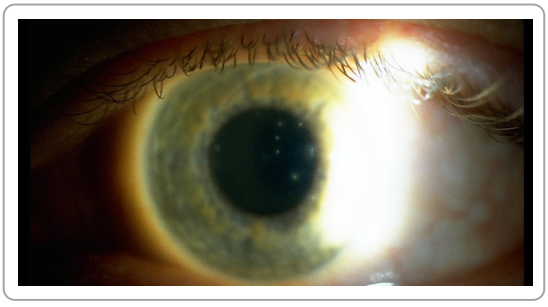


Figure 4: Infiltrative keratitis

Describing clinical appearance

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

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

Further Reading

199, 200, 201, 202, 203, 204, 205, 206, 207, 208

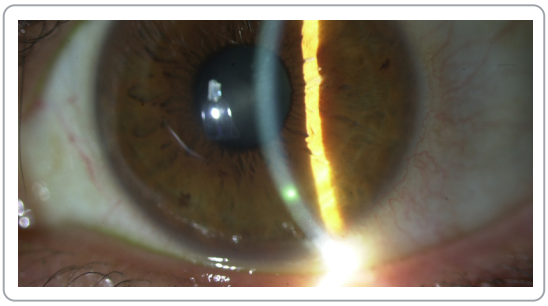


Figure 1: Active CLPU view in optic section

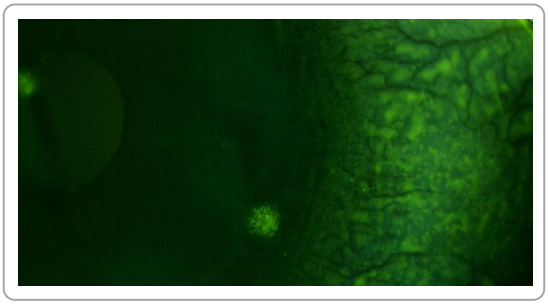


Figure 2: Active CLPU staining with fluorescein

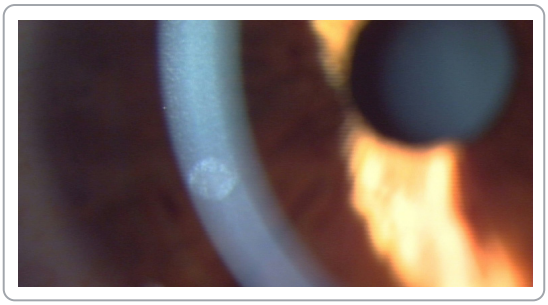


Figure 3: CLPU scar

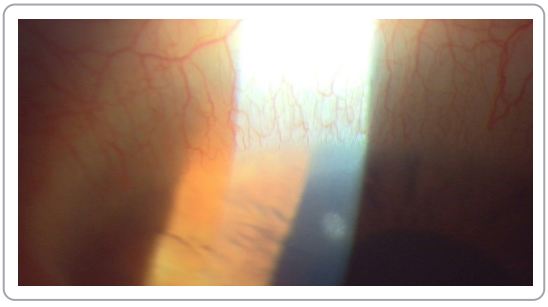


Figure 4: CLPU scar

Describing clinical appearance

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

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

Further Reading

209, 210, 211, 212, 213, 214, 215

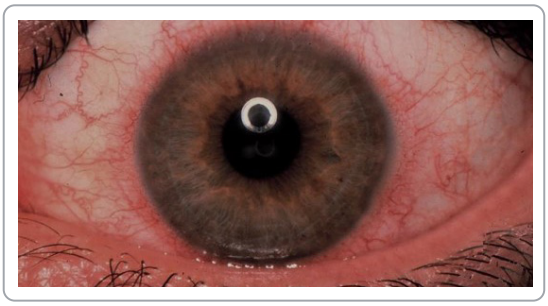


Figure 1: Contact lens associated red eye (CLARE)

Describing clinical appearance

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

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

Further Reading

216, 217, 218, 219, 220, 221, 222, 223, 224

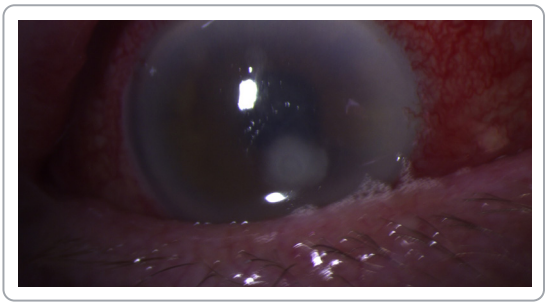


Figure 1: Large inferior central ulcer viewed under white light

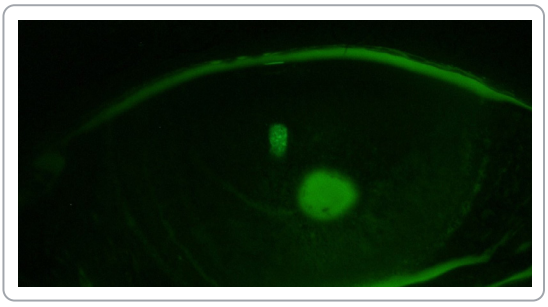


Figure 2: Large inferior central ulcer viewed with fluorescein

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

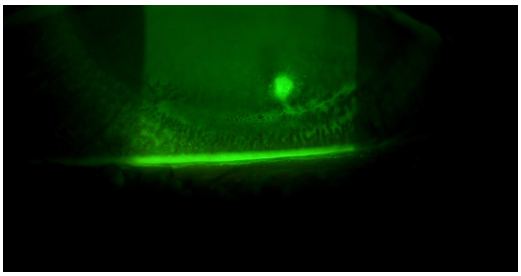


Figure 3: Inferior ulcer viewed with fluorescein

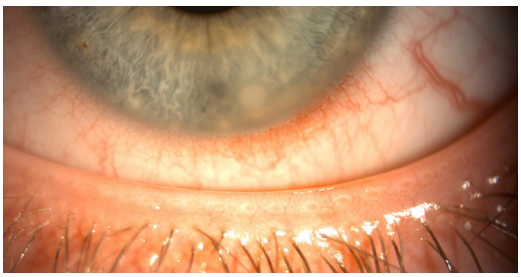


Figure 4: Microbial keratitis with associated limbal hyperaemia

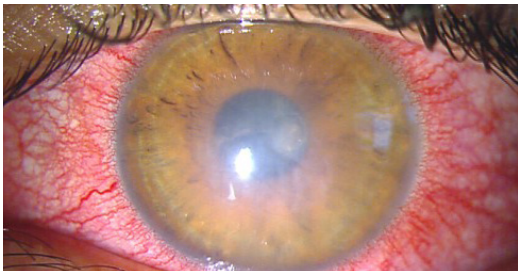


Figure 5: Microbial keratitis with associated conjunctival and limbal hyperaemia

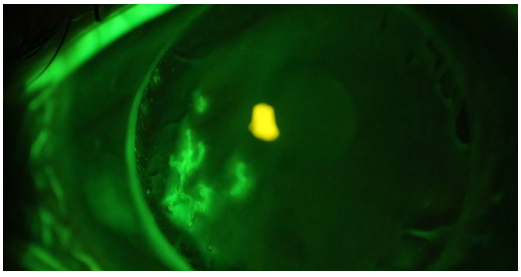


Figure 6: Herpes simplex keratitis viewed with fluorescein

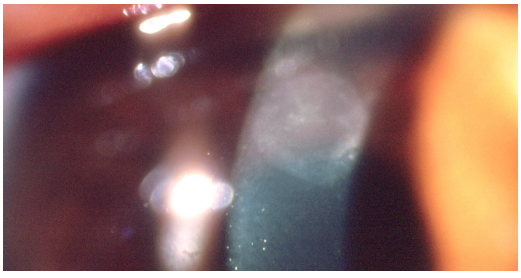


Figure 7: Acanthamoeba keratitis

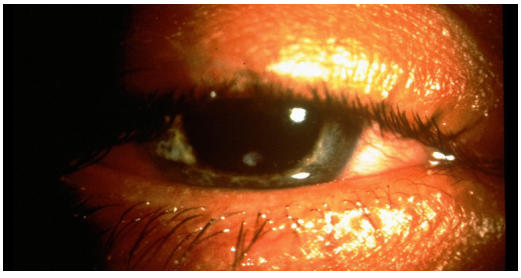


Figure 8: oedematous lids with active corneal ulcer visible

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

Further Reading

225, 226, 227, 228, 229, 230, 231, 232, 233

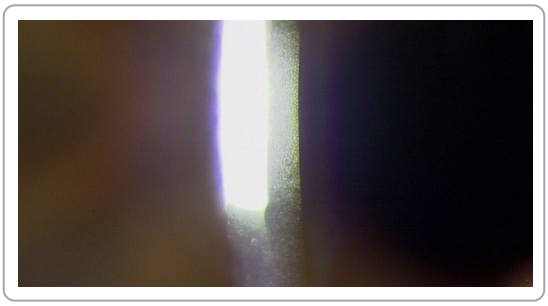


Figure 1: Corneal endothelial cells viewed in specular reflection

Describing clinical appearance

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

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, decentred 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

Further Reading

234, 235, 236, 237

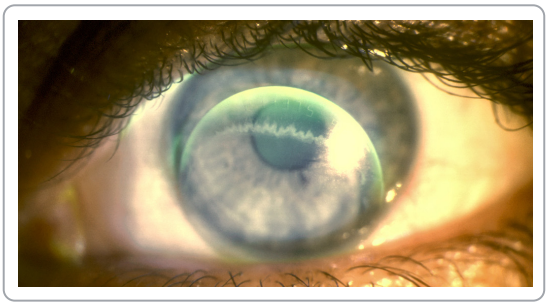


Figure 1: Rigid corneal lens bound to cornea

Describing clinical appearance

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

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
- Steepening 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/warpage – 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)

Further Reading

238, 239, 240, 241, 242, 243, 244, 245

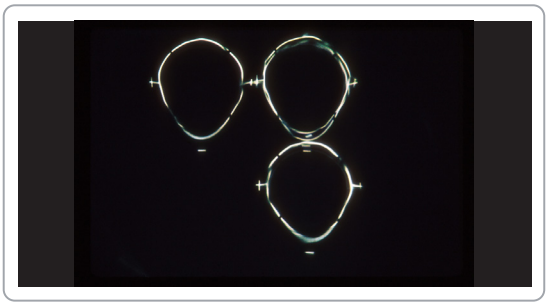


Figure 1: Corneal distortion illustrated by distorted keratometer mires

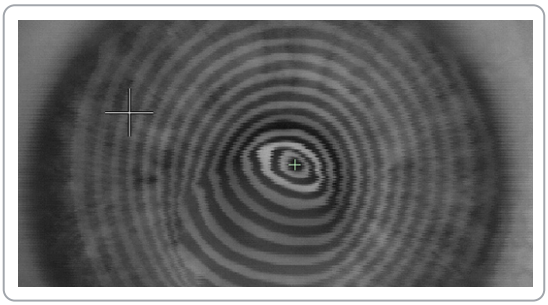


Figure 2: Corneal distortion illustrated by distorted placido disc reflection

Describing clinical appearance

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

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)

Further Reading

246, 247, 248, 249, 250, 251

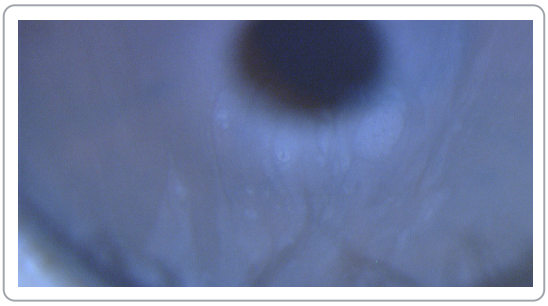


Figure 1: Tear film lipid layer viewed with EasyTearView+

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

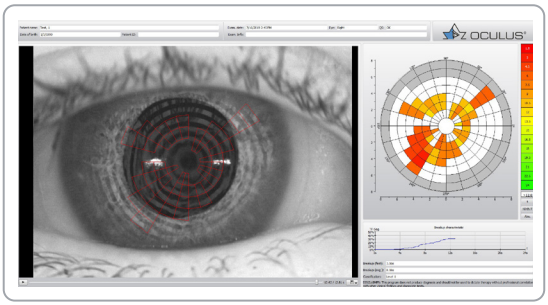


Figure 2: Image of non-invasive tear break up time viewed on Oculus K5

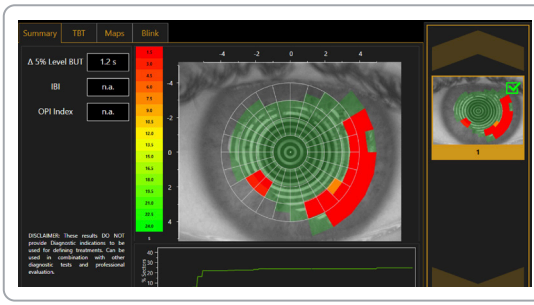


Figure 3: Image of non-invasive tear break up time

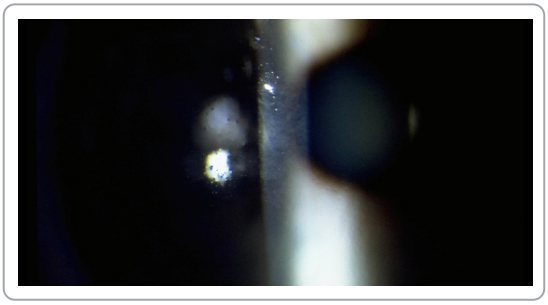


Figure 4: Example of poor tear film quality

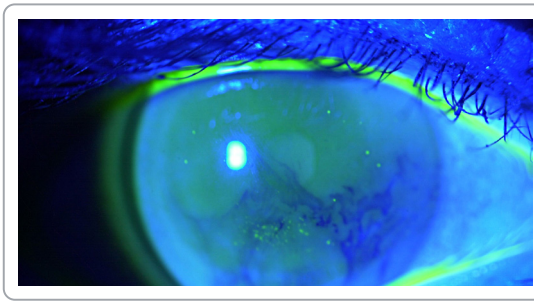


Figure 5: Fluorescein tear film break up

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

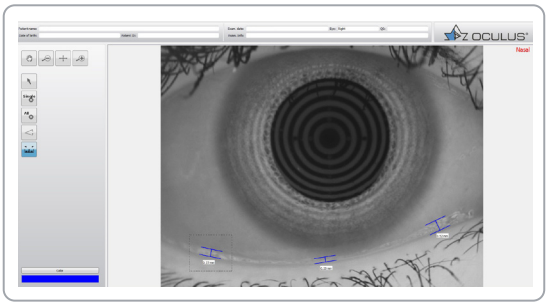


Figure 1: Tear meniscus height measurement on Oculus K5

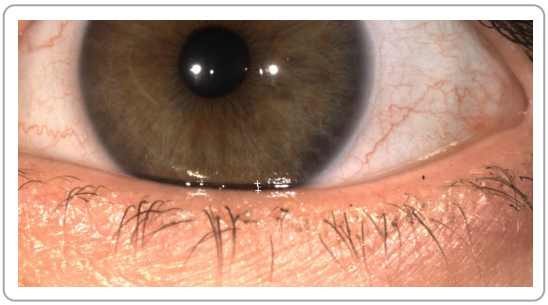


Figure 2 Tear meniscus height with graticule markings visible

Describing clinical appearance

- Low: ≤0.1mm or a difference of at least 0.06mm between the eyes.
- Medium: 0.1mm to 0.25mm
- High: ≥0.25mm (indicates reflex tearing and / or deficiency in naso-lacrimal drainage)

Further Reading

252, 253, 254, 255, 256, 257, 258, 259, 260, 261

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

Further Reading

262, 263, 264, 265, 266, 267

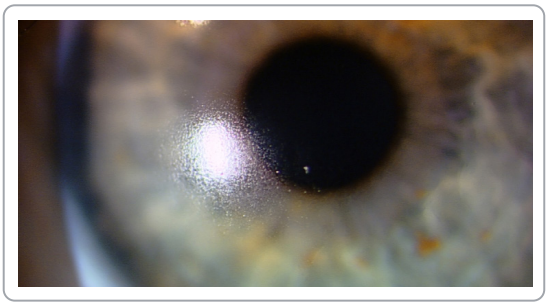


Figure 1: Lens surface drying between blinks

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

Further Reading

268, 269, 270, 271, 272, 273, 274, 275

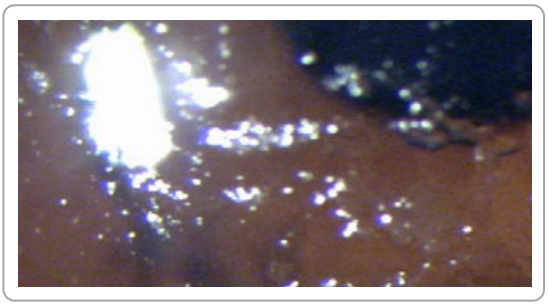


Figure 1: Calculi on lens

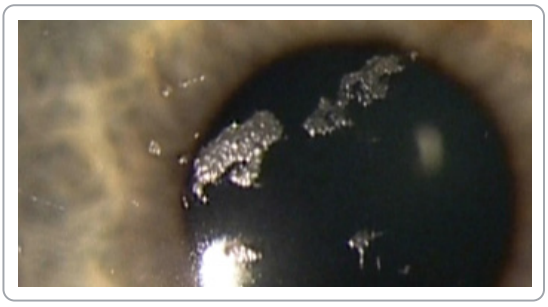


Figure 2: Calculi visible in centre of lens

Describing clinical appearance

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

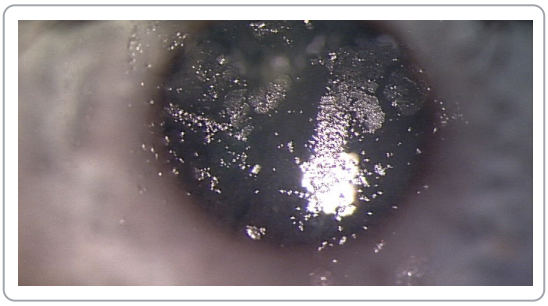


Figure 3: Heavily deposited lens

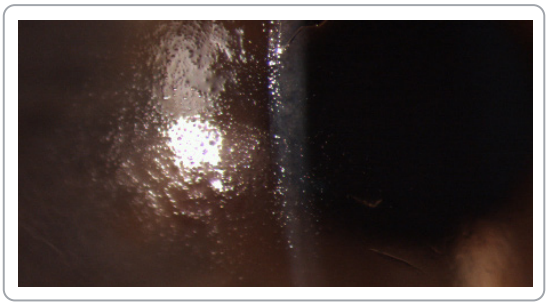


Figure 4: Deposits visible on a silicone hydrogel contact lens

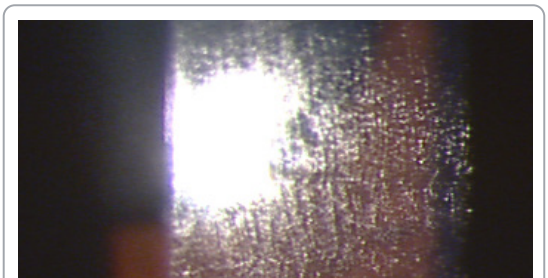


Figure 5: Lipid film on lens surface

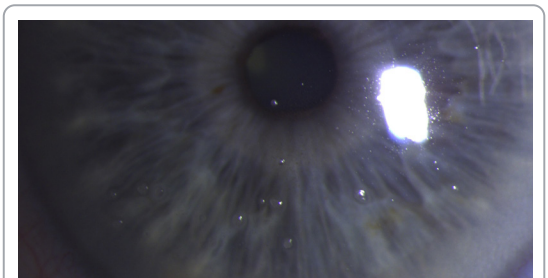


Figure 6: Discrete lipid type deposits on lens surface

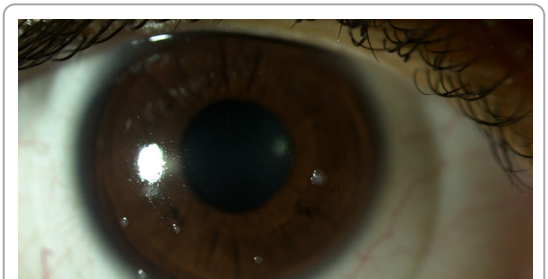


Figure 7: Calculi (Jelly bumps)

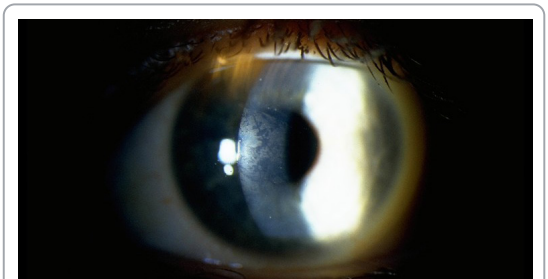


Figure 8: Protein film

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

Further Reading

276, 277, 278, 279, 280

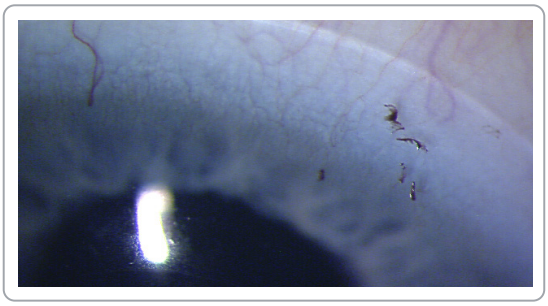


Figure 1: Low magnification view of make up on lens

Describing clinical appearance

Extent: 1-100% coverage of lens and description

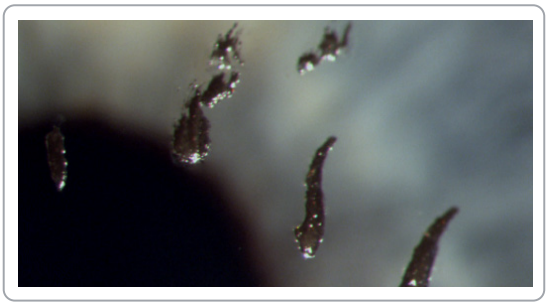


Figure 2: Higher magnification view of mascara on lens

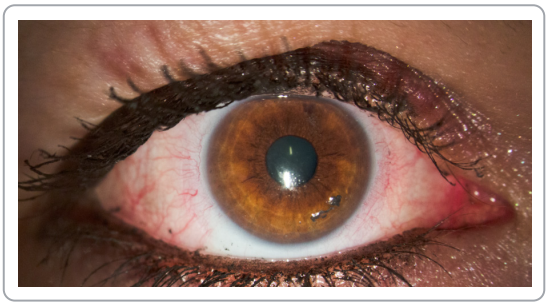


Figure 3: Make up on lens

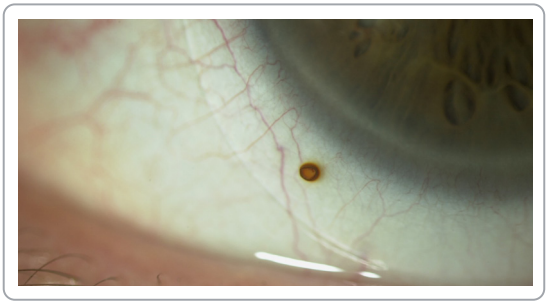


Figure 4: Rust spot

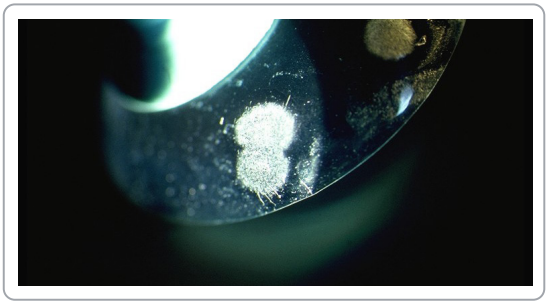


Figure 5: Fungal deposit

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

Further Reading

281, 282, 283, 284

Describing clinical appearance

Extent: 1-100% coverage of lens and description

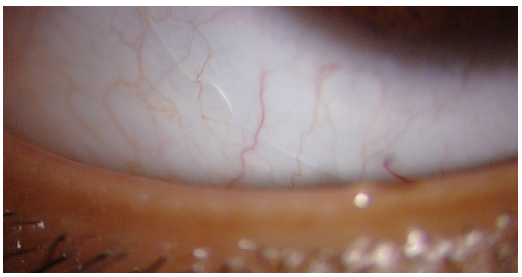


Figure 1: Nick in soft lens edge

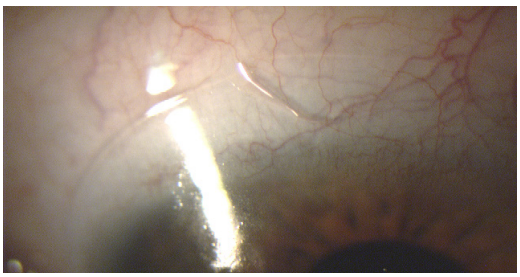


Figure 2: Tear in soft lens edge

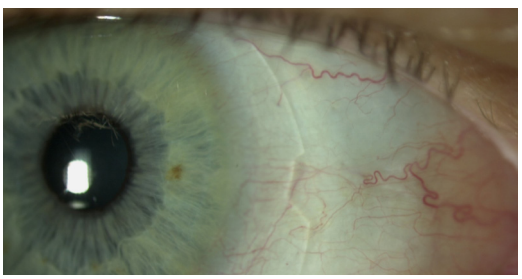


Figure 3: Torn soft lens edge

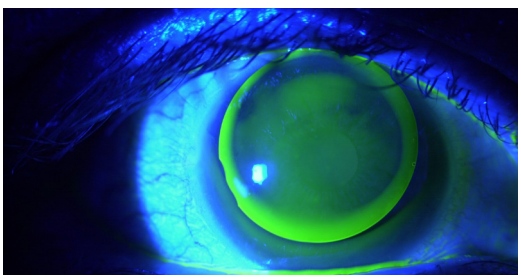


Figure 4: Chipped rigid corneal lens edge

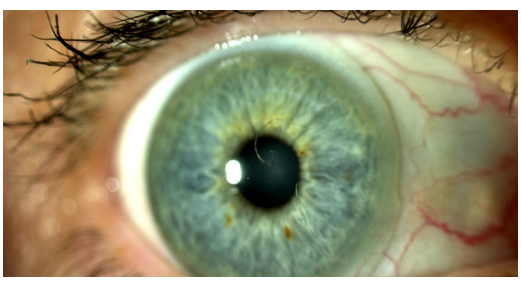


Figure 5: Crack in soft lens

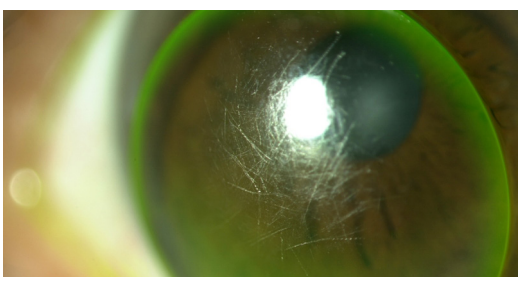


Figure 6: Scratched rigid corneal lens

Index

A

B

C

D

E

F

G

H

I

K

L

R

S

T

U

V

W

Further Reading & References

- 1. Wolffsohn et al. Contact Lens Evidence-based Academic Reports (CLEAR). Cont Lens Ant Eye 2021; 12(4): 22-34
- 2. Efron, N et al. Validation of grading scales for contact lens complications. Ophthalmic Physiol Opt 2001; 21(1): 17-29
- 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
- 4. Dumbleton K. Adverse events with silicone hydrogel continuous wear. Cont Lens Ant Eye 2002; 25(3): 137-146
- 5. Jalbert I et al. Increased numbers of demodex in contact lens wearers. Optom Vis Sci 2015; 92(6): 671-678
- 6. Nelson J et al. The international workshop on meibomian gland dysfunction: report of the definition and classification subcommittee. Invest Ophthalmol Vis Sci 2011 Mar; 52(4): 1930-7.
- 7. McCulley J et al. Eyelid disorders: the meibomian gland, blepharitis, and contact lenses. Eye Contact Lens 2003; 29: S93-5; discussion S115-8, S192-4.
- 8. Markoulli M et al. Contact lens wear and dry eyes: challenges and solutions. Clin Optom (Auckl) 2017; 9: 41-48.
- 9. Smith R et al. Chronic blepharitis: a review. CLAO J 1995; 21(3): 200-207
- 10. Tarkowski W et al. Demodex sp. as a potential cause of the abandonment of soft contact lenses by their existing users. Biomed Res Int 2015; 259109
- 11. Weissman B et al. Risk factor for contact lens associated microbial keratitis. Cont Lens Ant Eye 2002; 25(1): 3-9
- 12. Zhang A et al. Ocular Demodex: a systematic review of the clinical literature. Ophthalmic Physiol Opt 2020; 40: 389-432.
- 13. Arita R et al. Meibomian Gland Dysfunction and Contact Lens Discomfort. Eye Contact Lens 2017 Jan; 43(1): 17-22
- 14. Blackie C et al. The sustained effect (12 months) of a single-dose vectored thermal pulsation procedure for meibomian gland dysfunction and evaporative dry eye. Clin Ophthalmol 2016; 10: 1385-1396
- 15. Geerling G et al. Emerging strategies for the diagnosis and treatment of meibomian gland dysfunction: Proceedings of the OCEAN group meeting. Ocul Surf 2017; 15: 179-192
- 16. Lam P et al. A Review on Evidence-Based Treatments for Meibomian Gland Dysfunction. Eye Contact Lens 2020; 46: 3-16.
- 17. Schaumberg D et al. (2011) The international workshop on meibomian gland dysfunction: report of the subcommittee on the epidemiology of, and associated risk factors for, MGD. Invest Ophthalmol Vis Sci 2011; 52(4): 1994-2005

- 18. Siddireddy J et al. The eyelids and tear film in contact lens discomfort. Cont Lens Ant Eye 2018; 41(2) 144-153
- 19. Siddireddy J et al. Predictive Potential of Eyelids and Tear Film in Determining Symptoms in Contact Lens Wearers. Optom Vis Sci 2018; Nov 95(11): 1035-1045
- 20. Siddireddy J et al. The Effect of Microblepharon Exfoliation on Clinical Correlates of Contact Lens Discomfort. Optom Vis Sci 2019; 96(3): 187-199
- 21. Siddireddy J et al. Effect of Eyelid Treatments on Bacterial Load and Lipase Activity in Relation to Contact Lens Discomfort. Eye Contact Lens 2020; 46(4): 245-253
- 22. Tichenor A et al. Effect of the Bruder moist heat eye compress on contact lens discomfort in contact lens wearers: an open-label randomized clinical trial. Cont Lens Ant Eye 2019; 42: 625-32
- 23. Berry M et al. Mucins and ocular signs in symptomatic and asymptomatic contact lens wear. Optom Vis Sci 2008; 85(10): E930-8
- 24. Doughty M et al. Visualisation of “Marx’s line” along the marginal eyelid conjunctiva of human subjects with lissamine green dye. Ophthalmic Physiol Opt 2004; 24(1): 1-7
- 25. Efron N et al. Lid wiper epitheliopathy. Prog Retin Eye Res 2016; 53: 140-74.
- 26. Knop E et al. The lid margin is an underestimated structure for preservation of ocular surface health and development of dry eye disease. Dev Ophthalmol 2010; 45: 108-22
- 27. Korb D et al. Lid-wiper epitheliopathy and dry-eye symptoms in contact lens wearers. CLAO J 2002; 28(4): 211-6
- 28. Korb D et al. Lid wiper epitheliopathy and dry eye symptoms. Eye Contact Lens 2005; 31(1): 2-8
- 29. Korb D et al. Prevalence of lid wiper epitheliopathy in subjects with dry eye signs and symptoms. Cornea 2010; 29(4): 377-83
- 30. Lievens C et al. Optimal methodology for lid wiper epitheliopathy identification. Cont Lens Ant Eye 2020; S1367-0484(20)30099-0
- 31. Lin M et al. Mechanical complications induced by silicone hydrogel contact lenses. Eye & CL 2013; 39(1): 115–124
- 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
- 38. Campbell R, Caroline P. Vascularized limbal keratitis. Spectrum 1996 August: 72.
- 39. Jaros P, DeLuise V. Pinguecula and pterygia. Surv Ophthalmol 1988; 33: 41-49
- 40. 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.
- 41. Prediger J, Edmondson L. Management of CL patients with Pinguecula or pterygia Optom Vis Sci 1993; 70(1): 9-14
- 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. Noureddin G, Yeung S: The use of dry amniotic membrane in pterygium surgery. Clin Ophthalmol 2016; 10: 705-12.
- 47. Pham T et al. Pterygium/pinguecula and the five-year incidence of age-related maculopathy. Am J Ophthalmol 2005; 139 (3): 536-5377
- 48. Prediger J, Edmondson L. Management of CL patients with Pinguecula or pterygia. Optom Vis Sci 1993; 70(1): 9-14
- 49. Sheppard J et al.: An update on the surgical management of pterygium and the role of loteprednol etabonate ointment. Clin Ophthalmol 2014; 8: 1105-18.
- 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
- 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
- 58. Efron N. Contact lens induced conjunctival hyperaemia. Optician 1997; 213(5591): 22-27
- 59. Huntjens B et al. Evaluating a new objective grading software for conjunctival hyperaemia. Cont Lens Anterior Eye 2020; 43: 137-143.
- 60. Guillon M, Shah D. Objective measurement of CL induced conjunctival redness. Optom Vis Sci 1996; 73(9): 595-605
- 61. Murphy PJ et al. How red is a white eye? Clinical grading of normal conjunctival hyperaemia. Eye (Lond) 2007; 21: 633-8.
- 62. Pritchard N et al. Ocular and subjective responses to frequent replacement of daily wear soft contact lenses. CLAO J 1996; 22(1): 53-59
- 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.
- 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
- 67. Michel M et al. The effectiveness of questionnaires in the determination of contact lens induced dry eye. Ophthalmic Physiol Opt 2009; 29(5): 479-86
- 68. Pult H et al. A novel method to predict the dry eye symptoms in new contact lens wearers. Optom Vis Sci 2009; 86(9): E1042-1050
- 69. Pult H, Bandlitz S. Lid-Parallel Conjunctival Folds and Their Ability to Predict Dry Eye. Eye Contact Lens 2018; 44 Suppl 2: S113-S119.
- 70. Pult H, & Riede-Pult B. Impact of soft contact lenses on lid-parallel conjunctival folds. Cont Lens Ant Eye 2019; 42(4): 415-419
- 71. Tapaszto B et al: OCT Imaging of lid-parallel conjunctival folds in soft contact lens wearers. Optom Vis Sci 2011; 88: 1206-13.

- 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
- 74. Efron N. Contact lens induced papillary conjunctivitis. Optician 1997; 213(5583): 20-27
- 75. Jurkus J. Contact lens induced papillary conjunctivitis. In Anterior Segment Complications of contact lens wear 1994; Vol 1 (ed, Silbert J) pp 163—177. Churchill Livingstone Inc, New York
- 76. Lin M, Yeh T. Mechanical complications induced by silicone hydrogel contact lenses. Eye & CL 2013; 39(1): 115–124
- 77. Skotnitsky C et al. General and local contact lens induced papillary conjunctivitis (CLPC). Clin Exp Optom 2002; 85: 193-7.
- 78. Skotnitsky C et al. Two presentations of contact lens-induced papillary conjunctivitis (CLPC) in hydrogel lens wear: local and general. Optom Vis Sci 2006; 83: 27-36.
- 79. Sorbara L et al. Contact lens induced papillary conjunctivitis with silicone hydrogel lenses. Cont Lens Anterior Eye 2009; 32: 93-6.
- 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): 942-948Papas E. On the relationship between soft contact lens oxygen transmissibility and induced limbal hyperaemia. Exp Eye Res 1998; 67: 125-31.
- 82. Papas E. The role of hypoxia in the limbal vascular response to soft contact lens wear. Eye Contact Lens 2003; 29: S72-4; discussion S83-4, S192-4.
- 83. Pritchard N et al. Ocular and subjective responses to frequent replacement of daily wear soft contact lenses. CLAO J 1996; 22(1): 53-59
- 84. Pult H et al. Limbal and bulbar hyperaemia in normal eyes. Ophthalmic Physiol Opt 2008; 28: 13-20.
- 85. Szczotka-Flynn LB et al Contact Lenses Manufactured in Etafilcon A Are Noninferior to Two Silicone Hydrogel Lens Types With Respect to Hypoxic Stress. Eye & Contact Lens 2018;44:190-9.
- 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
- 88. Sendele D et al. Superior limbic keratoconjunctivitis in contact lens wearers. Ophthalmology 1983; 90: 616-22.
- 89. Stenson S. Superior limbic keratoconjunctivitis associated with soft contact lens wear. Arch Ophthalmol 1983; 101: 402-4.
- 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.
- 94. Efron N. Corneal neovascularisation. in Contact Lens Complications, N. Efron, Editor. London, Elsevier, 2012, pp 214 - 224.
- 95. Efron N et al. Thirty years of 'quiet eye' with etafilcon A contact lenses. Cont Lens Anterior Eye 2020; 43: 285-297.
- 96. Jones L, Jones D: Non-inflammatory corneal complications of contact lens wear. Cont Lens Anterior Eye 2001; 24: 73-9.
- 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.
- 100. Andrasko G, Ryen K. Corneal staining and comfort observed with traditional and silicone hydrogel lenses and multipurpose solution combinations. Optometry 2008 Aug; 79(8): 444-454
- 101. Bron AJ et al. Clinical staining of the ocular surface: mechanisms and interpretations. Prog Retin Eye Res 2015; Jan 44: 36-61
- 102. Carnt N et al. Solution toxicity in soft contact lens daily wear is associated with corneal inflammation. Optom Vis Sci 2007; Apr 84(4): 309-315
- 103. Efron N. Putting vital stains in context. Clin Exp Optom 2013; 96: 400-21.
- 104. Fadel D. Scleral lens issues and complications related to a non-optimal fitting relationship between the lens and ocular surface. Eye Contact Lens 2019; 45: 152-163
- 105. Fonn D et al. Corneal staining as a response to contact lens wear. Eye Contact Lens 2010; Sep 36(5): 318-321
- 106. Jones L et al. Asymptomatic corneal staining associated with the use of balafilcon silicone-hydrogel contact lenses disinfected with a polyaminopropyl biguanide-preserved care regimen. Optom Vis Sci 2002; Dec 79(12): 753-61
- 107.Keir N et al. Clinical performance of different care systems with silicone hydrogel contact lenses. Cont Lens Ant Eye 2010; Aug 33(4): 189-195
- 108. Morgan P, Maldonado-Codina C. Corneal staining: do we really understand what we are seeing? Cont Lens Anterior Eye 2009; 32: 48-54.
- 109. Peterson R et al. Impact of a rub and rinse on solution-induced corneal staining. Optom Vis Sci 2010; Dec 87(12): 1030-1036
- 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.
- 115. Dougal J. Abrasions secondary to contact lens wear. In Complications of contact lens wear 1992; Vol 1 (ed Tomlinson A), pp 123—156 Mosby Year Books, St Louis
- 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
- 118. Campbell R, Caroline P. Dellen formation in RGP contact lens patients. Contact Lens Spectrum 1995; 10 (6): 56
- 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.
- 124. Andrasko G, Ryen 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 balafilcon 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.
- 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
- 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
- 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
- 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
- 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
- 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.
- 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.
- 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.
- 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
- 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.
- 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.
- 216. Bergmanson 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.
- 225. Bergmanson J. Effects of contact lens wear on corneal ultrastructure. Cont Lens Anterior Eye 2001; 24: 115-20.

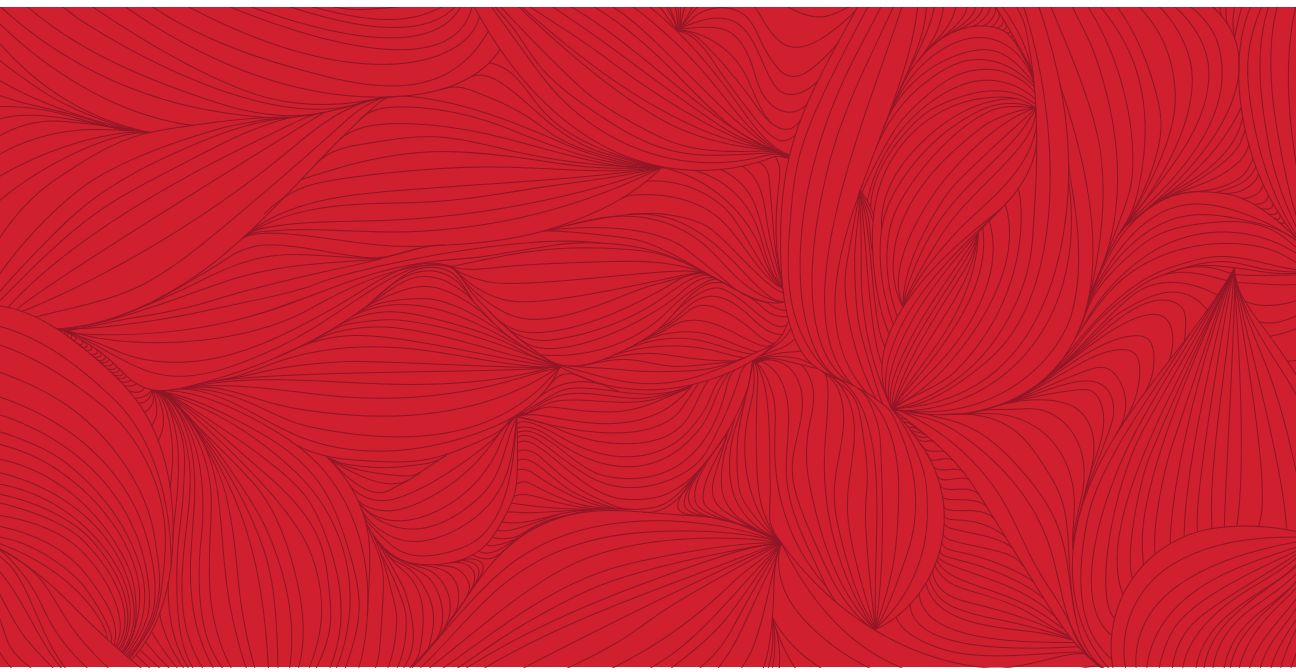
- 226. Bourne W. The effect of long-term contact lens wear on the cells of the cornea. CLAO J 2001 Oct; 27(4): 225-230
- 227. Efron N. Contact lens induced endothelial polymegathism. Optician 1996; 212(5575): 20-28
- 228. Doughty M, Fonn D. Pleomorphism and endothelial cell size in normal and polymegathous human corneal endothelium. Int Contact Lens Clin 1993; 20(5/6): 116-122
- 229. 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.
- 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.
- 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.
- 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.
- 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
- 249. 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
- 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
- 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
- 258. Kokke K et al. Oral omega-6 essential fatty acid treatment in contact lens associated dry eye. Cont Lens Ant Eye 2008; Jun 31(3): 141-146; quiz 170
- 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.
- 262. Guillon M et al. Effect of lens care system on silicone hydrogel contact lens wettability. Cont Lens Ant Eye 2015; 38(6): 435-441
- 263. Guillon M et al. Effect of lens care system on silicone hydrogel contact lens wettability. Cont Lens Anterior Eye 2015; 38: 435-41.
- 264. Guillon M et al. Association Between Contact Lens Discomfort and Pre-lens Tear Film Kinetics. Optom Vis Sci 2016; 93: 881-91.
- 265. Guillon M et al.: Quantification of contact lens wettability after prolonged visual device use under low humidity conditions. Cont Lens Anterior Eye 2019; 42: 386-391.
- 266. Jie Y et al. Evaluation of incomplete blinking as a measurement of dry eye disease. Ocul Surf 2019 Jul; 17(3): 440-446
- 267. Keir N, Jones L. Wettability and silicone hydrogel lenses: a review. Eye Contact Lens 2013; 39(1): 100-108
- 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
- 270. Nichols J. Deposition on silicone hydrogel lenses. Eye Contact Lens 2013; 39(1): 20-23
- 271. Omali, N et al. Lipid deposition on contact lenses in symptomatic and asymptomatic contact lens wearers. Cont Lens Ant Eye 2012; 44(1): 56-61
- 272. Subbaraman L. Protein deposition and clinical symptoms in daily wear of etafilcon lenses. Optom Vis Sci 2012; 89(10): 1450-1459
- 273. Tighe B. A decade of silicone hydrogel development: surface properties, mechanical properties, and ocular compatibility. Eye Contact Lens 2013; 39(1): 4-12
- 274. Walther H et al. Factors that influence in vitro cholesterol deposition on contact lenses. Optom Vis Sci 2013; 90(10): 1057-1065
- 275. Willcox M. Solutions for care of silicone hydrogel lenses. Eye Contact Lens 2013; 39(1): 24-28

- 276. Campbell D et al. The significance of hand wash compliance on the transfer of dermal lipids in contact lens wear. Cont Lens Anterior Eye 2012; 35: 71-6; quiz 96-7.
- 277. Gnadt G, Gordon A. Fungal contamination of soft contact lenses. Int Contact Lens Clin 1991; 18: 102-104
- 278. Ng A et al. Eye cosmetic usage and associated ocular comfort. Ophthalmic Physiol Opt 2012; 32(6): 501-507
- 279. Ng A et al. Migration of Cosmetic Products into the Tear Film. Eye Contact Lens 2015; 41(5): 304-309
- 280. Ng A et al. Impact of Eye Cosmetics on the Eye, Adnexa, and Ocular Surface. Eye Contact Lens 2016; 42(4): 211-220
- 281. Bhamra T, Tighe B. Mechanical properties of contact lenses: The contribution of measurement techniques and clinical feedback to 50 years of materials development. Cont Lens Anterior Eye 2017; 40: 70-81.
- 282. Dennis R et al. The durability of hydrogel extended-wear contact lenses worn for daily wear by USAF aircrew members. Aviat Space Environ Med 1991; 62: 565-8.
- 283. Jones L et al. Life expectancy of rigid gas permeable and high water content contact lenses. CLAO J 1996; 22: 258-61.
- 284. Tranoudis I, Efron N. Tensile properties of soft contact lens materials. Cont Lens Anterior Eye 2004; 27: 177-91.
- 285. Yoon K et al. Usefulness of double vital staining with 1% fluorescein and 1% lissamine green in patients with dry eye syndrome. Cornea 2011 Sept; 30(9): 972-976

J&J

Institute



Vision Made Possible™