

CONTACT LENS COMPANION

Contamac™

TABLE OF CONTENTS

3 and 9 o'clock Staining

Acanthamoeba Ulcer

Acute Hydrops

Asymptomatic Infiltrates (AI)

Asymptomatic Infiltrative Keratitis (AIK)

Bubbles in the Tear Reservoir of Scleral Lenses

Bulbar and Limbal Hyperaemia in Scleral Lens Wear

Conjunctival Prolapse

Contact Lens Acute Red Eye (CLARE) / Tight Lens Syndrome

Contact Lens Induced Lid Blepharoptosis (CLIP)

Contact Lens induced Superior Limbic Keratoconjunctivitis (CLSLK)

Contact lens Papillary Conjunctivitis (CLPC) / Giant Papillary Conjunctivitis (GPC)

Contact Lens Peripheral Ulcer (CLPU)

Corneal Neovascularisation (CNV)

Corneal Oedema

Corneal Scarring Superficial Fibroblastic Nodules or "Proud Nebulas"

Dimple Veiling

Effect of Contact Lenses on Blinking

Endothelial Bedewing

Endothelial Blebs

Endothelium Polymegathism

Epithelial Bogging

Epithelial Plug

Fungal Keratitis

Inferior Arcuate Staining or Smile Staining

Infiltrative Keratitis (IK)

Lid Wiper Epitheliopathy (LWE)

Limbal Redness

Microbial Keratitis (MK) or Corneal Ulcer

Midday Fogging in Scleral Lens Wear

Mucin Balls

Pseudomonas Ulcer

Solution Sensitivity

Stromal Oedema

Superior Epithelial Arcuate Lesion (SEAL) or Epithelial Splitting

Surface Soiling Poorly Wetting Scleral Lenses

Vascularised Limbal Keratitis (VLK)

INTRODUCTION

With advancements in the science of contact lens practice, management, and manufacturing, it has become increasingly uncommon for the average practitioner to encounter some of the complications associated with contact lens wear.

This booklet serves as a quick desktop reference for practitioners who may face unusual signs or symptoms in their clinical practice. It is not intended to replace a comprehensive textbook but rather to offer a concise, practical guide.

The content has been adapted from Dr Dirk Booysen's excellent textbook, *"In Contact – Clinical Contact Lens Practice"*. Dr Booysen has generously provided access to the full text via the QR code below.

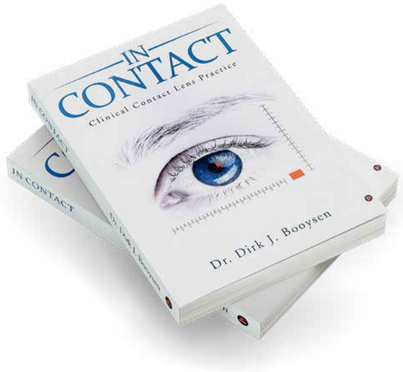
Most of the images are sourced from Dr Booysen's publication, while others have been kindly contributed by colleagues from the global contact lens community.



Martin Conway

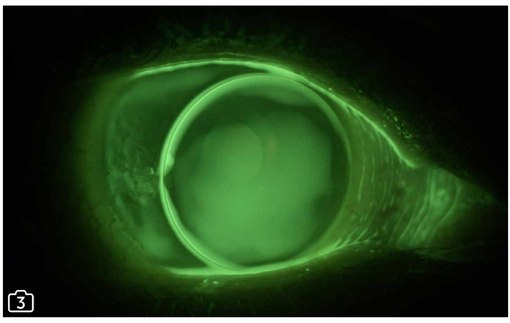
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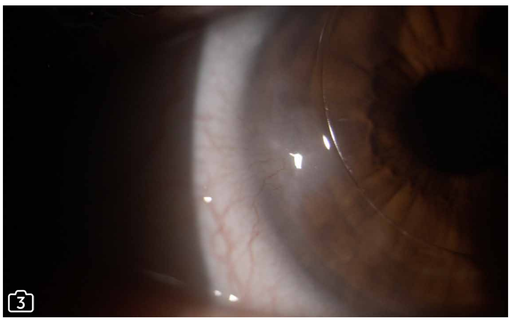
1. Dr Dirk Booysen
2. Dr Tom Arnold
3. Mark Darling
4. Dr Taldayen Rustem
5. Chris Steele
6. Aneel Suri
7. Marco Tovaglia



In Contact – Clinical Contact Lens Practice

3 AND 9 O'CLOCK STAINING





Aetiology

Corneal epithelium desiccation and disruption at limbus with RGP lens wear. More common in patients with marginal dry eye and poor or incomplete blinking patterns.

Mechanism

Rigid lenses bridge the lid away from the ocular surface leaving the ocular surface adjacent to lens edge not properly wetted. Additionally, poorly fitting RGP lenses can also abrade or chafe the cornea, especially in active eye turners.

Features/signs

Punctate or diffuse staining at the 3 & 9 o'clock locations on the cornea seen in rigid lens wearers. The staining typically has a triangular pattern with the apex pointing away from the central cornea and the base corresponding to the RGP lens edge.

Symptoms

Slight discomfort and dryness but symptoms can be more severe as the condition progresses toward Vascularised Limbal Keratitis (VLK) and corneal dellen.

Lid Oedema

Uncommon

Bulbar or Limbal Redness

Moderate to severe sectorial redness

Differential Diagnosis

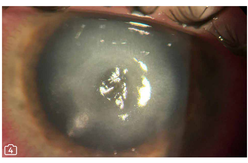
- VLK - Limbal Keratitis
- Dellen

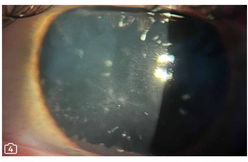
Treatment

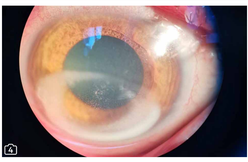
- Alter lens design
- Reduce thickness of lens edge
- Smaller lens diameter
- Toric periphery
- Blinking instructions
- Lubrication
- Treat marginal dry eye

BACK TO CONTENTS

ACANTHAMOEBA ULCER







Aetiology

Acanthamoeba keratitis, first recognised in 1973, is a rare, vision threatening, parasitic infection seen most often in soft contact lens wearers. It is both difficult to diagnose and difficult to treat. Two of the eight known species of Acanthamoeba, *A. Castellani and A. Polyphaga,* are responsible for most infections. Early signs may be mild and non-specific and the disease progresses slowly.

Mechanism

Acanthamoeba are commonly found. Free-living amoeba have been located in various environments including pools, hot tubs, tap water, shower water, and contact lens solution. Risk factors include contact lens wear, exposure to organism (often through contaminated water), and corneal trauma. It is thought that over 80% of Acanthamoeba keratitis appears in contact lens wearers. In one study, 75% of the patients were contact lens wearers; 40% wore daily soft lenses, 22% wore rigid gas permeable lenses, and 38% wore extended wear or other lenses. Acanthamoeba is ubiquitous. Corneal trauma, followed by exposure to the parasite (often through a water supply or contact lens solution) in a patient with low tear levels of anti-Acanthamoeba IgA leads to infection.

Acanthamoeba exist in two forms: active trophozoites (25 - 40µm) and dormant cysts (13 -20µm). The trophozoites are mobile and consume bacteria, yeasts, algae and small organic particles. The trophozoites form double walled cysts which are incredibly resistant to methods of eradication (including freezing, heating, chlorination, and irradiation). The cysts can survive in vitro for more than 20 years. When the environmental conditions are appropriate the cysts turn into trophozoites which produce a variety of enzymes that aid in tissue penetration and destruction.

Features/signs

Possible findings include epithelial irregularities, epithelial or subepithelial infiltrates, and pseudo dendrites. Later signs include stromal infiltrates (ring-shaped, disciform, or nummular), satellite lesions, epithelial defects, radial keratoneuritis, scleritis, and anterior uveitis (with possible hypopyon).
The radial keratoneuritis is due to the trophozoites clustering around corneal nerves and is pathognomonic for Acanthamoeba keratitis. Careful slit lamp examination is necessary to identify these infiltrative lesions which occur in up to 63% of all cases of Acanthamoeba keratitis. Advanced signs include stromal thinning and corneal perforation. There usually is minimal discharge or corneal neovascularisation and bacterial superinfection with corneal ulceration may occur later in the course.

Symptoms

It is often characterised by pain out of proportion to findings and the late clinical appearance of a stromal ring-shaped infiltrate. Patients also complain of decreased vision, redness, foreign body sensation, photophobia, tearing, and discharge. Symptoms may wax and wane; they may be quite severe at times.

Lid Oedema

Usual, severe blepharospasm may be present

Bulbar or Limbal Redness

Severe circumcorneal hyperemia and conjunctival chemosis may be present

Differential Diagnosis

The differential diagnosis for Acanthamoeba in its early clinical stages includes dry eye, herpes simplex virus keratitis, recurrent corneal erosion, staph marginal keratitis, and contact lens associated keratitis. The differential diagnosis of later clinical stages includes viral, bacterial, fungal, and sterile (such as from topical anaesthetic abuse) keratitis.

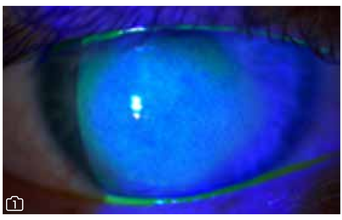
Treatment

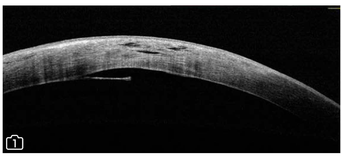
Since treatment is toxic, lengthy, and not necessarily effective, prevention is essential. Both trophozoites and cysts can adhere to the surface of soft and rigid contact lenses. Any breaks in the corneal epithelium or loss of corneal defence mechanisms may allow them to invade the corneal tissue. Importantly both trophozoites and cysts can harbour a variety of microorganisms including *Pseudomonas spp, salmonella, mycobacterium* and others which may enhance the attachment of the trophozoite to hydrogel contact lenses and cause co-infections. Early diagnosis ensures a good prognosis and vision recovery. Medical treatment for Acanthamoeba keratitis is still evolving and extremely challenging. Never use tap water in the cleaning regime of any contact lens.

BACK TO CONTENTS

ACUTE HYDROPS







Aetiology

Keratoconus is the primary cause of acute hydrops. Although hydrops is not technically a complication or adverse effect of contact lens wear, it occurs in keratoconic patients wearing bespoke contact lenses for visual correction and therefore contact lens practitioners will deal with this complication during their career.

Mechanism

Corneal hydrops occurs due to tears in Descemet's membrane causing the edges to roll, thereby creating a gap in the membrane through which aqueous from the anterior chamber percolates into the corneal stroma. Some sort of trauma such as vigorous eye rubbing may be the inciting factor.

Features/signs

Continuous accumulation of the aqueous leads to the separation of the collagen lamellae and the formation of large fluid-filled stromal pockets within the cornea. Meanwhile as a part of the reparative process the adjacent endothelium grows over the defect causing a partial seal so that the seepage is prevented with subsequent resolution of stromal oedema.

Symptoms

Symptoms include sudden onset of vision loss accompanied by pain and foreign body sensation as well as conjunctival injection with diffuse stromal opacity.

Lid Oedema

Uncommon

Bulbar or Limbal Redness

Mild

Differential Diagnosis

Scarring from prior corneal infection, epithelial and stromal corneal oedema, superficial punctate staining, HSV keratitis, Acanthamoeba keratitis.

Treatment

Medical therapy aims at providing symptomatic relief until spontaneous resolution occurs. It includes the use of topical lubricants, antibiotics (to prevent secondary infection), cycloplegics (to reduce pain and photophobia), hypertonic saline eye drops (to help draw fluid), anti-glaucoma medications (to lessen the hydrodynamic force on the posterior cornea), and topical steroids or Nonsteroidal Anti-inflammatory Drugs (NSAIDs).

Sometimes a bandage contact lens may be used to provide pain relief until the oedema subsides or patient is comfortable. Intracameral gas injection act as a tamponade preventing percolation of aqueous and unrolling Descemet's membrane.

According to various studies the resolution of corneal oedema may occur any time between 5 and 36 weeks. After resolution, depending on the location of the eventual scar, the final Best Corrected Visual Acuity (BCVA) can be as good as or better than that achieved by surgical intervention.

BACK TO CONTENTS

ASYMPTOMATIC INFILTRATES (AI)



Aetiology

Some authors believe that Asymptomatic Infiltrates (AI) does not represent a true inflammatory event and it may be a normal occurrence coincidental with contact lens wear.

Mechanism

All contact lenses are inherently inflammatory and AI may be a sign of contact lens induced inflammation.

Features/signs

AI's appear in the cornea with no apparent signs or symptoms. They can be unilateral or bilateral, with daily or extended wear lenses. The infiltrates are typically small and focal (<0.20mm in diameter), mild infiltration may be present but there is no epithelial staining. There is no anterior chamber involvement.

Symptoms

Patients are usually unaware of AI's

Lid Oedema

Uncommon

Bulbar or Limbal Redness

Uncommon

Differential Diagnosis

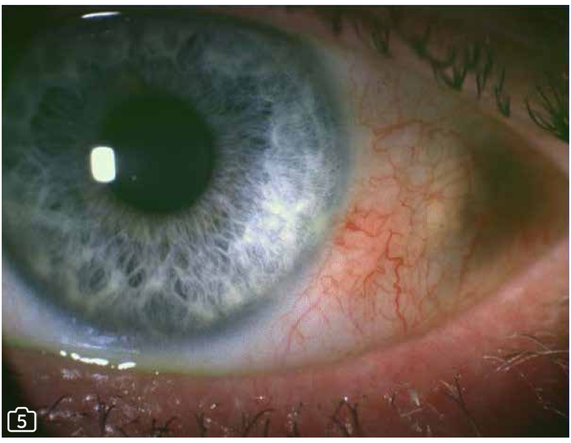
Staphylococcal hypersensitivity and peripheral corneal infiltrates

Treatment

AI should be managed by discontinuing contact lens wear, prescribing antibiotics if needed, and once resolved refitting the patient with high Dk/t lenses with an appropriate lens care system such as hydrogen peroxide. Consider daily disposable lenses. The prognosis of AI is good with full resolution.

BACK TO CONTENTS

ASYMPTOMATIC INFILTRATIVE KERATITIS (AIK)



Aetiology

Asymptomatic Infiltrative Keratitis (AIK) is an inflammatory event characterised by infiltration of the cornea without pain.

Mechanism

It is thought that AIK is the result of the corneas normal protective cellular response to contact lens wear, while others have postulated that it is caused by toxins from gram-negative bacteria.

Features/signs

Although the condition is typically unilateral it can affect both eyes at the same time. It is seen with daily and extended wear lenses. Signs include small focal, often multiple in the peripheral cornea up to 0.40mm in diameter, with or without diffuse infiltration. Punctate staining is often present and there may be mild-to-moderate limbal redness, but there is no anterior chamber reaction.

Symptoms

Patients are often completely unaware of the condition

Lid Oedema

Uncommon

Bulbar or Limbal Redness

Slight to moderate, localised

Differential Diagnosis

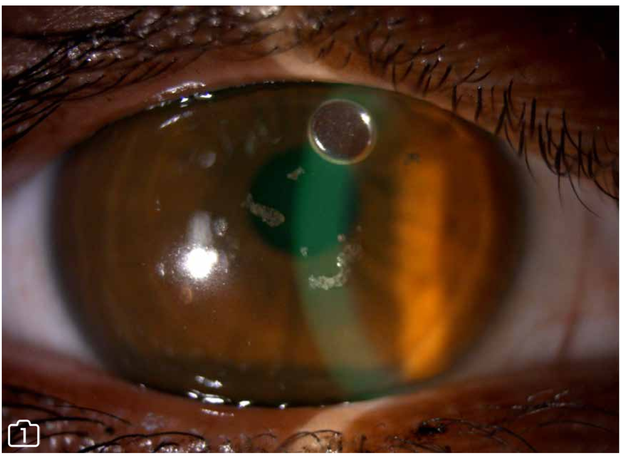
Staphylococcal hypersensitivity and peripheral corneal infiltrates

Treatment

AIK should be managed by discontinuing contact lens wear, prescribing antibiotics if needed, and once resolved refitting the patient with high Dk/t lenses with an appropriate lens care system such as hydrogen peroxide. Consider daily disposable lenses. The prognosis of AIK is good with full recovery.

BACK TO CONTENTS

BUBBLES IN THE TEAR RESERVOIR OF SCLERAL LENSES



Aetiology/mechanism

There are a variety of sources that cause bubbles to occur. Bubbles may be caused by incorrect application techniques or if an aerated solution is used to fill the bowl of the scleral lens. The training and re-training of scleral lens application is critical. If an insertion bubble occurs, it is important to remove and reinsert the lens. If the scleral lens landing zone has misalignment, bubbles may result. Edge lift in one or more quadrants or a lens with excessive movement may allow bubbles to enter.

Features/signs

Single or multiple well-defined bubbles visible in the tear lens reservoir

Symptoms

Scleral lens discomfort may be a result of bubbles trapped in the lens fluid reservoir. If the bubbles are not removed, corneal desiccation can occur leading to visual problems, discomfort and reduced wearing time.

Lid Oedema

None

Bulbar or Limbal Redness

None

Differential Diagnosis

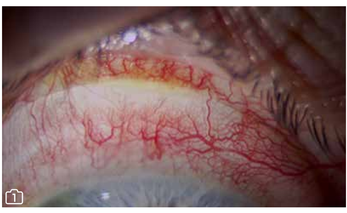
Corneal epithelial staining and dimple veiling

Treatment

Revision of the fit is required to ensure optimal fit on the sclera. Ensure that the patient receives adequate application and removal instructions when dispensing lenses.

BACK TO CONTENTS

BULBAR AND LIMBAL HYPERAEMIA IN SCLERAL LENS WEAR



Aetiology/mechanism

The most likely causes of bulbar redness include infection, mechanical trauma / irritation / blood vessel compression, and inflammation.

Infection

Only a few articles in the limited literature relate to adverse events related to scleral lens wear, such as acute red eye, microbial keratitis, and complications after post-surgery fittings. Specifically, microbial keratitis was related to extended wear, non-compliance, or an eye with severe ocular surface disease. Immunosuppressed patients can also be more at risk than others. Recent review papers reported no significant negative impacts from scleral lens usage.

Mechanical Trauma/Irritation/ Compression

The sclera is a non-symmetrical rotational toric surface. At 15mm, the sclera displays around 1.5D of toricity, which increases up to 5D at 18mm of chord length. Scleral lenses designed with spherical haptics will have meridional lens misalignment, leading to impingement in some quadrants. This triggers a compression of the conjunctival tissue and its vasculature when lenses are steeper than the scleral profile. Clinically, this is visible as a blanching of the conjunctiva where the pressure is present. Blood flow is impinged, causing engorgement before and after this area. This becomes visible as redness outside the lens edge and at the margin of the area where the pressure is exercised.

Currently, it is highly recommended to design lenses with back-toric peripheral curves for any scleral lens prescribed with a diameter of 16mm or larger.

Inflammation

With the increased usage of scleral lenses, inflammation (not related to an infectious event) will probably become more clinically visible. Inflammatory mediators, released from the ocular surface can remain trapped under the scleral lens, which may contribute to raising the inflammatory response. Cellular debris and toxins released from the normal corneal metabolism are also kept trapped under the scleral lenses, knowing that tear exchange is practically absent once the lens is settled. This could be another triggering factor to initiate inflammatory reaction such as overall redness and sterile corneal infiltrates.

Features/signs

Circumlimbal redness, blanching and bearing corresponding to tight zones of the scleral lens periphery

Symptoms

Discomfort and reduced wearing time

Lid Oedema

None

Bulbar or Limbal Redness

Significant hyperaemia may also be sectorial

Differential Diagnosis

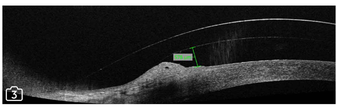
Limbal hyperaemia due to inflammation

Treatment

- Check if lens is not excessively bearing on the circumlimbal area of the sclera
- Increase limbal clearance
- Loosen peripheral curves or use a toric design
- Reduce diameter of the scleral lens
- Flatten and widen the lens landing zone
- Use preservative free saline in the lens bowl for insertion
- Medically treat any significant inflammation before lenses are worn again

BACK TO CONTENTS

CONJUNCTIVAL PROLAPSE





Aetiology

Caused by pressure or fluid forces generated behind a sealed scleral lens and occurs in locations of excessive limbal clearance. More common in patients with Conjunctivochalasis (CCH) and Pellucid Marginal Degeneration (PMD).

Mechanism

While the exact cause of conjunctival prolapse is unknown, it is thought to arise from the pressure or fluid forces generated behind a sealed scleral lens, and often occurs in a location of excessive limbal clearance (a thicker post-lens tear reservoir).

Conjunctival prolapse does not appear to adversely affect corneal function or scleral lens performance in the shortterm (apart from cosmesis), however, potential complications from persistent conjunctival prolapse over long-term lens wear remain unknown (vascularisation or conjunctivalisation of the affected limbal region can occur).

Features/signs

Conjunctiva is seen overlying the cornea under the scleral lens mostly in the inferior cornea. Removal of the lens results in the conjunctiva returning to its normal position, but corneal fluorescein staining may be evident in the affected areas.

Symptoms

Patients are normally not aware of any discomfort and conjunctival prolapse will not affect vision. However, they may notice the prolapse and complain of its cosmetic appearance.

Lid Oedema

None

Bulbar or Limbal Redness

Uncommon

Differential Diagnosis

Conjunctivochalasis, occurring more frequently as patients age and in female patients. It often occurs inferiorly, is related to dry eye disease and is thought to interfere with the lower tear prism, thus delaying tear clearance. It is more problematic in downgaze and when digital pressure is applied; the latter may represent a similar mechanism for conjunctival prolapse. CCH is also frequently seen in contact lens patients, especially RGP lens wearers - the mechanism may be like that of conjunctival prolapse. These mechanisms may include inflammation produced by mechanical stress that leads to friction and/or dryness.

Treatment

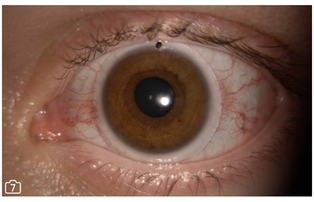
Minimising the vault over the limbal region without creating an area of touch appears to be the best treatment and prevention strategy in susceptible patients. This can be achieved using non-rotationally symmetrical designs, especially in lenses larger than 15.0mm. Prolapse is also associated with PMD due to the typical thin elevated inferior corneal profile.

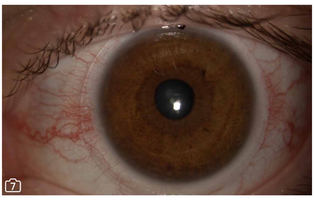
It was also reported that handling the lens with too much pressure can increase the risk of inducing prolapse. Conjunctival prolapse is considered benign, but the real potential for negative outcome in the long term is not known. Considering that the conjunctiva remains draped over the stem cells for many hours, it is probably wiser to alleviate prolapse. One way to accomplish this is to limit the clearance over the limbus by modifying the peripheral curves or selecting a smaller diameter scleral lens. Handling should be revisited as well.

BACK TO CONTENTS

CONTACT LENS ACUTE RED EYE (CLARE)

FORMERLY KNOWN AS TIGHT LENS SYNDROME





Aetiology

An acute inflammatory condition associated with poorly fitting, usually dehydrated, tightfitting extended hydrogel contact lens wear. The literature also recognises poorly fitting and inadequately cleaned scleral lenses as a potential source.

Mechanism

Patients with CLARE will have a history of poor contact lens hygiene, excessive wearing time or frank contact lens abuse. In some cases, the contact lens shows inadequate movement, and the patient may be physically unable to remove the lens from the eye during the initial episode. Immobility of the lens is, however, not uniformly encountered. CLARE may occur with both well-fitted and poorly fitted lenses and may be seen with low or high Dk/t materials, including silicone hydrogels. The hypoxic, pro-inflammatory closed eye environment, endotoxins from gram-negative bacteria and exotoxins from gram-positive bacteria accumulating in the biofilms on posterior contact lens surfaces, are thought to be the cause of CLARE.

Features/signs

A combination of multiple focal infiltrates and diffuse infiltration can be seen in the mid-periphery and periphery of the cornea, generally without any NaFL staining. The diffuse infiltration stems from the encroaching limbal vessels with no clear space between the infiltrates and the limbus, and the infiltrates are restricted to the epithelium and anterior stroma. Associated clinical signs include moderate to severe conjunctival and limbal hyperemia, corneal oedema and mild to moderate blepharospasm. Corneal epitheliopathy and anterior chamber reaction are variable depending upon the severity of the condition and the delay in presentation to the treating clinician.

Symptoms

Patients are typically awakened by their symptoms or they are noticed after awakening. Symptoms include moderate-to-severe circumlimbal redness, irritation, moderate to severe pain, photophobia, and tearing. Vision is affected in the acute phase but recovers quickly. CLARE is often bilateral.

Lid Oedema

Typically absent, however if the superior palpebral conjunctiva is inflamed or the condition has induced eye rubbing it may be present

Bulbar or Limbal Redness

Moderate-to-severe circumlimbal redness

Differential Diagnosis

- CLPU - Contact Lens Peripheral Ulcer
- IK - Infiltrative Keratitis
- AI - Asymptomatic Infiltrates
- AIK - Asymptomatic Infiltrative Keratitis
- MK - Microbial Keratitis

Treatment

- CLARE should be managed by discontinuing contact lens wear, prescribing antibiotics, cycloplegics and in some cases with severe infiltration and intact epithelium, topical steroids
- Once resolved refitting the patient with high Dk/t lenses with an appropriate lens care system such as hydrogen peroxide should be considered. Daily disposable lenses should also be considered
- The prognosis of CLARE is good with most episodes resolving within 3 days, and most infiltrates clear within 7 – 14 days with appropriate treatment
- Complete resolution can take up to 6 weeks. It rarely results in scar formation but CLARE may recur with extended wear

BACK TO CONTENTS

CONTACT LENS INDUCED LID BLEPHAROPTOSIS (CLIP)



Aetiology

CLIP is associated with a number of mechanisms which can be classified as either aponeurogenic (involving some form of dysfunction of the levator aponeurosis) or non-aponeurogenic.

Mechanism

Aponeurogenic

- Forced lid squeezing may cause increased traction of the levator aponeurosis leading to disinsertion or dehiscence
- Lateral eyelid stretching during lens removal and forced blinking can lead to stretching and thinning of the levator aponeurosis
- Rigid lens displacement of the tarsus during removal of the lens can exert pressure on the palpebral conjunctiva and the levator aponeurosis leading to stretching and thinning of the aponeurosis
- Blink induced rubbing can cause lens rubbing against lid structures, displacement of the lid away from the globe, as well as stretching and gradual thinning of the levator aponeurosis

Non-aponeurogenic

- **Oedema –** Constant irritation and trauma to the lid caused by an RGP lens can lead to a sub-clinical inflammation and subsequent oedema. The oedema leads to ptosis due to physical enlargement of the lid in all dimensions and gravity lowers the lid
- **Blepharospasm –** RGP lenses are intrinsically uncomfortable, especially during adaptation, due to the constant bumping of the lens edge against the lid margins. Patients involuntary narrow their palpebral aperture to stabilise the lens and prevent bumping of the lens against the lid margin
- **Papillary conjunctivitis –** Severe papillary conjunctivitis is associated with inflammation, oedema, and ptosis of the upper lids. This is more prevalent in soft lens wearers

Features/signs

Superior lid “droops”. Classified as blepharoptosis when measurement of marginal reflex distance-1 (MRD-1) from central upper eyelid margin to corneal light reflex is less than 2.8mm.

Symptoms

Drooping lid may affect vision, head posture and reduce the superior visual field. Patients may also complain of cosmetic appearance.

Lid Oedema

Possible depending on aetiology

Bulbar or Limbal Redness

None

Unilateral or Bilateral

Can be bilateral or unilateral

Differential Diagnosis

Aponeurotic – (Senile or involutional ptosis) The most common type of acquired ptosis related to aging. This includes disinsertion of the levator aponeurosis

Mechanical – Occurs when the eyelid is too heavy for the muscles to keep it elevated due to reasons such as a mass or excess skin

Myogenic – Unlike aponeurosis, caused by dysfunction of the levator muscle itself, which prohibits the eyelid from being elevated into proper position. This is less likely to be associated with damage to the Müller muscle

Neurogenic – Occurs as a result of dysfunction or damage to the oculomotor or sympathetic nerves or to the central nervous system

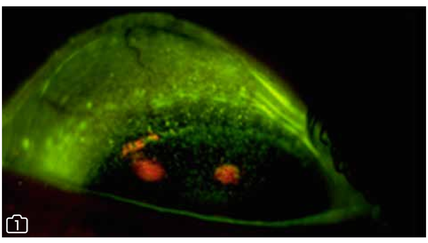
Traumatic – Can encompass aponeurotic, mechanical, myogenic, and neurogenic ptosis that occurs as a result of traumatic damage

Treatment

Management depends on the aetiology. To differentiate between the causes may be difficult but by ceasing lens wear for a period of at least 1-3 months lid oedema, and involuntary blepharospasm can be ruled out. Everting the lid will reveal the presence of papillary conjunctivitis. Consider fitting a scleral or semi-scleral contact lens. If ptosis persists after cessation of lens wear or change to scleral lenses, and the resolution of papillary conjunctivitis the cause is most likely aponeurogenic and surgical intervention may be needed.

BACK TO CONTENTS

CONTACT LENS INDUCED SUPERIOR LIMBIC KERATOCONJUNCTIVITIS (CLSLK)



Aetiology

- Thimerosal hypersensitivity and/or toxicity
- Lens deposits and biofilms on posterior lens surface
- Mechanical irritation
- Corneal/limbal hypoxia under lid
- Immunological reaction

Mechanism

CLSLK is caused by mechanical effects of the contact lens edge with excessive movement, lens deposits and biofilms, sensitivity to cleaning solutions (thimerosal) and hypoxia. Tissues affected include the limbus, bulbar and tarsal conjunctiva with epithelial keratinisation, neutrophilic response and reduced numbers of conjunctival goblet cells.

Features/signs

- Superior limbic vascular injection and oedema
- Microcysts and infiltrates in superior cornea
- Fibrovascular micro-pannus with filament
- Superior sub-epithelial haze extending down in V pattern into cornea
- Papillary hypertrophy
- Irregular thickening of superior bulbar conjunctiva
- Fluorescein, Rose Bengal and Lissamine Green punctate corneal and conjunctival staining
- Corneal warpage and astigmatism

Symptoms

- Contact lens awareness or intolerance
- Foreign body sensation and increased tearing
- Burning, itching and photophobia
- Vision loss with extensive pannus

Lid Oedema

Not Common

Bulbar or Limbal Redness

Radial injection of the bulbar conjunctiva below the superior lid at the limbus

Unilateral or Bilateral

Usually bilateral but not symmetrical

Differential Diagnosis

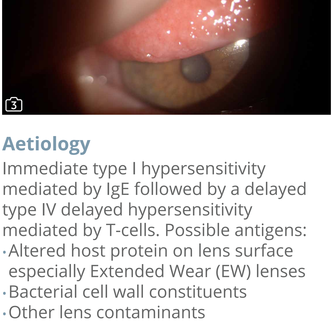
Idiopathic SLK of Theodore, an uncommon, chronic, bilateral, inflammatory disorder which typically affects middle-aged women with thyroid disease

Treatment

- Cease lens wear, refit or change to daily wear lenses
- Change solutions
- Treat MGD and blepharitis
- Aggressive lubrication and punctal occlusion may be helpful In severe cases the use of topical steroids, prostaglandin inhibitors and immunosuppressive drugs such as cyclosporin or tacrolimus may be useful
- Recovery is slow with resolution within 3 weeks to 9 months after cessation of lens wear

BACK TO CONTENTS

CONTACT LENS PAPILLARY CONJUNCTIVITIS (CLPC) / GIANT PAPILLARY CONJUNCTIVITIS (GPC)



Aetiology

Immediate type I hypersensitivity mediated by IgE followed by a delayed type IV delayed hypersensitivity mediated by T-cells. Possible antigens:

- Altered host protein on lens surface especially Extended Wear (EW) lenses
- Bacterial cell wall constituents
- Other lens contaminants

Mechanism

CLPC/GPC is the result of chronic irritation of the tarsus during the 14,000 to 19,000 blinks per day, associated with contact lens edges and surface deposits, prosthesis, or sutures. The hypersensitivity reaction causes degranulation of mast cells and the release of inflammatory mediators which in turn recruit basophils, eosinophils and neutrophils to the conjunctival epithelium.

Features/signs

Polygonal, hyperaemic elevations (papillae) ranging in size from 0.5 to 2mm located in the upper tarsal conjunctiva usually localised to a region of the bulbar conjunctiva in contact with contact lens edge but could also be evenly distributed across the tarsus. Apices of papillae may stain with fluorescein when inflammation is active or appear whitish due to scarring in chronic cases.

Symptoms

Itching and non-specific irritation which may increase after lens removal due to increased mast cell degranulation from mechanical manipulation. Mucus discharge associated with increased lens movement, decreased comfort, lens intolerance, and blurred vision.

Lid Oedema

Not Common

Bulbar or Limbal Redness

Not common but can be present in severe cases

Unilateral or Bilateral

Unilateral or bilateral

Differential Diagnosis

- VKC - Vernal Keratoconjunctivitis
- AKC - Atopic Keratoconjunctivitis
- SAC - Seasonal Allergic Conjunctivitis
- SLK - Superior Limbic Keratoconjunctivitis

Treatment

Non Pharmacological

- Cold compresses and improved lid hygiene – resolve MGD/Blepharitis
- Replace soft lenses more frequently, improve hygiene – more rigorous surfactant cleaning, consider hydrogen peroxide and polish or replace rigid lenses
- Abandon extended wear and reduce daily wearing time to minimum possible. It may be prudent to cease wear for an extended period in some cases

Optimise Lens Fit

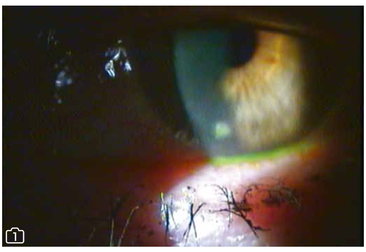
- Rigid lens - alter overall diameter (repositions lens edge relative to tarsus), reduce edge clearance and edge thickness
- Soft lens - change material (silicone to hydrogel) to one with improved deposit resistance, and/or lower modulus or use daily disposable soft lenses

Pharmacological

In mild or moderately severe cases topical mast cell stabilisers or combined anti-histamine/mast cell stabilisers can be used while lens wear continues but preserved drops should not be instilled with soft lenses in situ. Non-preserved lubricant should also be used to improve comfort. In moderate to severe cases that do not respond to other treatment, consider a six-week treatment period of low or moderate strength (preferably "non-penetrating" ester based) topical steroid. Monitor Intraocular Pressure (IOP) at beginning, at two weeks, and at end of treatment period. In recalcitrant cases more potent ketone-based steroids may be needed.

BACK TO CONTENTS

CONTACT LENS PERIPHERAL ULCER (CLPU)



Aetiology

CLPU is a non-infectious self-limiting event probably caused by exotoxins secreted by gram-positive bacteria. All cases of CLPU are related to extended wear and therefore the closed eye environment is also of etiologic significance.

Mechanism

The hypoxic, pro-inflammatory closed eye environment combined with exotoxins secreted by gram-positive bacterial colonies present in biofilms on soft contact lenses cause this inflammatory event.

Features/signs

- Unilateral inflammatory reaction of the cornea characterised by a focal excavation of epithelium, infiltrates, and necrosis of anterior stroma
- A small, round peripheral infiltrate (0.5-1.0mm in diameter), with slight surrounding infiltration is present in the mid-periphery of the cornea
- The region of infiltration may extend from the limbal vessels beyond the location of the infiltrate, often in a triangular pattern
- The infiltration is usually limited to the anterior stroma, but there may be some anterior chamber involvement
- In most cases an erosion with full loss of the epithelium occurs and the area stains brightly with NaFl with some diffusion of the dye into the surrounding tissue
- Bowman's membrane usually remains intact

Symptoms

- Limbal and bulbar redness
- Tearing
- Photophobia
- Moderate-to-severe pain
- Foreign body sensation
- Some may even report seeing a white spot on their cornea

Lid Oedema

Uncommon

Bulbar or Limbal Redness

Moderate, severe in the region orresponding to the focal infiltrate

Differential Diagnosis

- CLARE - Contact Lens Acute Red Eye
- IK - Infiltrative Keratitis
- AI - Asymptomatic Infiltrates
- AIK - Asymptomatic Infiltrative Keratitis
- MK - Microbial Keratitis

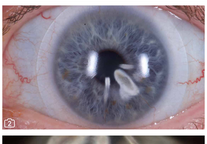
Treatment

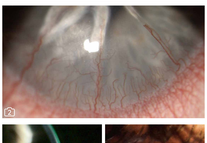
- All the cases of CLPU are related to extended wear and therefore the closed eye environment is also of etiologic significance
- CLPU should be managed by discontinuing contact lens wear, prescribing antibiotics, and once resolved refitting the patient with high Dk/t lenses with an appropriate lens care system such as hydrogen peroxide
- Consider daily disposable lenses
- The prognosis of CLPU is good with most episodes resolving within 7 days, most within 2–3 days with appropriate treatment but it will leave a dense circumscribed scar corresponding to the area of focal infiltrates. The scar has a typical "Bull's eye" appearance

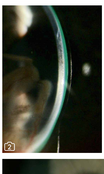
BACK TO CONTENTS

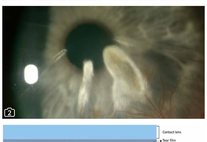
CORNEAL NEOVASCULARISATION (CNV)

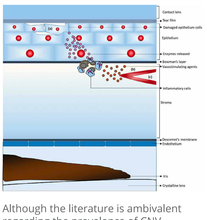










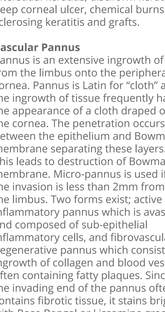


Although the literature is ambivalent regarding the prevalence of CNV in contact lens wear, 10–30% of patients diagnosed with corneal neovascularisation wear contact lenses, while corneal neovascularisation develops in 1-20% of contact lens users.

Aetiology

Stimuli that can promote vessel penetration into the normally avascular cornea includes nutritional, inflammatory, mechanical, traumatic, and toxic factors. One or all these stimuli are present during contact lens wear, particularly overnight or extended wear. The contact lens creates tissue hypoxia which leads to corneal oedema and stromal softening. The lens also contributes to mechanical trauma to the epithelium which results in inflammation and the release of cytokines and prostaglandins which are angiogenic.

Inflammatory cells migrate to the area and release vasostimulating agents that initiate new vessel growth. The cellular distress signal acts directly as triggering agent with the vascular endothelial mesenchyme forming; endothelial cells, pericytes, fibroblasts and the smooth muscle of new blood vessels.



Features/signs

CNV refers to the presence of blood vessels in the normally clear avascular cornea. The normal extent of limbal vessels measured from the limit of the visible iris was 0.13mm inferiorly in non-lens wearers, 0.22mm in RGP wearers and 0.47mm in daily hydrogel lens wearers. In hydrogel extended wear the vascular extent of limbal vessels into the cornea was 0.50–0.52mm.

Superficial Neovascularisation

This is the most common vascular response induced by contact lenses. Normally episcleral branches of the anterior ciliary artery form a plexus around the limbus called the superficial marginal arcade. In contact lens wear, small branches form at right angles to this plexus, encroach the cornea looping inward toward the corneal apex. The vascular loops are semi-circular, they anastomose, each arc becomes smaller forming a rich vascular plexus around the limbus. New vessels often leak and a creamy lipid like fluid can be seen under high magnification around the vessels. Can be extensive and interfere with vision if it encroaches the visual axis.

Deep Stromal Neovascularisation

Contact lenses can induce CNV at any level of the cornea including the stroma, from just below Bowman's membrane to Descemet's membrane. Stromal neovascularisation typically consist of a large feeding vessel that emerges from the limbus and enters the stroma. It develops into finer, tortuous branches that ends in buds with numerous small vessel anastomoses. The vessels are generally derived from anterior ciliary arteries. Deep vessels may be arranged as terminal loops, brush, parasol, umbel, network, or interstitial arcades. If the CNV is extensive enough and lipid leaks into the stroma, vision can be affected. Deep stroma CNV is often associated with other corneal pathology such as Herpes simplex or zoster keratitis, interstitial keratitis, disciform keratitis, deep corneal ulcer, chemical burns and sclerosing keratitis and grafts.

Vascular Pannus

Pannus is an extensive ingrowth of tissue from the limbus onto the peripheral cornea. Pannus is Latin for "cloth" and the ingrowth of tissue frequently has the appearance of a cloth draped over the cornea. The penetration occurs between the epithelium and Bowman's membrane separating these layers. This leads to destruction of Bowman's membrane. Micro-pannus is used if the invasion is less than 2mm from the limbus. Two forms exist; active or inflammatory pannus which is avascular and composed of sub-epithelial inflammatory cells, and fibrovascular or degenerative pannus which consist of an ingrowth of collagen and blood vessels often containing fatty plaques. Since the invading end of the pannus often contains fibrotic tissue, it stains brightly with Rose Bengal or Lissamine green.

CORNEAL NEOVASCULARISATION (CNV)

Symptoms

Discomfort, reduced wearing time and even reduced vision

Lid Oedema

None

Bulbar or Limbal Redness

Common and may be more pronounced in the areas affected by CNV

Differential Diagnosis

CNV is not only caused by contact lenses and is common with corneal infections such as HSV, trauma and corneal inflammatory conditions not related to contact lens wear. Rule out all possible causes other than contact lens wear.

Treatment

- Exchanging the lens with a more oxygen permeable contact lens, changing wearing schedule from extended wear to daily wear, switching to RGP lenses instead of soft lenses, and discontinuing contact lenses in cases of active progressive corneal new vessels are recommended

- Anti angiogenic therapy of the cornea (subconjunctival or intrastromal), as well as corticosteroids and non-steroidal anti-inflammatory agents, can help in cases with active neovascularisation that may endanger the survival of corneal graft or ocular surface health

- Laser photocoagulation of new vessels, photodynamic therapy, electrocoagulation, and stem cell transplant are surgical interventions recommended in severe cases

BACK TO CONTENTS

CORNEAL OEDEMA



Corneal oedema results in an increase in the thickness of the cornea, therefore it is usually expressed as the percentage increase in thickness. An increase of 55µm would equate to ± 10% increase in thickness. Essentially all contact lenses, including silicone hydrogel lenses, induce some level of oedema related to the extent of corneal hypoxia induced by the lens. However, the oedema response of the cornea is not uniform due to:

- Variations in thickness across powered contact lenses
- Resistance of the peripheral cornea to swelling due to limbal vasculature

Aetiology

Although it is typically the corneal stroma that swells, the corneal epithelium can also become oedematous. Different mechanisms are involved with epithelial and stromal swelling.

Epithelial Oedema

It is important to note that epithelial oedema does not occur in response to contact lens induced hypoxia. The aetiology of epithelial oedema is two-fold; first, trauma can cause epithelial cells loss compromising the fluid barrier (zonula occludens) allowing fluid to enter the corneal epithelial layers. Secondly, epithelial oedema follows hypotonic ocular exposure which inhibit the fluid barrier.

Vacuoles

Vacuoles can be observed in up to 10% of the normal non-lens-wearing population. The prevalence of vacuoles is related to lens material. Up to 32% prevalence occur with low Dk/t thick extended wear hydrogel lenses. Vacuoles do not cause vision loss and indicate hypotonic stress rather than hypoxia.

Bullae

Bullae may indicate chronic corneal epithelial oedema and needs to be managed.

Epithelial Microcysts

Microcysts form as a direct effect of chronic hypoxia and acidosis resulting in altered cellular growth patterns. Compared to extended wear of hydrogel lenses, lower prevalence is associated with daily wear hydrogel lenses. Prevalence is nearly 100% with all extended wear hydrogel and low Dk/t RGP lenses and zero with silicone hydrogel lenses.

Epithelial Wrinkling

Several theories exist to explain the cause of epithelial wrinkling. The first propose that excessive elastic force in contact lens wear draw the corneal tissue inwards from the limbus causing the wrinkled appearance.

Mechanism

Epithelial Oedema

Hypotonicity occurs with reflex tearing, RGP lens adaptation, and environment exposure (wind and low humidity) allowing fluid to enter the epithelial layers.

Vacuoles

The mechanism of vacuole formation is like that of epithelial oedema and involves hypotonic stress rather than hypoxia. Vacuoles are common if excess tearing occurs in the adaptation phase of RGP wear due to discomfort.

Epithelial Microcysts

Several theories exist to explain the formation of microcysts: Microcysts represent an extracellular accumulation of broken-down cellular material trapped in the basal layers of the epithelium. Another theory suggests that microcysts represent apoptotic dead cells which either become phagocytosed by living neighbouring cells or remain involuted in intercellular spaces. The latest theory suggests that microcysts form at the deepest level of the epithelium where they are partially formed and difficult to observe. They then migrate to the surface of the epithelium which is constantly growing in an anterior direction. Here they are more readily observed. They eventually break through the surface and leave minute pits that stain with NaFl.

Epithelial Wrinkling

Epithelial wrinkling involves the epithelium and anterior stroma, and it is interesting to observe that in all reported cases of epithelial wrinkling the lens parameters were remarkably similar possibly indicating a physical elastic force generated by the soft contact lens:

- Highly elastic hydrogel materials
- Custom designs
- Extremely thin
- Mid water content (50-55%)
- Steep fitting lenses

Features/signs & Symptoms

A small number of vacuoles and bullae can sometimes be observed in the corneas of contact lens wearers.

Epithelial Oedema and Sattler's Veil
Sattler's veil refers to a diffraction phenomenon arising in the corneal epithelium due to oedema. The extra cellular space increase and is filled with fluid which has a lower refractive index than the cells creating light scatter. This leads to halos and coronas being seen around lights.
It appears that vision loss with physiologically induced levels of corneal oedema is primarily epithelial in origin, with stromal thickness increases up to 10% having little effect on vision. This indicates that the measurement of vision loss is not a good quantitative predictor of the presence or magnitude of physiological corneal oedema.

Vacuoles and Unreversed Illumination
Vacuoles appear to be small spherical bodies within the corneal epithelium (5-30µm in diameter). Bullae are like vacuoles but are irregular in shape (oval) and have indistinct edges. They appear flattened, pebble-like, and can occur as single entities or coalesce into clusters that contain many distinct elements. When viewed using indirect illumination against the dark background of the pupil they display unreversed illumination (distribution of light in vacuole is the same as the background). This is an important feature which distinguishes vacuoles from microcysts. Vacuoles are more commonly seen toward the periphery of the cornea and seldom number more than 10. The fact that vacuoles display unreversed illumination suggests that their contents are probably gaseous and have a lower refractive index than the surrounding tissue. They often disappear without intervention.

Bullae and Reversed Illumination
Bullae display reversed illumination and therefore have a lower refractive index than the surrounding tissue. Due to their indistinct edges this refractive index difference is not as pronounced as in vacuoles indicating that bullae may contain fluid rather than gas.

Epithelial Microcysts and Reversed Illumination
Microcysts are typically 5–30µm in diameter and have a uniform spherical or ovoid shape. They appear as scattered opaque grey dots with focal illumination and as transparent refractile inclusions with indirect retro-illumination usually after 1 week of extended wear, increasing in number and severity as extended wear continues. They display reversed illumination indicating that the refractive index of the microcyst is higher than that of the surrounding tissue. Vision is generally unaffected, and patients are unaware of microcysts except in extreme reactions where they may have a mild anterior uveitis accompanying the reaction.

Epithelial Wrinkling
It is a severe complication of contact lens wear and is characterised by the appearance of a series of deep parallel grooves in the corneal surface – wrinkled effect. Vision loss can be dramatic and proportional to the degree of distortion. Wrinkling is also extremely painful.

Lid Oedema

Uncommon

Bulbar or Limbal Redness

In severe cases of corneal oedema, limbal hyperemia may be present

Differential Diagnosis

Epithelial inclusions

Treatment

Epithelial Oedema and Vacuoles
Epithelial oedema and vacuoles represent a normal adaptation response due to excessive tearing with contact lens wear and should clear up once adaptation is complete.

Epithelial Microcysts

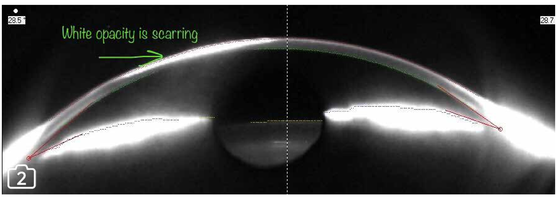
- Epithelial microcysts are managed according to their severity
- Less than Grade 2 no action is usually required, and the patient is monitored carefully. Grade 3 and higher extended wear should be immediately stopped and lens wear discontinued for at least 1 month until the cornea clears
- When refitting the patient daily wear should be recommended in high Dk/t soft and RGP materials
- It is also important to remember that reverse geometry lenses used in orthokeratology induce microcysts within 3 months of wear, therefore these patients need careful follow-up and if microcysts occur reverse geometry lens designs should be avoided

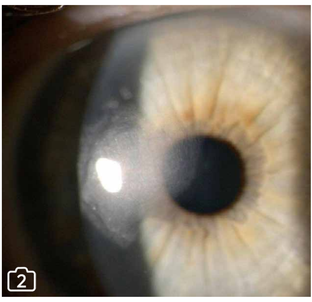
Clinicians should be aware that ceasing contact lens wear may not have an immediate improvement in the microcystic reaction, in fact, the amount of microcysts may increase when the epithelial metabolism normalises before decreasing. This is due to the accelerated growth and mitosis leading to accelerated removal of cellular debris and rapid movement of microcysts to the surface. Numbers of microcysts gradually decrease until they are eliminated.

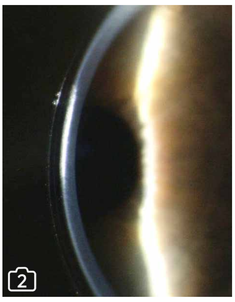
Epithelial Wrinkling
Epithelial wrinkling is managed by ceasing lens wear after which vision should return to normal within 24 hours. The patient can then be refitted with a more appropriate design devoid of inherent elastic forces.

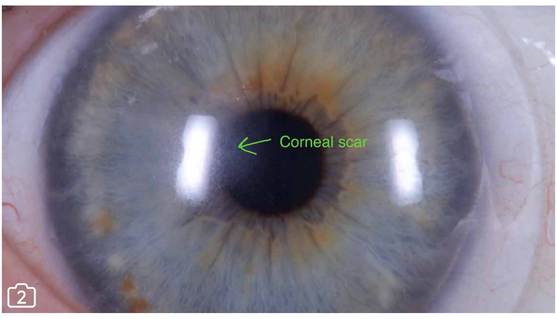
BACK TO CONTENTS

CORNEAL SCARRING
SUPERFICIAL FIBROBLASTIC NODULES OR "PROUD NEBULAS"









Aetiology
Flat fitting RGP's cause a chronic abrasion resulting in corneal scarring

Mechanism
Scars often occur in the area of bearing or mechanical trauma from the lens causing abrasions or "hurricane staining". If left untreated the scars can form "proud nebulas" or Superficial Fibroblastic Nodules (SFN's) which represent chronic abrasions and buildup of scar tissue with each repetitive cycle of abrasion.

Features/signs
Corneal scar over apex of the cone

Symptoms
Discomfort

Lid Oedema
None

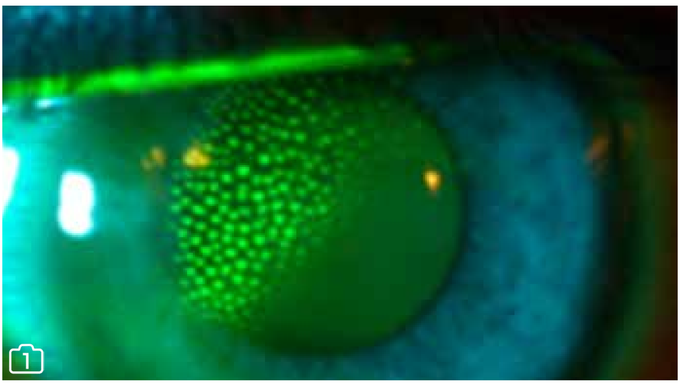
Bulbar or Limbal Redness
Mild

Differential Diagnosis
Scarring from hydrops and corneal ulcers

Treatment
SFN's represent quite a fitting challenge as the goal is to fit the RGP lens away from the heaped-up scar tissue to avoid recurrent abrasions. Semi-scleral or mini-scleral lenses are often used to refit these patients. SFN's can also be surgically removed.

BACK TO CONTENTS

DIMPLE VEILING



Aetiology

This is usually the result of poorly fitting RGP or scleral lenses. It occurs centrally in steep fitting lenses and peripherally in high riding lenses fitted on with-the-rule corneal astigmatism. It does not occur with soft contact lenses.

Mechanism

This is not true staining but rather caused by NaFL pooling in indentations in the cornea left by bubbles that become trapped between a contact lens and the cornea.

Features/signs

Multiple well defined round areas of fluorescein pooling in a specific area of the cornea

Symptoms

If the dimple veiling lies in the visual axis, vision can be affected. It does not cause discomfort

Lid Oedema

None

Bulbar or Limbal Redness

None

Differential Diagnosis

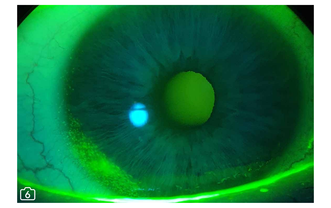
Corneal epithelial staining

Treatment

Refit the contact lens with a more appropriate design for the cornea. In some cases, it may help to fenestrate the lens in the area where dimple veiling occurs.

BACK TO CONTENTS

EFFECT OF CONTACT LENSES ON BLINKING



Blink rate is increased (± 21 blinks per minute) with RGP lenses due to the continual irritation caused by the edge of the lens on the lid margin. This alteration in blink rate is only evident while lenses are worn and returns to normal levels once the lenses are removed. With soft lenses the blink rate is closer to normal but some authors did find an increase in blink rate. The inter blink rate of long term RGP lens wearers is reduced, but the type of blink with RGP and soft lenses is not affected.

Complications of Abnormal Blinking with Contact Lenses

Lens Surface Drying and Deposits
As mentioned previously, the tear film over the contact lens is different than that of the ocular tear film. The lipid layer is thinner or absent, the aqueous layer is of variable thickness, the tear film is less stable, and the TBUT is reduced to between 3 and 10 seconds. This may lead to intermittent drying of the lens surface if the blink rate falls below the TBUT and depending on lens material subsequent deposits on the lens surface.

Visual Degradation
Blink induced lens movement causes a reduction in visual performance that is potentially greater with toric rather than spherical contact lenses. The visual degradation is attributed to the prismatic shift of the retinal image induced by the movement of the lens due to the blink.

Prolonged Lens Settling
Lens settling or degree of post-inserting movement is affected by blink rate. Slower blink rates (<10 blinks per minute) = longer settling times.

Epithelial Desiccation
Can occur with thin high water content lenses but also with high water content hydrogel lenses due to TBU at the inferior tear prism margin Superior Epithelial Arcuate Lesion (SEALS) leading to drying of the lens and mechanical abrasion of the corneal epithelium.

Post-lens Tear Stagnation
Organic material such as desquamated epithelial cells, microorganisms, mucus, proteins, lipids, and inflammatory cells as well as environmental material such as dust, pollen, atmospheric pollutants frequently occur in the tear film. This material is washed away constantly by the tear movement and blink to the lacrimal drainage system. With a contact lens in situ the post lens tear film can stagnate leaving the pollutants in contact with the cornea. This can lead to toxic, allergic, traumatic, and infectious insult to the cornea and inflammation with all its sequelae.

It is estimated that the tear exchange with well fitted RGP lenses is between 10 and 17% per blink, with soft lenses 1% per blink and with scleral lenses 0.2% per minute. Adequate lens movement is essential to avoid tear stagnation, adverse reactions and discomfort.

Tear stagnation in daily wear lenses is of less concern that in extended wear lenses. However if left uncorrected it can lead to the formation of a biofilm on the inside of the lens which results in a host of toxic, infectious and inflammatory or immunological reactions.

Hypoxia and Hypercapnia
Gaseous exchange is enhanced by blinking, lens movement and tear exchange. The distribution of oxygenated tears (tear mixing) is also affected by blinking and lens movement which is especially important when fitting lenses of non-uniform thickness.

3 and 9 o'clock Staining
This is a common problem with RGP lenses and is due to lens-induced disturbance of the normal blink movement of the upper lid over the lens and the cornea. The RG lens bridges the upper lid away from the cornea so that during the blink, the lid is unable to wet the bridges regions of the cornea, typically at the 3 and 9 o'clock positions. This leads to local drying, consequent staining and inflammation.

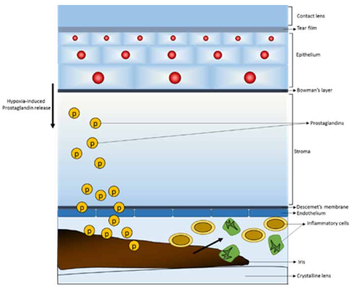
Treatment

Clinicians have two options when dealing with abnormal spontaneous blinking caused by contact lens wear:

- Train patients to modify their blinking activity
- Modify the lens design, type of fit. For instance, changing from an RGP to a soft lens to alleviate 3 and 9 o'clock staining

BACK TO CONTENTS

ENDOTHELIAL BEDEWING



Aetiology

Chronic corneal hypoxia, oedema and mechanical trauma induces a release of prostaglandins and other inflammatory mediators from the corneal tissues. These mediators diffuse into the anterior chamber and aqueous initiating an inflammatory response from the iris/ uveal tissue.

Very uncommon/rare with newer high DK/t lens materials in current contact lens practice

Mechanism

Inflammatory cells are released from the uveal tissue into the aqueous which eventually come to rest on the endothelium where they make their way in between the endothelium cells.

Features/signs

Characterised by the appearance of small inclusions in the region of the inferior cornea near or immediately below the inferior pupil margin, at the level of the endothelium. The condition is usually bilateral.

With retro-illumination the inclusions appear as bright circular optically translucent entities, displaying “reversed illumination.”

With direct illumination, bedewing appears as fine white precipitates or an orange, brown (newly deposited cells are white and longer standing deposited cells orange-brown) dusting of cells at the level of the endothelium.

Other signs are epithelial erosions, epithelial oedema with reduced corneal transparency.

Symptoms

The main associated feature associated with endothelial bedewing is intolerance to contact lens wear and mild conjunctival redness.

‘Fogging’ of vision or stinging have also been reported.

Lid Oedema

None

Bulbar or Limbal Redness

Mild to Moderate

Differential Diagnosis

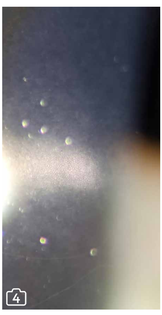
- Keratic Precipitates (KP’s) from uveitis - Fuch’s heterochromic cyclitis
- Krukenberg spindle – pigment dispersion syndrome
- Corneal guttata – not reversed illumination
- Contact lens induced endothelial oedema or blebs – not reversed illumination
- Epithelial microcysts – affects anterior cornea

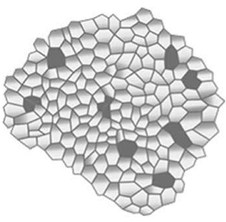
Treatment

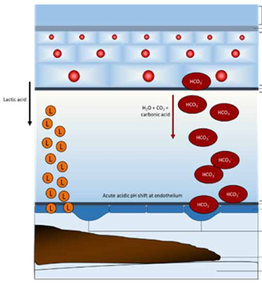
- Reduce wearing time and change to silicone hydrogel material
- Check for concurrent pathology - uveitis
- Check for raised intraocular pressure due to inflammatory cell migrating into anterior angle creating a blockage of aqueous outflow. Perform gonioscopy if IOP is elevated
- Medical treatment may be indicated in severe cases to resolve the uveitis - corticosteroids
- Bedewing can take months rather than weeks to resolve and lens intolerance can persist for many months even after the bedewing has disappeared

BACK TO CONTENTS

ENDOTHELIAL BLEBS







Aetiology

Lens induced corneal hypoxia is the cause of endothelial blebs. The prevalence of endothelial blebs is essentially 100% amongst contact lens wearers and blebs can be observed within 10 minutes of lens insertion.

Mechanism

Acidic pH change in the aqueous humour and endothelium due to an increase in carbonic acid caused by hypercapnia and increased levels of lactic acid induced by contact lens related corneal hypoxia is the cause of endothelial blebs.

Features/signs

Blebs and polymegethism occur mainly with hydrogel soft lenses, uncommonly with RGPs, and are not seen with silicone hydrogel lenses due to the high oxygen permeability of these lenses. The blebs appear as black, non-reflecting areas in the endothelial mosaic, giving the impression that individual cells have fallen off the posterior surface of the cornea, leaving behind holes or gaps in the endothelial layer. With confocal microscopy the bleb appears to have a bright centre spot surrounded by a dark annulus due to the light reflected away from the objective by the bulged posterior surface of the cell. The number of blebs peak within 20–30 minutes and the response subside within 45–60 minutes to a low-level response which can be observed throughout the wearing period.

Symptoms

Patients are usually unaware of blebs and blebs seem to be harmless

Lid Oedema

None

Bulbar or Limbal Redness

None

Differential Diagnosis

Corneal guttata, pseudoguttata, and endothelial denudation

Treatment

The bleb response may indicate that the endothelium has lost its capacity to respond to changes in its immediate environment. Blebs may be linked to polymegethism, the later thought to be a long-term adaptation to the bleb response. The prognosis for recovery from endothelial blebs is excellent. After removal of the contact lenses the blebs will disappear within minutes and they will recur within minutes when lenses are reintroduced. Patients with a bleb response should be refitted with high Dk/t silicone hydrogel lenses.

BACK TO CONTENTS

ENDOTHELIUM POLYMEGATHISM



Aetiology

Endothelial polymegethism is a natural age related change that occurs in all humans and contact lenses have the effect of accelerating such changes. Lenses of extremely high oxygen permeability (silicone hydrogels) do not seem to accelerate these changes which indicate that some measure of hypoxic stress induce a degree of polymegethism and polymorphism. In the normal endothelium of a young adult, the endothelium displays a low degree of polymegethism, the ratio of the smallest cell to the largest cell that can be seen is around 1:5. In advanced cases of polymegethism this ratio can increase to 1:20.

Mechanism

Acute anterior corneal hypoxia leads to filling of potential spaces between cells, straightening the interdigitated lateral cell walls, orienting them obliquely. Chronic anterior corneal hypoxia causes an oblique reorientation of cell walls, smaller anterior cell surfaces and larger posterior surfaces. The shape of the endothelial cell has changed but its volume remains constant.

It seems that the increased carbonic and lactic acid may cause an acidic shift in the pH of the extracellular fluid surrounding the endothelial cells. This may induce changes in the membrane permeability or membrane pump activity that result in fluid movement into the cells, resulting in the elongation of cell walls and changes in cell shape in order to preserve cell volume.

Although the cells showed some inter and intracellular oedema, the cells were otherwise healthy and contained normal organelles.

Features/signs

Blebs and polymegethism occur mainly with hydrogel soft lenses, uncommonly with RGPs, and are not seen with silicone hydrogel lenses due to the high oxygen permeability of these lenses. Observing the anterior surface of the cells on specular reflection it seems that the cells sizes differ considerably while the cells have merely become reoriented in three-dimensional space.

Symptoms

None

Lid Oedema

Uncommon

Bulbar or Limbal Redness

Uncommon

Differential Diagnosis

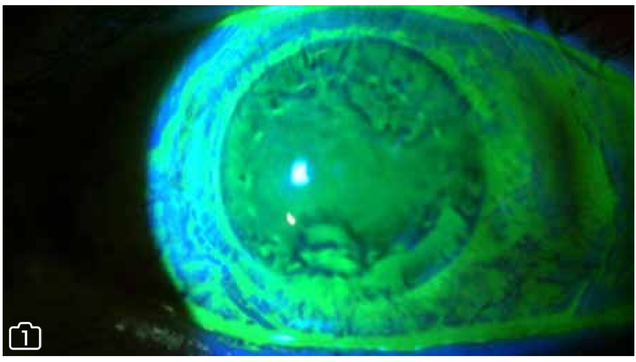
None

Treatment

- The general strategy is to alleviate acidosis by using higher Dk/t lens materials such as silicone hydrogels or RGP lenses
- Lens thickness can be reduced, as well as wearing time (no extended wear), and changing to daily disposable lenses
- The recovery from polymegethism is poor and polymegethism induced by contact lenses is essentially a permanent change unlikely to change back to age-related normality

BACK TO CONTENTS

EPITHELIAL BOGGING



Aetiology

Exact aetiology unknown but it is possibly due to epithelial cells remaining in contact with the stagnant tear film between the lens and the eye becoming waterlogged during lens wear.

Mechanism

The mechanism seems to be related to epithelial oedema and loss of glycocalyx layer leading to surface wetting problems, or alternatively an osmotic imbalance in the stagnant tear film. Interference with normal lid sweeping action over the epithelium may allow non-vital cells to build up.

Features/signs

Corneal epithelium appears rough, irregular, or waterlogged after scleral lens removal

Symptoms

Patients are usually unaware of epithelial bogging. However, if corneal oedema is present, vision may be compromised.

Lid Oedema

None

Bulbar or Limbal Redness

None

Differential Diagnosis

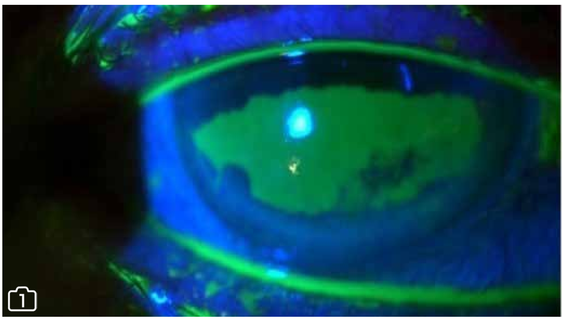
Epithelial oedema and epithelial staining

Treatment

Epithelial bogging is considered benign, but it may be an indication that things are not entirely well. It is recommended that preservative free saline or a cocktail of saline and non-preserved artificial tears should be used in the lens bowl for insertion.

BACK TO CONTENTS

EPITHELIAL PLUG



Aetiology

This is usually caused by severe corneal metabolic compromise due to prolonged lens induced hypoxia.

Mechanism

Many changes occur in the corneal epithelium due to contact lens wear. These changes include decrease in the number of cell layers, appearance of cuboidal rather than columnar basal cell shapes, and a reduction in the number of hemidesmosomes which is the cause of epithelial plug formation.

Features/signs

This is a large discrete area (typically round or oval) of full thickness epithelial loss

Symptoms

Severe discomfort, photophobia, pain, and tearing

Lid Oedema

Depending on severity mild lid oedema can be present

Bulbar or Limbal Redness

Depending on severity, moderate to severe hyperemia can occur

Differential Diagnosis

Corneal ulceration, recurrent corneal erosion, chemical burns, exposure keratopathy, and corneal abrasions

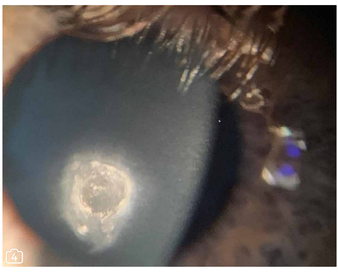
Treatment

- Treat as a corneal abrasion or corneal erosion
- Stop contact lens wear, use cycloplegic drops to alleviate pain and antibiotic drops to prevent corneal infection
- If the epithelial defect is large a bandage contact lens should be used concurrently with antibiotic drops
- Never patch contact lens wearers
- Resolution can take several days and once healed high Dk/t lenses should be considered if patient continues to wear lenses
- Extended wear should be discouraged

BACK TO CONTENTS

FUNGAL KERATITIS





Aetiology

A study by Gray et al., 1995 found that 24% of contact lens cases were colonised by fungi with a majority growing Cladosporium species or Candida species. Other fungi that were also isolated include Fusarium Solani, Aspergillus Versicolor, Exophiala, and Phoma. Most of the fungi contaminants were also found to be associated with bacterial contaminants.

Mechanism

Immune-compromised

Fungal keratitis is frequently seen in immunocompromised hosts (HIV, AIDS), with oral or topical steroid users, and in patients on chemotherapy for cancer. Factors which contribute to fungal contamination of contact lenses include, but are not limited to, hygiene and negligence such as:

- Improper sterilisation and disinfection of contact lenses
- Use of contaminated lenses
- Contaminated contact lens case
- Contaminated contact lens solution
- Wearing of contact lenses during eye infections
- Introduction of micro-organisms from the environment

Features/signs

Fungi are notorious as lacrimal apparatus infectors. Filamentous fungi (Fusarium and Aspergillus species) cause a grey-white serpiginous infiltrates with feathery borders. The epithelium over the infiltrate may be elevated or there may be an epithelial defect with stromal thinning. Non-filamentous fungi (Candida species) cause a yellow-white stromal infiltrate that looks similar to a bacterial ulcer.

Symptoms

Pain, photophobia, red eye, tearing, discharge, and foreign body sensation

Lid Oedema

Common

Bulbar or Limbal Redness

Moderate to severe

Differential Diagnosis

Microbial keratitis, HSV keratitis, and Acanthamoeba keratitis

Treatment

Bacterial and Fungal Contaminants

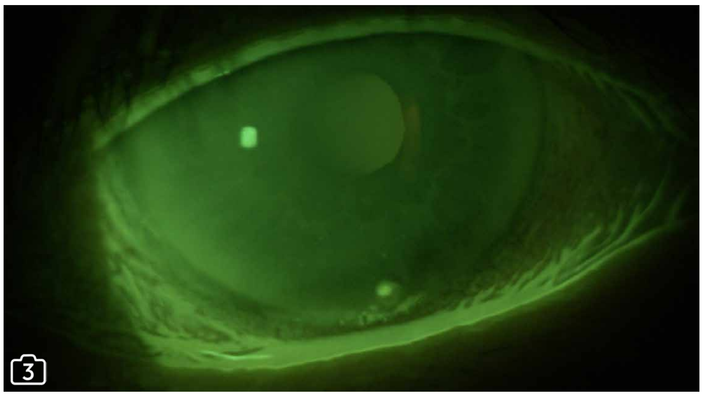
- Cleaning the contact lens case by scrubbing the interior of the case in order to disrupt biofilms
- Rinsing the contact lens case with very hot water, temperatures greater than 70°C which will kill Acanthamoeba contaminants
- Allow contact lens cases to air dry between uses If using hydrogen peroxide as a disinfecting agent, use a two-step system. And lastly, replace contact lens case regularly

Fungal Keratitis

- This involves the use of topical as well as systemic antifungal medication and cycloplegics
- Steroids are not used and if the patient is on steroid medication it should be tapered and discontinued
- Admission to hospital may be required and it may take weeks to achieve complete healing

BACK TO CONTENTS

INFERIOR ARCUATE STAINING OR SMILE STAINING



Aetiology/mechanism

The aetiology includes a combination of factors such as an insufficient post lens tear film, lens adherence, and or lens dehydration which lead to epithelial desiccation. Metabolic causes such as hypoxia may also play a role.

Features/signs

Epithelial staining that occurs on the inferior cornea (1 -2mm from limbus) between 4 and 8 o'clock position. Usually in soft contact lens wearers.

Symptoms

Mild to moderate discomfort when wearing lenses, symptoms worsen with increased wearing time

Lid Oedema

Uncommon

Bulbar or Limbal Redness

Possibly adjacent to affected peripheral cornea

Differential Diagnosis

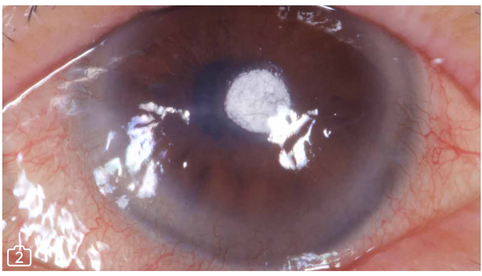
Corneal staining from dry eye and blepharitis

Treatment

- Alter lens fit or type of lens to ensure more movement
- Reduce wearing time until the condition resolves
- Thicker higher gas permeable lenses may also alleviate the problem
- Change from soft to RGP design

BACK TO CONTENTS

INFILTRATIVE KERATITIS (IK)



Aetiology

The cause of IK differs from CLPU and CLARE due to the fact that it is not associated with the closed eye environment, but it is still presumed that toxins released from bacteria adherent to the lens plays a role in the aetiology.

Mechanism

Endotoxins from gram-negative bacteria and exotoxins from gram-positive bacteria accumulating in the biofilms on posterior contact lens surfaces, are thought to be the cause of IK.

Features/signs

IK is a unilateral inflammatory reaction of the cornea characterised by anterior stromal infiltration in the mid-periphery of the cornea, with or without epithelial involvement. Epithelial staining is usually punctate, but it may be severe enough to represent an erosion.

Symptoms

IK is associated with both extended and daily wear lenses, but not sleeping with lenses and its symptoms are therefore rarely reported in the morning. Infiltrates are small and multiple and can be accompanied with mild-to-moderate diffuse infiltration. The infiltrates are typically sub-epithelial and there is no anterior chamber involvement. Symptoms include limbal redness, mild-to-moderate irritation, but rarely pain. A watery or purulent discharge may be present.

Lid Oedema

Uncommon

Bulbar or Limbal Redness

Slight to moderate and localised

Differential Diagnosis

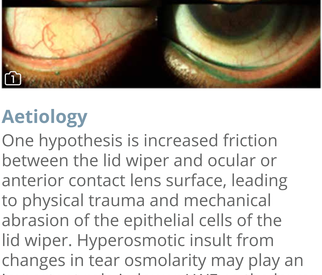
- CLARE - Contact Lens Acute Red Eye
- CLPU - Contact Lens Peripheral Ulcer
- AI - Asymptomatic Infiltrates
- AIK - Asymptomatic Infiltrative keratitis
- MK - Microbial Keratitis

Treatment

- IK should be managed by discontinuing contact lens wear, prescribing antibiotics, and once resolved, refitting the patient with high Dk/t lenses with an appropriate lens care system such as hydrogen peroxide
- Consider daily disposable lenses
- The prognosis of IK is good with most episodes resolving within 14 days, the greater the severity of the infiltrates the longer the recovery
- IK rarely results in scar formation

BACK TO CONTENTS

LID WIPER EPITHELIOPATHY (LWE)



Aetiology

One hypothesis is increased friction between the lid wiper and ocular or anterior contact lens surface, leading to physical trauma and mechanical abrasion of the epithelial cells of the lid wiper. Hyperosmotic insult from changes in tear osmolarity may play an important role in lower LWE as the lower lid wiper is less subject to friction-related damage due to a shorter excursion distance during blinks. There may also be an inflammatory component as upregulation of inflammatory cells have been observed.

Mechanism

In a healthy eye, the tear film, glycocalyx of the cornea, and the conjunctival mucus (secreted mainly by the goblet cells) form a hydrated gel between the lid wiper and the ocular surface to provide lubrication and decrease friction during blinking.

Increased friction in LWE may result from inadequate lubrication due to alterations in the normal tear film by contact lens or other underlying dry eye pathology. Possible alterations include: insufficient mucins, altered composition of mucin at the ocular surface, altered rate of evaporation, and altered lipid layer characteristics. Increased friction may also occur in the setting of adequate lubrication due to increased eyelid pressure or abnormal blinking activity, especially during the complete blink following prolonged interblink intervals from incomplete blinks.

Features/signs

Lid-wiper epitheliopathy is defined as a disruption to the surface epithelium of the lid wiper, a portion of the marginal conjunctiva of the upper and lower lid that acts as a wiping surface to spread the tear film over the ocular surface. It is often observed as staining of the lid wiper area by vital dyes.

Symptoms

The symptoms of LWE are those associated with dry eye, with or without contact lens wear, including but not limited to dryness, grittiness (scratchiness), soreness (irritation), and burning (watering).

Lid Oedema

Uncommon

Bulbar or Limbal Redness

Uncommon

Differential Diagnosis

- Staining of the normal line of Marx should be differentiated from staining of the lid wiper
- Other conditions to consider include lid imbrication syndrome, blepharitis, papillary conjunctivitis, demodicosis, Meibomian gland dysfunction
- Latrogenic lid wiper staining is also possible if care is not taken when manipulating lids during eversion

Treatment

- Reducing lens wearing time
- Increasing lens replacement frequency
- Changing lens material properties
- Changing lens dimensions (thickness, diameter, radius of curvature)
- Altering lens fit

Treatment of the underlying dry eye condition targeted at either the symptoms or aetiology may also be useful in preventing LWE in those who have not yet developed the condition. Topical corticosteroids, oil-in-water emulsion lubricant eye drops, basic fibroblast growth factors, and topical rebamipide.

Insertion of punctal plugs alleviated signs and symptoms of aqueous tear deficient dry eye, including LWE. Other treatments that have been proposed include strategies to improve blinking behaviour, which may be beneficial in LWE associated with incomplete blinking behaviours, as well as improving contact lens wear comfort by altering lens materials or adding Hydra-PEG coatings to lenses. Topical vitamin A ointment may be helpful.

BACK TO CONTENTS

LIMBAL REDNESS



Aetiology/mechanism

Hypoxia – Papas demonstrated a strong inverse relationship between the oxygen transmissibility of contact lenses and limbal redness. The lower the Dk/t, the greater the limbal redness. The mechanism by which hypoxia induces limbal redness is unclear, but Papas proposed the following sequence of events. Hypoxias stimulate the vascular endothelium to release nitrous oxide or prostaglandins. These mediators diffuse toward the smooth muscle cells that compromise the pre-capillary sphincters which relax, resulting in increased blood flow to the hypoxic region.

Infection – Infection of the cornea leads to a cascade of inflammatory events to cause limbal redness. Vasodilating agents (histamine, prostaglandins, and nitrous oxide) are released increasing perfusion. This allows immune cells to approach the site of the infection. The limbal vessel walls increase in permeability and cells as well as fluid pass into the surrounding tissue leading to a milky haze surrounding the engorged limbal vessels.

Inflammation – An increase in the inflammatory cell population occurs overnight in the conjunctival sac, this is why our eyes appear red upon awakening. Contact lenses can alter the concentrations of inflammatory mediators in the tear film which may explain why patients who wear lenses on an extended wear basis are more prone to contact lens acute red eye.

Trauma – The constant physical presence of a contact lens on the limbal area may release inflammatory mediators which result in limbal redness.

Solution toxicity or hypersensitivity – Solution preservatives may cause a delayed hypersensitivity reaction or act directly on the pre-capillary sphincters or the vessel walls, causing vessel distention and limbal redness.

Features/signs

When the limbus is physically or physiologically stressed, the normal anterior limbal loops of the limbal vascular plexus and the terminal arcades dilate making the vessels much more visible giving rise to limbal redness.

Symptoms

The development of limbal redness is not associated with any subjective symptoms, however patients with severe complications and coincidental limbal redness will suffer from discomfort and pain.

Lid Oedema

None

Bulbar or Limbal Redness

Mild to severe depending on the aetiology

Differential Diagnosis

- Corneal neovascularisation
- Pannus
- Contact lens-induced superior limbic keratoconjunctivitis
- Prominent palisades of Vogt which may appear like a prominent plexus of limbal vessels

Treatment

Limbal redness is harmless and does not cause discomfort for the lens wearer. However, it is an important sign of ocular distress and an indicator that action should be taken. The severity, extent, and whether it is chronic or acute will often give clues to its cause.

A useful strategy to use when examining patients with limbal redness would be to consider whether the limbal redness is acute, chronic, localised or circumlimbal. Acute local limbal redness is most probably due to keratitis near to the region of limbal redness. This requires aggressive treatment with anti-inflammatory and anti-infective drugs.

Chronic local limbal redness is most probably due to a defect in the edge of a RGP or soft lens and should be easily dealt with.

Acute circumlimbal redness is most likely due to a solution immediate hypersensitivity or toxic reaction and the solutions should be changed.

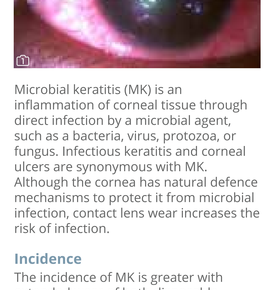
Chronic circumlimbal redness is most likely the result of chronic hypoxia induced by a contact lens which requires replacement with a high Dk/t lens.

Recovery once the causative agent is removed is rapid and limbal redness will generally completely resolve within 2-7 days.

BACK TO CONTENTS

MICROBIAL KERATITIS (MK) OR CORNEAL ULCER





Microbial keratitis (MK) is an inflammation of corneal tissue through direct infection by a microbial agent, such as a bacteria, virus, protozoa, or fungus. Infectious keratitis and corneal ulcers are synonymous with MK. Although the cornea has natural defence mechanisms to protect it from microbial infection, contact lens wear increases the risk of infection.

Incidence

The incidence of MK is greater with extended wear of both disposable and conventional hydrogel lenses, in comparison to silicone hydrogel and RGP lenses For daily wear of RGP lenses, the incidence ranges from 0.4 – 4 per 10000 patients per year and 0.2 per 10000 with extended wear of RGP lenses.

For patients wearing silicone hydrogel lenses on an extended wear basis, the incidence is 0.53 per 10,000.

Guillion found a much higher incidence of MK per 10,000 daily soft lens wearers in the UK, 39 for daily conventional soft lens wearers and 18 for disposable lens wearers. Using this data from Guillion as well as that of other authors one can estimate that the average contact lens practitioner who sees 10 conventional soft lens patients per week will see at least 2 cases of MK per year.

Aetiology

- Presence of microorganisms in the biofilms that exist on the lenses
- Mechanical and hypoxic trauma caused by the lenses which compromise the corneal defences against infection
- Inefficiency of contact lens solutions
- Patient non-compliance
- Poor lens case care
- Poor patient hygiene
- Swimming with lenses

The initial step in the pathogenesis of MK with hydrogel extended wear lenses involves the colonisation of the contact lens with microbes and the formation of a biofilm.

Organisms such as Pseudomonas and Acanthamoeba adhere to hydrogel lenses. Acanthamoeba readily attaches to silicone hydrogel lenses. These organisms can adhere to, damage or invade the epithelium and replicate in the corneal stroma. This leads to tissue inflammation, destruction and a dense corneal infiltrate with overlying epithelial compromise.

Mechanism

- Microbial adherence to contact lens
- Epithelial trauma
- Microbial adherence to corneal epithelium
- Replication and invasion of corneal layers
- Release of toxins and enzymes
- Activation of immune reaction
- PMNs infiltration
- Dense corneal infiltrate
- Release of enzymes and toxins into the corneal tissue
- Collagen lysis and suppuration

Features/signs

Characterised by a focal white infiltrate in the corneal stroma. If the infiltrate is accompanied by an overlying epithelium defect that stains with fluorescein, it is deemed a corneal ulcer.

Mucopurulent discharge with stromal oedema, anterior chamber reaction with or without hypopyon formation, conjunctival injection, corneal thinning, folds in Descemet membrane, upper eyelid oedema, hyphaemia and raised IOP may occur depending on the severity of the infection.

Symptoms

Red eye, moderate to severe ocular pain, photophobia, decreased vision, discharge, and acute contact lens intolerance

Lid Oedema

Usual, severe blepharospasm may be present

Bulbar or Limbal Redness

Severe, conjunctival chemosis may be present

Differential Diagnosis

Fungal keratitis, HSV keratitis, Acanthamoeba keratitis, staphylococcal hypersensitivity and peripheral corneal infiltrates, sterile corneal infiltrates, corneal scars from prior injury, and neurotrophic corneal ulcers from topical anaesthetic or crack cocaine use.

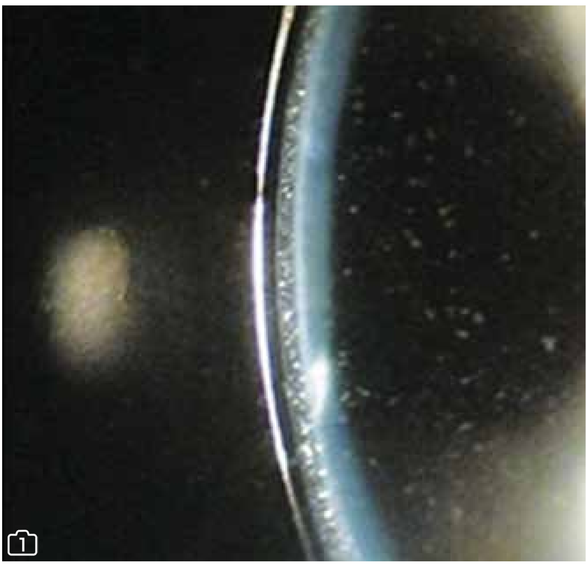
Treatment

Always remember that inflammation is worse in bacterial keratitis due to the release of exotoxins with replication and endotoxins after cell death – which causes ring infiltrates. Enzymes, collagenases, coagulase, proteases, nucleases, lipases, elastase, fibrinolysins and haemolysins are also released.

Microbial keratitis is a medical emergency and should be treated aggressively using fortified topical antibiotics or newer generation fluoroquinolones, topical cycloplegic drugs as well as topical steroids to prevent significant scarring. In severe cases patients are hospitalised to ensure compliance with treatment and to prevent vision loss.

BACK TO CONTENTS

MIDDAY FOGGING IN SCLERAL LENS WEAR



Aetiology

Related to lens induced mechanical trauma to conjunctiva/limbus/cornea, hypoxic inflammation and meibomian gland lipids accumulating in the tear reservoir. Corneal oedema due to hypoxia can also contribute.

Mechanism

Increased mucin production from rubbing the conjunctival tissue can also contribute to accumulation of protein and lipid on the lens surface.

Features/signs

Particulate matter in the post-lens tear film associated with blurred vision

Symptoms

Blurred vision and discomfort after a few hours of lens wear. Patients often remove and reinsert scleral lenses when this occurs. This may exacerbate the problem.

Lid Oedema

None

Bulbar or Limbal Redness

None

Differential Diagnosis

Corneal oedema

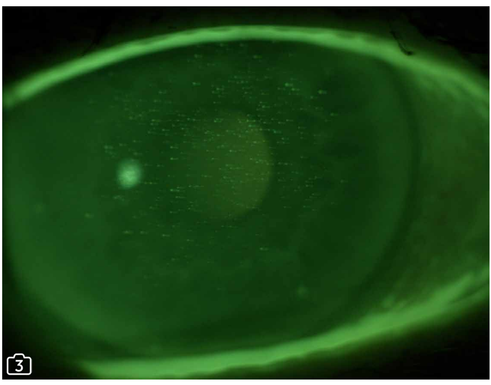
Treatment

Rule out obvious causes and adjust the lens fit to reduce ingress of material into the tear reservoir.

Using non-preserved saline mixed with thick preservative free artificial tears when inserting the lens can also help to alleviate midday fogging.

BACK TO CONTENTS

MUCIN BALLS



Aetiology

They are typically observed with lens materials of higher modulus or stiffness

Mechanism

Stiffer high Dk silicone hydrogel lenses may exacerbate the mechanical interaction of the lens with the ocular surface creating shear and surface tension forces within the tear film. These forces cause the tear film (mucin layer) to roll into beads as the lens moves over the ocular surface.

Features/signs

Measuring approximately 40 to 120µm in diameter, mucin balls are relatively large, generally spherical bodies that we easily observed with the biomicroscope under direct white light illumination, indirect and retro-illumination.

Mucin balls remain motionless under the lens, even during blinking, and indent the corneal epithelial surface during contact lens wear. After removal of the lens the mucin balls dislodge, and fluorescein will pool in the depressions.

Symptoms

Mucin balls form within minutes of lens insertion but appear to have no adverse effects on ocular health. They do not affect vision and comfort, and they are not associated with any adverse reactions.

Lid Oedema

None

Bulbar or Limbal Redness

None

Differential Diagnosis

Epithelial vacuoles and microcysts - less visible, smaller in size and require retro-illumination to be viewed. Vacuoles and microcysts do not stain with fluorescein.

Treatment

- Indentations resolve spontaneously between 30 minutes and several hours after lens removal
- Optimise the lens fit, flat fitting lenses are thought to exacerbate mucin ball formation
- Advise the patient to use lubricating drops after waking and before sleep
- Suggest a shorter wearing schedule, daily wear rather than extended wear

BACK TO CONTENTS

PSEUDOMONAS ULCER



Aetiology

Pseudomonas Aeruginosa is a common gram-negative, rod-shaped bacterium that can cause disease in plants and animals, including humans. A species of considerable medical importance, *P. aeruginosa* is a multidrug resistant pathogen recognised for its ubiquity, its intrinsically advanced antibiotic resistance mechanisms, and its association with serious illnesses. It is citrate, catalase, and oxidase positive. It is found in soil, water, skin flora, and most man-made environments throughout the world. It thrives not only in normal atmospheres, but also in low oxygen atmospheres, thus has colonised many natural and artificial environments. It uses a wide range of organic material for food; in animals, its versatility enables the organism to infect damaged tissues or those with reduced immunity.

Two types of *P. aeruginosa* cause clinical disease and the pathogenesis of the two types are entirely different. One type (invasive strain) invades the corneal epithelial cells without killing them and therefore causing the disease largely through the host cell immune response. The other type is cytotoxic (cytotoxic strain) for corneal and other epithelial cells, killing the hosts cells. Corneal oedema due to hypoxia can also contribute.

Mechanism

Because it thrives on moist surfaces, this bacterium is also found on and in medical equipment, including catheters and contact lenses, causing cross-infections in hospitals and clinics. *P. aeruginosa* is not extremely virulent in comparison with other major pathogenic bacterial species – for example *Staphylococcus aureus* and *Streptococcus pyogenes* – though *P. aeruginosa* is capable of extensive colonisation, and can aggregate into enduring biofilms. Biofilms seem to protect these bacteria from adverse environmental factors. *P .aeruginosa* produces glycocalyx associated with bacterial adhesion, persistence and survival in infected tissues and on surface of contact lenses. The glycocalyx also protects the pathogen from immune cells and antibiotics. *P. aeruginosa* can survive over wide temperature range, 10 - 42°C and even grow in an anaerobic environment rich in nitrates. Epithelium damage must be present for *P. aeruginosa* to infect the cornea.

Features/signs

It is an opportunistic pathogen that causes a rapidly spreading, severely destructive corneal ulcer. However, surface damage of the epithelium must be present for *P. aeruginosa* adherence and infection. Exotoxins and endotoxins are liberated and inhibit protein synthesis similar to the diphtheria toxin. Rapid death of epithelium, stroma and endothelium cells and necrosis results. The ulcer spreads rapidly and perforation of the cornea can occur within 2 to 5 days. The cornea has a diffuse ground glass appearance and copious mucopurulent discharge with a greenish colour adheres to the ulcer surface. An anterior chamber reaction and hypopyon is usually is present. Ring infiltrates are common and a necrotising enzyme, proteoglycanase, rapidly causes stromal destruction with descemetocele.

Symptoms

The symptoms of such infections are generalised inflammation and sepsis. If such colonisations occur in critical body organs, such as the lungs, the urinary tract, and kidneys, the results can be fatal.

Lid Oedema

Usual, severe blepharospasm may be present

Bulbar or Limbal Redness

Severe, conjunctival chemosis may be present

Differential Diagnosis

- Fungal Keratitis
- HSV Keratitis
- Acanthamoeba Keratitis
- Staphylococcal Hypersensitivity and Peripheral Corneal Infiltrates
- Sterile Corneal Infiltrates
- Corneal Scars from prior injury
- Neurotrophic Corneal Ulcers from topical anaesthetic or crack cocaine use

Treatment

A pseudomonas aeruginosa corneal infection (microbial keratitis) is a serious and aggressive condition requiring urgent treatment, approaches include:

- **Empiric Therapy** (Immediate Intensive Antibiotic Treatment) including fortified antibiotics for severe cases or high resistance risks.

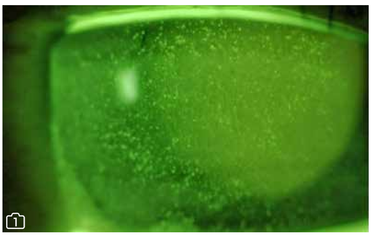
- **Adjunctive Therapy** such as cycloplegics to relieve pain and prevent synechiae. Oral antibiotics if Scleral involvement or Orbital Cellulitis suspected. The use of corticosteroids is controversial and considered only under ophthalmologist supervision and after 48+ hours of antibiotic therapy.

Monitoring and Follow up
Daily follow-up initially to assess response. If no improvement in 24-48 hours, consider modifying antibiotics based on culture results. Severe cases may require hospitalisation, intrastromal injections, or surgical intervention (e.g. corneal transplant).

Prevention of Recurrence
Cease lens wear until fully healed, review hygiene and address underlying factors.

BACK TO CONTENTS

SOLUTION SENSITIVITY



Aetiology

If patients develop eye irritation after months or more of symptom free contact lens wear, solution sensitivity should be suspected. Such delayed hypersensitivity reactions were more common with earlier cleaning and soaking solutions that contained the mercury-based preservative thimerosal, but it has also been seen with solutions containing other preservatives, such as chlorhexidine and benzalkonium chloride.

Even a nonallergic person who is subjected daily to any form of chemical, solution, or eyedrop has the potential for developing a delayed hypersensitivity reaction. Patients with a history of atopy, allergy, eczema, or prior contact lens–related problems will be more easily triggered into sensitivity or an allergic reaction.

Mechanism

Type 4 hypersensitivity reactions to protein antigens are delayed and take up to 12 hours to develop. Unlike other forms of hypersensitivity, type 4 cannot be transferred by serum but can be transferred by T cells that have become sensitised to a particular antigen. These cells act in concert with other cell types that have been recruited to the site of the reaction. Examples of type 4 delayed hypersensitivity reactions include ocular allergy, corneal graft rejection, cosmetic-induced conjunctivitis, idiopathic uveitis, and sympathetic ophthalmia.

Features/signs

Moderately red, constantly weepy, itchy, and irritated eyes. The hypersensitivity reaction is diffuse and involves bulbar and palpebral conjunctiva, with or without a follicular reaction. Fluorescein staining of the cornea consists of diffuse fine punctate staining more concentrated in the corneal periphery than centrally.

Symptoms

- Moderately red
- Constantly weepy
- Itchy, and irritated eyes with or without accompanying vision problems
- Patients may report that the eyes are more comfortable when fresh lenses are inserted but recur after using the lenses and solution for a few days

Lid Oedema

Uncommon

Bulbar or Limbal Redness

Mild to moderate

Differential Diagnosis

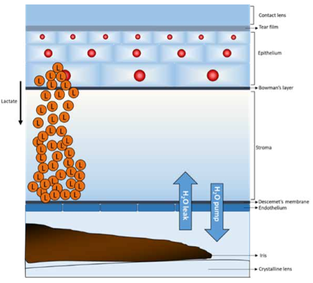
- Contact Lens Papillary Conjunctivitis (CLPC)
- Giant Papillary Conjunctivitis (GPC)
- Allergic conjunctivitis

Treatment

Whatever the source of irritation, the first step is to have the patient temporarily cease contact lens wear while gaining control of the reaction. If infiltration or ulceration is apparent, topical antibiotics may be in order, avoid steroids in case a bacterial or viral infection is involved. Mild topical steroids, mast cell stabilisers and decongestant drops may also be useful at this can be used once bacterial or viral infections are ruled out. Once the eye is quiet, switch to an MPS with different ingredients or to a hydrogen peroxide– based regimen. A change of contact lens material or regimen may also help.

BACK TO CONTENTS

STROMAL OEDEMA



Aetiology

Contact lenses restrict the amount of oxygen available to the corneal epithelium and stroma causing a hypoxic environment.

Mechanism

Anaerobic respiration in the epithelium results in lower amounts of energy and more importantly excess lactate. The lactate moves into the corneal stroma which is then osmotically balanced by the influx of water from the anterior chamber into the stroma. The lack of oxygen and aerobic metabolism results in lack of energy to maintain the endothelial pump-mechanism. This results in corneal oedema and loss of corneal transparency.

Features/signs

Central Corneal Clouding (CCC)

Corneal oedema associated with tight fitting PMMA lenses is known as central corneal clouding (CCC). A round area of clouding forms under the lens in the central cornea. CCC indicates a gross level of oedema which is rarely observed with modern contact lenses and materials. More subtle signs of oedema can be seen with careful slit lamp examination of the cornea and include striae, folds, and haze.

Striae

Striae appear as fine wispy, white vertically oriented lines in the posterior stroma using direct focal illumination. You can also visualise them as dark lines in the red fundus reflex using retro illumination. Striae are present when oedema reaches at least 5% and they do not cause vision loss. Striae represent fluid separation of the predominantly vertically oriented collagen fibres in the posterior stroma.

Folds

Folds can be observed in the epithelial mosaic as compressed grooves or raised ridges, or as areas of buckling when the level of oedema ≥ 8%. Folds are best observed using specular reflection. Folds are caused by physical buckling of the posterior stromal layers in response to high levels of oedema.

Haze

At about 15% oedema the stroma takes on a milky appearance due to the loss of transparency. The haze appears as a fine grey haze against the dark background of the pupil using indirect illumination. Sclerotic scatter enhances the clinical picture. Stromal haze can affect vision especially if the oedema exceeds 20%. Haze is a more advanced form of striae with gross separation of collagen fibres throughout the full thickness of the stroma. This causes failure of the optical coherence of the stromal lamellar layers, reducing transparency.

Symptoms

Depending on severity can cause loss of vision, discomfort, and poor contact lens tolerance

Lid Oedema

Uncommon

Bulbar or Limbal Redness

Not common but can occur depending on the severity of the physiological compromise

Differential Diagnosis

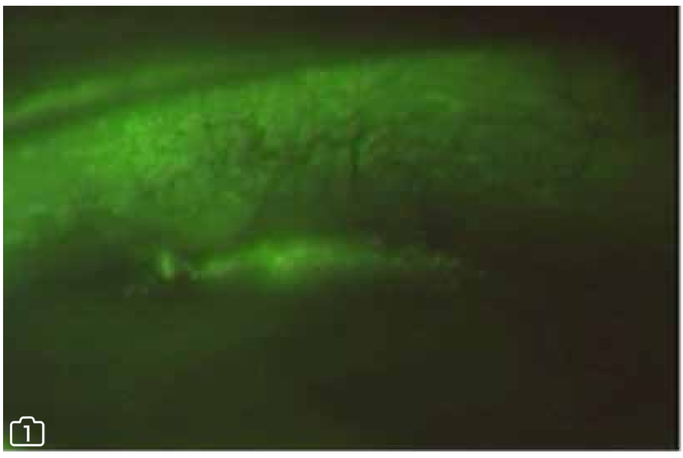
Epithelial oedema, corneal haze from surgery and oedema from infectious causes

Treatment

- Increase Dk/t of the RGP lens material
- Reduce the RGP lens thickness
- Address RGP lens fit, use flatter base curve, increase the edge lift, reduce lens diameter, and or add a fenestration to the lens
- With soft lenses change from hydrogel to silicone hydrogel material, or from daily wear to disposable monthly or daily disposable lenses
- Reduce wearing time (no extended wear), postpone, or abandon lens wear

BACK TO CONTENTS

SUPERIOR EPITHELIAL ARCUATE LESION (SEAL) OR EPITHELIAL SPLITTING



Aetiology/mechanism

Infrequent, asymptomatic complication of conventional soft lens wear. It is also referred to as epithelial splitting and its aetiology is multifactorial. Mechanical chafing because of the upper lid resting on the lens resulting in excessive frictional pressure and abrasive shear forces on the corneal epithelial surface. Higher modulus lens materials such as silicone hydrogels can contribute to the formation of SEAL's.

Features/signs

This is usually a full thickness lesion of the epithelium in the cornea covered by the superior lid, within 2–3mm from the limbus in the 10 to 2 o'clock region.

Symptoms

Usually, asymptomatic

Lid Oedema

Uncommon

Bulbar or Limbal Redness

Possibly in the affected area

Differential Diagnosis

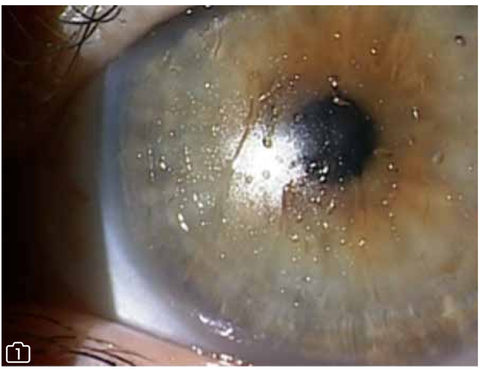
Contact Lens Induced Superior Limbic Keratoconjunctivitis (CLSLK)

Treatment

- Alter lens design to lens with less mid-peripheral bearing
- Alter lens type to lower modulus material and better surface characteristics

BACK TO CONTENTS

SURFACE SOILING POORLY WETTING SCLERAL LENSES



Aetiology

Mucoid, lipid, or protein deposits are normally the reason for poor wetting. These deposits can be caused by untreated OSD or external factors such as makeup, face creams, and hand soaps.

Mechanism

On average, most people blink around 15 to 20 times each minute, 900–1.200 times an hour or 14.400–19.200 times a day. Abrasion, inflammation, and poor spread of the tear film over the lens surface can cause lid wiper epitheliopathy which contributes to inflammation, tear layer changes and poor wetting of the lenses. The lipid and mucin layers of the tear film and the conjunctival goblet cells are affected by the lens which alters the mucin layer. Add to this MGD, dry eye, blepharitis, atopy, hormonal changes, medication, high DK/t silicone-based materials and the result is lipid deposits and poor wetting of the lens surface.

Features/signs

Dry spots forming on anterior surface of the lens within minutes of inserting the lens

Symptoms

Discomfort, poor vision, frequent lens removal and cleaning

Lid Oedema

Uncommon

Bulbar or Limbal Redness

Mild in some cases where patients remove lenses frequently

Differential Diagnosis

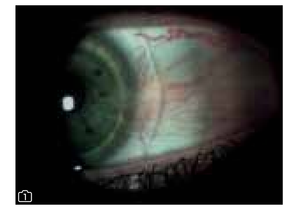
Other lens deposits such as protein and mucus deposits

Treatment

Encourage patients to apply makeup and face creams after inserting the lens and only use mild, moisturiser-free soaps to wash their hands. Material choice also plays a role in proper wetting, so finding the appropriate balance between oxygen permeability and wetting angle is imperative. Additionally, treating the lens with plasma and Tangible Hydra-PEG can enhance wettability and decrease deposits. Finally, practitioners should encourage patients to gently rub the front surface with nonabrasive cleaners to further reduce deposition.

BACK TO CONTENTS

VASCULARISED LIMBAL KERATITIS (VLK)





Aetiology/mechanism

Vascularised Limbal Keratitis (VLK) is a complication of corneal RGP lens wear (both medium and high gas permeable materials). It is characterised by an inflammation of the limbus in association with a process of vascularisation in the 3 and 9 o'clock cornea positions.

The aetiology is unknown but Grohe and Lebow, 1989 hypothesised that VLK is caused by an interruption of the normal tear film dynamics at the limbus caused by the corneal RGP lenses.

Constant ongoing physical irritation of the poorly lubricated ocular surface by the RGP lens and lids induces inflammation which progress to the stages of VLK if left untreated. VLK is can therefore be seen as a more severe form or at least the result of 3 and 9 o'clock staining caused by corneal RGP lenses.

Features/signs

VLK typically develops over a period of 6–24 months. It can be graded according to severity into four stages, stage 1 being the least severe.

Stage 1
Epithelium adjacent to the limbus appears disrupted and punctate staining is evident. An elevated whitish opaque mass of hyperplastic corneal and limbal epithelial tissue can be observed at the 3 and 9 o'clock corneal locations with ill-defined borders. The mass appears to bridge from the conjunctiva across the limbus onto the cornea and the tear film meniscus is absent or disrupted. The patient typically experiences no symptoms.

Stage 2
This stage is characterised by symptoms such as mild discomfort, lens intolerance and conjunctival redness. Corneal infiltrates may be present, and staining is more severe.

Stage 3
Symptoms of discomfort reduced wearing time, conjunctival and corneal staining, and conjunctival hyperemia as well as a more significant infiltrative response is evident. The limbus and conjunctiva may appear to be oedematous, and it is not uncommon to see a vascular leash emanating from the conjunctiva encroaching upon the hyperplastic mass.

Stage 4
This stage is characterised by severe discomfort and photophobia as well as pain when the lens edge impinges on the hyperplastic mass and wearing lenses become intolerable. Significant conjunctival hyperemia and staining present, often associated with erosion of the elevated hyperplastic mass. Superficial and deep vascularisation is common.

Symptoms

Depending on the stage, early on patients may have no symptoms, but as the condition progress they will experience mild to severe discomfort and photophobia.

Lid Oedema

Uncommon

Bulbar or Limbal Redness

Sectorial from mild to severe

Differential Diagnosis

Phlyctenulosis, peripheral corneal ulceration, pterygium, pannus, and pinguecula

Treatment

Management depends on the severity of the condition. At the very least the lens design should be altered, wearing schedule adjusted, lubricating drops prescribed, and punctal plugs considered.

In the more severe cases lens wear should be suspended and medical treatment instituted. Corticosteroid antibiotic combinations should be considered if the inflammatory response is severe and soft or scleral lenses may be considered after the inflammation has resolved to avoid a repeat episode.

Prognosis for recovery of VLK is generally very good and even severe cases can recover within a few weeks with appropriate treatment. However, if lens wear recommences prematurely, a rebound can occur whereby the condition flares up and progresses rapidly to the previous stage.

BACK TO CONTENTS

DR DIRK JOHAN BOOYSEN



Dirk Booysen was educated at Monument High School, Witwatersrand Technicon, Pennsylvania College of Optometry, London Refraction Institute, New England College of Optometry, State University of New York and Aston University. He holds a Doctorate from Aston University as well as Fellowships and Memberships from the British College of Optometrists, Scleral Lens Society, International Society of Contact Lens Specialists, and South African Optometric Association.

Dirk practices full time and lectures part time. He represents the profession of optometry internationally as a member of the WHO's Development Group on Glaucoma Interventions and locally as a member of the Aeromedical Committee of the South Africa Civil Aviation Authority (SACAA). He has received numerous awards including the:

- South African Distinguished Service award for Exceptional Contribution to the Profession of Optometry
- Certificate of Tribute from the South African Optometric Association
- World Top 100 Doctor to Doctor Award for exemplifying leadership, excellence and entrepreneurship in advancement of the healthcare industry
- Optometrist of the Year in the Healthcare & Pharmaceutical Awards 2022, hosted by Global Health & Pharma
- South African Optometric Association Centenary Celebration Award for Academic Excellence in 2024

Dirk has published many academic papers as well as two clinical text books; *"In Contact, Clinical Contact Lens Practice and The Southern African Guide to Topical Ophthalmic Drugs"* and lectures extensively in South Africa and abroad. He consults for numerous contact lens companies and the SAOA.

Dirk is married to Sarah and has two children Meagan and John Leslie and when he has time restores and flies vintage aircraft.

ABOUT THE PUBLISHER

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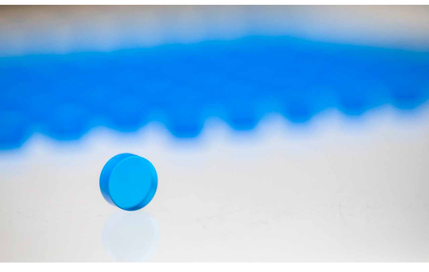


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