Contact lens complications

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Competencies covered: Contact lenses, contact lens practice
Target groups: Contact lens specialists, dispensing opticians, optometrists

Contact lenses remain a popular method of visual correction despite advances in techniques of refractive surgery improving treatment options available. In 2005 the number of contact lens wearers worldwide was estimated at approximately 125 million[1]. Although contact complications are generally rare (with one study quoting a figure of 6% of wearers developing a complication each year[2]), the overall total affected is significant due to the number of individuals who use contact lenses. Unfortunately there is a wide range of potential contact lens complications that have been classified in terms of their underlying aetiology or cause, the initial location of condition (for example lids, conjunctiva or various layers of the cornea) or the clinical subtype[3]. It is important that such complications are diagnosed correctly and managed effectively. This article will highlight a number of contact lens complications ranging from mild to potentially sight threatening. It will discuss characteristic signs, symptoms and treatment.

Giant papillary conjunctivitis (GPC)

GPC is a non-infectious inflammatory condition affecting the superior tarsal conjunctiva. It has been found to be most common in soft contact lens wearers though it may also occur as a result of rigid lens use. It is thought to have a multifactorial aetiology meaning that a combination of factors are responsible[4]. These include an immunological basis involving an allergic reaction to the contact lens material or deposits on the contact lens surface such as mucus, protein, bacteria, cell debris or air pollutants which act as an antigen[5]. These antigens are phagocytosed by cells on the mucosal epithelium of the conjunctiva and a series of events results in the production of an antibody immunoglobulin IgE by B-lymphocytes. IgE mediates the Type 1 immediate hypersensitivity reaction. It binds to receptors on various cells such as mast cells and basophils. The patient generally does not experience any symptoms after the initial contact with the antigen but repeated exposure triggers increased synthesis of IgE. Subsequent episodes result in the antigen binding to and cross-linking IgE molecules already bound to receptors on sensitised mast cells. This triggers mast cell degranulation and the release of various inflammatory mediators such as histamine, prostaglandins and leukotrienes[6]. The action of these substances culminates in capillary vasodilation (conjunctival redness), increased vascular permeability (conjunctival oedema) and increased mucus secretion[7]. Chemotactic elements are also released from the mast cells causing a migration of eosinophils and other inflammatory mediators to the site[8].

The Type 4 delayed hypersensitivity reaction is also thought to be implicated in the pathogenesis of GPC. It is mediated by T-lymphocytes and generally occurs 24-48 hours after the initial exposure to the antigen[9]. Its action results in the amplification of the inflammatory response[4].

Trauma resulting from the interaction of the contact lens with the lid has also been implicated in the development of GPC[9]. The damaged conjunctival cells are thought to secrete another type of chemotactic compound known as neutrophil chemotactic factor[10]. This protein also potentiates the inflammatory response.

Symptoms

The onset of symptoms of GPC can vary greatly, occurring from weeks to

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years after contact lens wear has commenced[11]. The affected patient normally suffers from heightened awareness of their contact lenses. Increased lens movement and mucus production which coats the lens together with other protein deposits may result in the patient experiencing blurred vision a few hours after lens insertion. This symptom together with burning, itching and foreign body sensation often culminates in a reduced tolerance to contact wear[12]. Individuals with GPC may also complain of sticky eyes especially in the morning[13]. Symptoms generally worsen with continued lens wear and may not completely resolve for weeks after cessation of lens use. The vast majority of GPC cases occur bilaterally, although one eye may be affected more than the other.

Signs

GPC is a progressive condition, worsening if the causative agent is not removed. This is the continuing exposure to the contact lens material, substances coating the contact lenses or mechanical effects relating to the lenses. Early signs normally include subtle thickening and hyperaemia of the conjunctiva[14]. As the condition progresses the thickening worsens and conjunctival opacification develops due to the accumulation of various inflammatory cells. Mucus secretion evolves from small strands observed in milder forms of GPC to a thick cord-like pale discharge[15]. This accumulates medially and in the inferior fornix. Histologically the presence of papillae increase the surface area of the conjunctiva and the number of goblet cells present resulting in increased mucus production[16].

Papillae, the extent of which will depend on the severity of the condition can be examined using lid eversion in conjunction with the slit lamp - see **Figure 1**. Papillae are cone shaped structures that form as a consequence to any sub-acute or chronic inflammation. They are comprised of a central vascular core that runs perpendicularly to the tarsal plate[17]. It is important to differentiate these from follicles that may present in response to topical ophthalmic medications as well as part of a variety of ocular conditions including chlamydial conjunctivitis, adenovirus and molluscum contagiosum[17]. Follicles are similar in appearance to papillae but have vessels running between their bases and no central vascular core.

Initially in GPC small papillae develop, but if the condition progresses they increase in number and coalesce forming macropapillae (0.3-1.0mm) and finally giant papillae (> 1mm in diameter) which may have a cobblestone appearance. These are best viewed under cobalt blue light after the insertion of fluoroscein. Staining due to superficial erosion of the thickened conjunctival epithelium overlying the papillae may also be present[15]. The location of papillae tends to vary depending on the lens type worn. In soft lens wearers the overall number of papillae is greater[18]. They are most concentrated near the fold of the everted lid (tarsal plate) and are rounder and flatter in shape[18]. Rigid contact lens wearers who suffer from GPC generally have crater-shaped papillae concentrated near to the lid margin[18].

Treatment

Management of GPC tends to be dependent on the severity of the condition. Patients experiencing low grade GPC may be advised to reduce their contact lens wearing time and subsequently monitored to see if any further action is advisable. Improved contact lens hygiene such as enzymic cleaning or more thorough surfactant cleaning measures may also be needed. It may also be advisable to change patients to preservative free cleaning, rinsing and storage systems if they are not already using them[18]. Artificial tears may also be recommended to help irrigate the lenses, washing away debris and protein and to dilute the tears reducing the concentration of IgE (which is increased in GPC) and other mediators present[15]. If these measures are not enough a more frequent lens replacement or refit with daily disposable contact lenses may also help alleviate the situation. This may be undertaken together with a change in lens material. Alternatively, if mechanical trauma is thought to be the main reason for the development of GPC refitting with a thinner soft lens to reduce lens movement and subsequent irritation is another possible treatment option[18]. Cessation of contact lens wear has been shown to alleviate GPC but unsurprisingly tends not to be a popular solution with contact lens wearers[16].

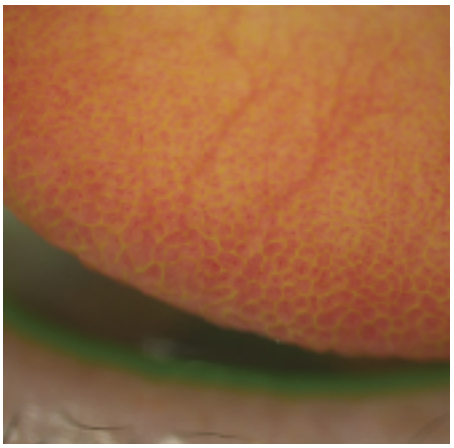

Figure 1: Papillae characteristic of GPC (*courtesy of Alan Rosen*[18])

For moderate cases of GPC intervention with medication may be necessary. The most commonly used topical medications are combination drugs which can act by preventing the release of histamine from mast cells (mast cell inhibitor) and also as H1-receptor antagonists, blocking the action of histamine at this type of receptor. An example of this type of drug is ketotifen fumigate. Non-steroidal anti-inflammatories (NSAIDs) or steroids can be prescribed in severe cases of GPC. Corticosteroids tend to be a final resort due to the increased risk of cataract formation and raised intra-ocular pressure associated with their long-term use as well of the risk of exacerbating existing herpetic ocular infection[20]. However they inhibit a variety of inflammatory signs including oedema, capillary dilation and leucocyte migration[21].

Sterile corneal infiltrates

Sterile corneal infiltrates are also the consequence of an immunological reaction. Various immune cells migrate to the corneal site of injury that can be caused by a number of factors such as toxins, hypoxia or sensitivity to lens care systems. The immunological response results in inflammation. The collective migration of inflammatory cells may be observed under the slit lamp as an infiltrate. Infiltrates can also be observed in contact lens wearers as a result of infection. An ocular swab or scraping can be taken from the infiltrate site and used to produce microscope slides and cell cultures in various liquid or on solid media[22]. If a culture can be grown the type of

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media and various staining techniques such as Gram staining can be used to narrow down the possible organism responsible and to confirm if the cause is bacterial rather than fungal, viral or acanthamoebal. If a culture is not able be produced from the sample the infiltrate is known as sterile or non-infectious. However, conversely infiltrates that have been considered sterile have been found to produce positive cultures though these microorganisms tend to be less virulent (disease causing)[23].

It may be advisable to arrange treatment of sterile infiltrates with topical antibiotics if there if any doubt regarding differential diagnosis rather than risk the outcome of untreated infectious keratitis[23]. Fortunately even if microbial keratitis is diagnosed the availability of multi-spectrum antibiotics enables treatment to be initiated whilst waiting for the results of the corneal scrape as this may take at least a few days. Culturing is especially important if the initial treatment regimen has no or reduced effectivity, other additional organisms are also suspected and have specific culture requirements (such as fungus) that confirm diagnosis or if the infection is progressing rapidly, threatening the visual axis or likely to lead to corneal perforation[24]. A corneal scrape will also debride necrotic tissue and aid penetration of antibiotic drops subsequently administered increasing the liklihood of a good treatment prognosis[25].

Sterile infiltrates tend to present as small (usually less than 2mm) single or multiple white or gray opacities situated in the sub-epithelium of the cornea, though they may also be found in the epithelium or anterior stroma. However corneal infiltrates with a similar appearance may also occur at the early stages of microbial keratitis. The ability to differentially diagnose various conditions that present with corneal infiltrates is essential skill for the contact lens practitioner as infective keratitis may result in permanent visual loss if left untreated. The infiltrate may be very subtle in appearance so a thorough examination is advisable in contact lens wearers. They may be observed initially under low magnification using a diffuse beam and then in more detail under high magnification using direct or indirect retroillumination[27]. There may also be adjacent localised conjunctival injection with both sterile infiltrates and in the early stages of microbial keratitis. Corneal oedema surrounding the infiltrate tends to be indicative of an infective cause[23]. The infiltrate with overlying epithelial staining following insertion of fluorescein is more likely to be due to infection but can also be sterile in nature[23]. Presence of anterior chamber activity is rare in conjunction with sterile infiltrates. Although a link between contact lens wear and sterile corneal infiltrates has been determined there is also an association between this type of inflammation and various other conditions. These include blepharitis, keratoconjunctivitis sicca and superior limbic keratoconjunctivitis[26].

Sterile infiltrates may be considered an umbrella term that includes all corneal infiltrates not associated with micro-organisms. They are not always linked to contact lens wear but even those conditions that are vary in their cause and clinical characteristics. These include contact lens peripheral ulcer (CLPU) and contact lens acute red eye (CLARE).

Contact lens peripheral ulcer

Contact lens peripheral ulcer (CLPU) is normally observed unilaterally. The contact lens surface is colonised by Gram-positive bacteria (staphylococcus aureus being the most common culprit) that release exotoxins. These are recognised as foreign antigens by the wearer's immune system and the consequent defensive collective migration of leucocytes observed as a corneal infiltrate. This can occur with daily wear contact lenses but there is a higher incidence associated with extended wear lenses.

Signs

CLPU normally affects one eye only. It is a small single round anterior stromal gray lesion (1-2mm in size) situated in the mid-peripheral or peripheral cornea, often under the upper eyelid[27]. During the active phase there is an overlying full thickness epithelial defect that stains following insertion of fluoroscein. Staining may also indicate anterior stromal involvement at this stage of the condition[23]. However the cornea surrounding the lesion tends to be clear[27]. Limbal and bulbar conjunctival redness adjacent to the infiltrate may also occur during the acute episode of CLPU[28].

Symptoms

The patient is often asymptomatic or may experience mild discomfort (foreign body sensation) and slight watering. They may complain of experiencing mild sensitivity to light and/or ocular pain.

Management

Fortunately CLPU is self-limiting and generally resolves without intervention once contact lens wear has ceased. The importance of lid hygiene measures should be discussed and demonstrated to the patient and appointments arranged to allow frequent monitoring of healing. Occasionally prophylactic antibiotics may be prescribed to prevent infection from occurring whilst the corneal integrity is breached. Lubricating drops or NSAIDs may also be given to improve patient comfort. A topical corticosteroid may be recommended if the inflammation persists without any signs of infection for more than a few days but it is important to rule out the differential diagnosis of microbial keratitis. CLPU usually takes approximately a week to resolve and a small faint scar with a bull's eye appearance may remain at the level of Bowman's layer[29]. This may take up to six months to disappear completely[30].

Contact lens acute red eye (CLARE)

CLARE is an acute inflammatory reaction of the cornea and conjunctiva in response to contamination of contact lenses, solutions or contact lens case by colonisation of a Gram-negative bacteria pseudomonas aeruginosa. It is usually unilateral. Contact lens wearers who also suffer from certain other ocular conditions such allergies, blepharitis and dry eye have been found to be at an increased risk of developing CLARE[31].

Symptoms

The patient tends to report ocular pain and foreign body sensation that may initially have woken them in the night. It is common for them to complain of a red, watery eye and they may mention that they are sensitive to light. Their vision acuity is normally

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unaffected. These symptoms are often amplified by continual contact lens wear[31].

Signs

Small multiple sub-epithelial infiltrates are present near to the limbus in all four quadrants of the peripheral cornea[28]. There does not tend to be any overlying epithelial defect. However although the infiltrate itself doesn't stain there may be mild generalised punctate staining. Bulbar conjunctival hyperaemia with circumlimbal injection is usually present. Anterior uveitis (cells and flare in the anterior chamber) is also a feature in more severe cases of CLARE.

Management

Normally CLARE is self-resolving once contact lens wear is stopped. Patients should be advised to cease contact lens wear and use of preservative free artificial tears and cold compresses may be recommended. As with CLPU the infiltrates should be monitored frequently to ensure that they are resolving. Most signs and symptoms should have settled within 48 hours, apart from infiltrates, which take between two and three weeks to disappear[32] If the contact lens appears to be tight a refit with a looser lens may be carried out. Immediate therapeutic intervention is advisable if the condition worsens. If contact lens wear is restarted after resolution it should be gradual with overall reduced wearing time and no overnight wear initially in extended wear users. As with CLPU lid and lens hygiene regimens should be discussed and demonstrated if necessary. The risk of reoccurrence of CLARE should be made clear (approximately one third of cases will reoccur[28]) and the importance of arranging an assessment if they experience similar symptoms in the future. If repeat episodes happen with extended wear contact lenses it may be vigilant to recommend a refit, changing contact lens modality to daily wear or to advise that contact lens wear is stopped permanently. The incidence of CLARE has been found to be greater in individuals with upper respiratory tract infections caused by haemophilus influenzae. Extended wear contact lens patients should be encouraged to switch to daily contact lens wear or ideally stop contact lens wear in this instance until the infection resolves[33].

Microbial keratitis

Microbial keratitis is a serious eye infection that is caused by various microorganisms including bacteria, fungi, viruses and parasites[34]. Ocular trauma, contact lens wear and ocular surface disease (such as bullous keratopathy and tear film deficiencies) have been found to be the main risk factors for developing this condition[35]. The incidence of microbial keratitis in contact lens users is highest with extended wear[36].

Bacterial keratitis

In contact lens wearers bacterial contamination of contact lenses may result from the individual's normal bacterial flora on their lid margins, tap water, the patient's hands during lens handling or contaminated contact lens cases. The bacteria bind to deposits and debris coating the lens. A subsequent interaction between adhesins on the bacterial cell surface with host cells surface receptors on the corneal epithelium is the first step of bacterial infection in microbial keratitis. Bacteria do not infect a patent cornea, so although contact lenses adversely affect the mucous layer of the tear film and the epithelial glycocalyx which act to prevent bacterial adherence there needs to be epithelial compromise present to enable bacterial infection to develop[28,37]. Otherwise the bacteria are removed by normal sloughing of epithelial cells or by the activity of antimicrobial enzymes in the tears. Epithelial damage may be caused by a breach in the epithelium related to contact lens wear (such as an abrasion occurring during contact lens insertion or removal or due to a faulty lens.) In addition contact lens induced hypoxia has an adverse affect on epithelial repair after such an event.

The Gram-negative rod shaped bacteria pseudomonas is the pathogen responsible for the majority (70%) of bacterial keratitis cases in soft contact lens wearers[38]. When pseudomonas binds to the corneal epithelium this can stimulate phagocyosis of the bacteria by the epithelial cells. Once the bacteria reaches the cytoplasm of the cell rapid replication occurs. Unchecked this can result in epithelial cell death[39]. However if the cornea is intact the basal lamina a thin layer beneath the epithelium prevents bacteria from reaching the stroma as they are impeded by their size unless this layer is damaged. The individual's immune system recognises antigens on the bacterial surface as foreign causing increased production of a family of proteins known as cytokines. Cytokines stimulate the migration of polymorphonuclear leukocytes to the site by chemotaxis and an amplification of the inflammatory response. Although the function of the immune system is to destroy bacteria present, inflammation and resultant corneal damage also results. This is partly due to the action of lysosomal enzymes. Oxidative damage also results from the action of oxidising agents also released by the host[34]. In addition pseudomonas also releases a number of enzymes as well as toxins that contribute to corneal breakdown[34]. Eventually stromal necrosis results from the cumulative damage. Misdiagnosis and lack of treatment or ineffective management of severe microbial keratitis caused by highly virulent organisms can result in corneal perforation, the development of endophthalmitis and even the loss of the affected eye[40].

Signs

The signs of early stage microbial keratitis before ulceration occurs are subtle. They may include superficial punctate keratitis and epithelial disruption[28]. Bulbar limbal hyperaemia also occurs adjacent to an epithelial infiltrate. As the condition develops the stroma becomes hazy with a ground-glass appearance and there is epithelial breakdown overlying the infiltrate that stains with fluoroscein[41]. The limbal hyperaemia becomes less specific with more generalised bulbar and circumlimbal redness - see **Figure 2**. The patient may also develop a serous (watery) or yellow-green mucopurulant (mucus and pus containing) discharge[42]. Unchecked, further progression results in development of an ulcer, stromal melting and eventually corneal perforation[28]. The development of perforation may be extremely rapid (less than 24 hours)[41].

Anterior chamber activity (the presence of cells and flare) can be observed in early stage microbial keratitis whereas hypopyon and

keratic precipitates may be associated with more developed cases[43]. Lid swelling may also occur at some stage in the disease process.

Symptoms

The patient may complain of a foreign body sensation that has not improved on contact lens removal or has worsened[41]. Symptoms tend to worsen rapidly developing into pain and excessive watering with sensitivity to light.

Management

This condition should be treated as an emergency and an urgent ophthalmological assessment is needed. Initial treatment is usually with a multi-spectrum antibiotic such as a fourth generation fluoroquinolone. The aim is to stop the infection and any further adverse corneal effects[43]. Fluoroquinolones are bactericidal and their mode of action involves inhibiting the activity of DNA gyrase. The resultant structural change in bacterial DNA results in death of the microorganism[45]. If this proves unsuccessful the condition may be caused by a resistant species of bacteria. A fortified antibiotic comprising a combination of antimicrobials may be prescribed which is effective against both Gram-positive and negative organisms[36]. Cycloplegic eyedrops may also be given to prevent the formation of synechiae associated with secondary uveitis and reduce patient discomfort by alleviating ciliary spasm. The patient may also be advised to use cold compresses with cooled boiled water to reduce inflammation. Secondary glaucoma may also occur as a result of inflammatory cells blocking the trabecular meshwork and topical beta-blockers prescribed to treat the raised intra-ocular pressure.

Successful treatment results in alleviation of the epithelial defect, resolution of the infiltrate and surrounding oedema and the development of a corneal scar[45]. Its corneal location influences whether the patient's visual acuity is affected.

Acanthamoeba keratitis (AK)

Acanthamoeba is a single-celled organism that occurs in two forms: as an active trophozoite that is able to replicate and a highly resistant dormant cyst. Acanthamoebal keratitis (AK) is similar to bacterial in that adherence of the micro-organism to the corneal surface is the initial step required to enable infection to occur. The action of a receptor on the pathogens cell surface in tandem with the secretion of various amoebal enzymes results in the destruction of the host's epithelial cells. This is followed by basement membrane and underlying anterior stromal breakdown and deeper corneal penetration[46]. Immunoglobulin A (IgA) an antibody present in the tears may provide protection against acanthamoeba infection but some species of acanthamoeba are able to produce proteases to break the antibody down.

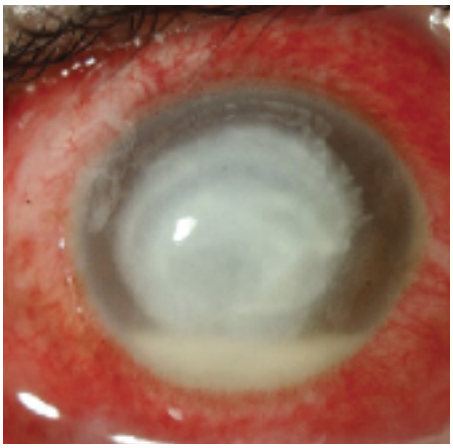

Figure 2: Pseudomonas aeruginosa keratitis *(courtesy of Singapore National Eye Centre (SNEC)[44])*

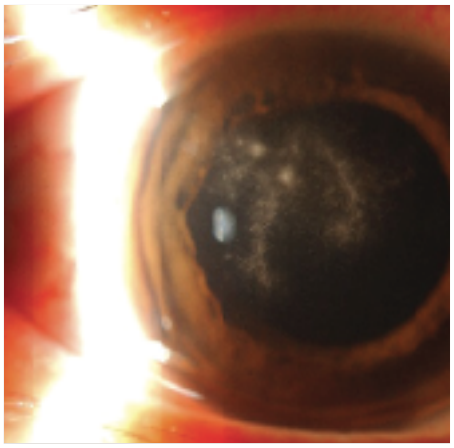

Figure 3: Acanthamoeba keratitis - satellite lesions *(courtesy of SNEC[47])*

Signs

The early signs of AK are often non-specific. They may include punctate epithelial erosions and irregularities as well as epithelial and sub-epithelial opacities[47]. The most common is sub-epithelial dendritic keratitis that may be incorrectly diagnosed as herpes simplex keratitis. The possibility of AK should be considered if there is a history of contact lens wear, antiviral therapy has not been successful and the patient complains of severe and persistent pain. However this symptom may not develop until later in the course of the condition. Radial perineural infiltrates (infiltrates that are situated along the path of the corneal nerves) can occur in the early stages and are highly indicative of AK. If the disease progresses the infiltrates may coalesce forming a stromal ring infiltrate (usually a number of weeks after the initial symptoms)[48]. Infiltrates are due to inflammation resulting from the host's immune response to antigen markers on the amoeba cell wall and coincides with symptoms of excruciating pain[48,49]. There may also be satellite lesions present adjacent to the main infiltrates - see **Figure 3**. Acanthamoebal cysts can be observed in the stroma with confocal microscopy[50]. The presence of the ring infiltrate is usually accompanied by perilimbal and ciliary injection of the conjunctiva as well as chemosis. Hypopyon may also develop and the sclera may become affected (scleritis)[47]. The patient is also at risk of developing glaucoma as well as secondary cataract[47].

Symptoms

The clinical presentation of AK can vary greatly between individuals. Most patients complain of a watery, extremely painful eye, the onset of which has occurred suddenly. The pain is often significantly greater than that expected in relation to the clinical signs observed. Other symptoms such as redness, watering, sensitivity to light and blurred vision worsen over a number of weeks[51].

Management

A combination of early diagnosis and prompt aggressive treatment of AK is essential for a good prognosis. Therefore suspected AK should be referred urgently for ophthalmological management. Recommended therapy involves a combination of antimicrobial agents (cystocidal and trophozoicidal drugs) that act synergistically. These include a biguanide (eg, 0.02% polyhexamethylene biguanide PHMB or 0.02% chlorhexidine digluconate (CHX) and diamidine (eg, 0.01% propamidine isethionate (Brolene)

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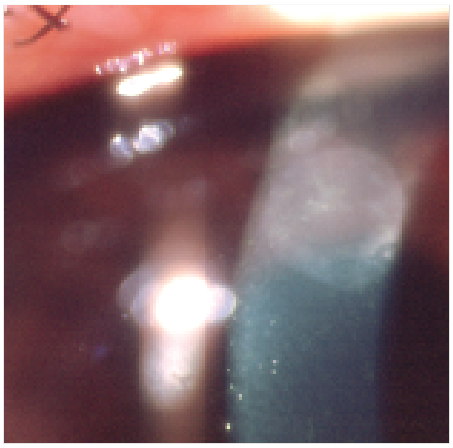

Figure 4: Mild scarring following acanthamoeba keratitis - fortunately the scarring is situated away from the visual axis *(courtesy of Alan Rosen[18])*

respectively[52]. Biguanides interact with the phospholipid bilayer of the plasma membrane of the acanthamoeba leading to membrane disruption and pore formation. This causes cell breakdown[49]. Diamidines slot in to the acanthamoebal DNA structure (intercalation) resulting in the inhibition of polyamine metabolism[49]. This results in acanthamoebal cell growth being severely compromised or stopping altogether.

Fungal keratitis

Fungal keratitis is most commonly caused by filamentous fungus (fusarium and aspergillus species) but may also be due to yeasts such as candida and dematiaceous fungi. The former is rare particularly in countries that do not have tropical climates. This condition is an opportunistic infection and there are a variety of risk factors. These include a history of trauma (especially involving exposure to vegetation such as plant and soil matter) or corneal surgery such as penetrating keratoplasty. There is also a greater incidence in immune-compromised individuals, patients with ocular surface disease, long term steroid users and soft contact lens wearers[53]. A breach in the epithelium is required for fungal penetration in common with other types of microbial keratitis.

Signs

Keratitis caused by filamentous fungi and pseudomonas can both present with a dendritic ulcer in the early stages that may be mistaken for herpes simplex keratitis. (as observed in acanthamoebal keratitis). The accompanying signs of inflammation associated with fungal keratitis are minimal in contrast to bacterial keratitis[54]. The infiltrates are shades of grey or yellow in colour and the majority have indistinct raised feathery borders and appear dry and rough in texture[54]. The overlying epithelial may appear to be intact over the deep stromal infiltrate. A small number of cases (10%) also have lesions surrounding the main infiltrate (satellite lesions)[54]. If the condition progresses unchecked conjunctival hyperaemia, the formation of a endothelial fibrin plaque, anterior chamber inflammation (cells and flare in the anterior chamber and the development of hypopyon), iritis and eventual corneal perforation can result.

Symptoms

The present may complain of pain or foreign body sensation that is usually unilateral. The former symptom may appear to improve as the condition progresses and corneal nerves are damaged[55]. Lid twitching (blepharospasm) is another common feature as well as watering (epiphora) and an increased sensitivity to light. They may also be aware of a decrease in their vision - the larger the infiltrate present the greater the adverse effect on the patient's visual acuity[56]. The severity of symptoms and rate of progression of fungal keratitis is highly variable between cases[56].

Management

As with other types of microbial keratitis prompt action is necessary to improve prognosis and an urgent ophthalmological opinion and management is merited. Evaluation of patient history, a clinical examination and results from a corneal scrape aid diagnosis. Corneal smears viewed with microscopy to detect fungal features in conjunction with staining techniques and inoculation of various types of growth media can both help elucidate the organism responsible[55]. Polymerase chain reaction (PCR) of the DNA from the sample can also be utilised to determine the microorganism[56]. Anti-fungals and frequent monitoring tends to be the primary treatment approach but may prove unsuccessful due to the drug's poor penetration of the cornea. Debridement to remove superficial necrotic corneal tissue may also be necessary. A significant association has been found between poor contact lens care and development of fungal keratitis in contact lens wearers[56]. Prevention is the best strategy especially for this condition as treating it can be particularly challenging.

Keratitis and need for surgery

Surgical treatments such as penetrating keratoplasty (full thickness corneal graft) may be required for any type of keratitis if there is extensive scarring or if previous treatment is unsuccessful -see **Figure 4**[55, 56].

Conclusion

This article has discussed a number of contact lens complications. It highlights the importance of the ability to differentially diagnose such conditions and self-manage or refer promptly for an ophthalmological opinion and treatment if necessary. The importance of regular aftercares, the action for the patient to take in the event of any unusual signs or symptoms developing (ceasing contact lens wear and contacting the practice for advice for example) should be discussed. It is also sensible to reiterate the importance of hand hygiene prior to handling lenses, as well as regular contact lens, contact lens solution and contact lens case replacement if relevant to the contact lens modality worn. The best option with regards to such complications is taking such prophylactic steps to avoid them occurring in the first place and ideally this should also be communicated to the contact lens wearer.

References

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MCQs overleaf

Multiple choice questions (MCQs): Contact lens complications

1. Giant papillary conjunctivitis (GPC) is most common in:
a. Soft contact lens wearers
b. Rigid gas permeable contact lens wearers
c. Hard contact lens wearers
d. None of the above

2. Management of GPC does NOT include:
a. Change to preservative-free regimen
b. Administering artificial tears
c. Application of H1-antagonist
d. Application of H2-antagonist

3. A bulls-eye appearance as the condition resolves is characteristic of . . .
a. Contact lens peripheral ulcer (CLPU)
b. Contact lens acute red eye (CLARE)
c. Microbial keratitis
d. None of the above

4. What percentage of CLARE cases reoccur?
a. 10.0%
b. 33.3%
c. 50.0%
d. 100.0%

5. Which symptom is characteristic of bacterial keratitis?
a. Foreign body sensation which resolves on contact lens removal
b. Foreign body sensation which worsens on contact lens removal
c. Pain which improves as condition progresses
d. Metamorphopsia

6. Treatment for microbial keratitis may include . . .
a. Fortified antibiotics
b. Cycloplegic drops
c. Cold compresses
d. All of the above

7. Complications associated with microbial keratitis include:
a. Primary cataract
b. Glaucoma
c. Diabetes
d. Conjunctivitis

8. The most common type of bacterial keratitis is caused by . . .
a. Staphylococcus aureus
b. Pseudomonas aeruginosa
c. Streptococcus
d. None of the above

9. Which is the most appropriate management of CLPU?
a. Contact patient triage at local casualty unit to arrange an ophthalmological consult urgently
b. This condition will self-resolve. See patient routinely or sooner if symptoms develop
c. Patient to cease contact lens wear at present. Frequent monitoring arranged to ensure that condition is resolving. See patient sooner if symptoms develop
d. None of the above

10. A risk factor for fungal keratitis is . . .
a. Previous corneal surgery
b. Contact lens wear
c. Chronic steroid use
d. All of the above

11. Radial perineural infiltrates are characteristic of which condition?
a. Fungal keratitis
b. Contact lens peripheral ulcer
c. Acanthamobeal keratitis
d. Bacterial keratitis

12. An infiltrate with indistinct raised borders and a dry appearance is characteristic of which condition?
a. Acanthamoebal keratitis
b. Contact lens peripheral ulcer
c. Contact lens acute red eye
d. Filamentous fungal keratitis

The deadline for posted or faxed response is 14 June 2012 to the address on page 4. The module code is C-18353

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Reminder for the end of CET period

Registered practitioners are reminded that the current CET period ends on 31 December 2012. By that date, to ensure continued registration with the GOC, you must have accrued 36 points as a dispensing optician or an optometrist, or 54 (36 +18) points as a contact lens optician on the GOC's CL Specialty list. The points must be confirmed on your CEToptics record. Pending, or unconfirmed points will not count towards your requirement. If you have any enquiries about your points record, contact CEToptics (0843 208 5487), or for other enquiries, contact the ABDO CET Office (01206 734155 Tuesday - Friday).