Contact lenses are medical devices being used to correct vision but their role in cosmetic or therapeutic applications is no less. In 1888, the German ophthalmologist Adolf Gaston Eugen Fick constructed and fitted the first successful contact lens and optical correction via contact lens have come a long way since then. With rising demand, the expectations from these devices has increased. Hence, the importance of adequately explaining the potential complications to prospective users, and dealing effectively with those complications, is even more prudent. Most contact lens related complications can be avoided by a proper pre-fitting assessment, evaluation of the lens fit and detail instructions on its use including regular aftercare visits. The key to identifying a contact lens related problem is thorough history taking and a detailed anterior eye assessment with and without the contact lens in the eye. Grading scales have been developed to assist practitioners in recording the level of severity of ocular complications of contact lens wear.

Abnormal blinking can cause contact lens surface drying, deposition on the lens surface, corneal epithelial desiccation, post lens tear stagnation, hypoxia, hypercapnia and 3 & 9 o clock staining. Blink training and altering lens design and fit can improve blinking and avoid the related complications.

Blepharoptosis Eyelid ptosis can occur in younger adult patients who wear contact lenses. Due to continuous micro trauma from insertion and removal of the lenses, blink induced lens rubbing, contact lens migration and papillary conjunctivitis. It happens mainly in rigid corneal lens wearers, but can also happen in soft lens wearers. The patient will most likely complaint of poor cosmesis when the ptosis is excessive. Ptosis in contact lens wearers occurs due to lid oedema caused by papillary conjunctivitis or due to levator aponeurosis.

Blinking Abnormalities According to Abelson and Holly blinking can be classified into four types:

- **Complete blink** – the upper eyelid covers more than 67% of the cornea
- **Incomplete blink** - the upper eyelid covers less than 67% of the cornea
- **Twitch blink** - a small movement of the upper eyelid
- **Forced blink** – lower lid raises to complete eye closure.

The list of potential contact lens related complications is long and varied, ranging from relatively benign dry eye to potentially sight-threatening microbial keratitis. In this article, we shall discuss several common complications that the lens-wearing patients may experience.

Figure 1: Meibomian Gland Dysfunction
If it is due to papillary conjunctivitis, cure the papillary conjunctivitis. If it is due to rigid gas permeable corneal lenses then refit the patient with soft lenses. In severe cases, surgical intervention is required.

**Meibomian Gland Dysfunction**

It has been found that contact lens wear is associated with a decrease in the number of functional meibomian glands, which may be a cause of dry eye in contact lens patients. Daniel Nelson, defined meibomian gland dysfunction as a "chronic, diffuse abnormality of the meibomian glands, commonly characterized by terminal duct obstruction and/or qualitative/quantitative changes in the glandular secretion. This may result in alteration of the tear film, symptoms of eye irritation, clinically apparent inflammation and ocular surface disease.

Symptoms of MGD include foreign body sensation, burning, itching, watering and fluctuating or decreased vision. The diagnosis of MGD is difficult because many of the symptoms mirror the everyday complaints by contact lens wearers. One method to assess the stability of the tear film is by performing a tear break-up test (TBUT). A TBUT can be performed noninvasively using a Keeler Tearscope, a non-automated keratometer or a topographer, or it can be performed invasively using NaFL and a cobalt blue filter at the slit lamp. In normal control eyes the values of the fluorescein TBUT can range from 3 to 132 seconds, with an average of 27 seconds. In dry eye patients, TBUT is reduced to mean values lower than 5 seconds, but the inter-individual variability is still high. Despite the wide variation in TBUT among individual subjects, there is general agreement that a TBUT shorter than 10 seconds reflects tear film instability, whereas a TBUT shorter than 5 seconds is a marker of definite dry eye.

If these signs are present in contact lens patients complaining of reduced wear time or contact lens intolerance, the patient may be suffering from meibomian gland dysfunction.

Depending on the type and etiology of MGD, it can be treated with warm compresses, lid scrubs, mechanical expression, antibiotics, calcineurin inhibitors and cyclosporine essential fatty acids, artificial tears, topical lid supplements or surfactant cleaning.

**Dry Eye**

Dry eye is a multifactorial disease of the tears and ocular surface that results in symptoms of discomfort, visual disturbance, tear film instability with potential damage to the ocular surface. It is accompanied by increased osmolality of the tear film and inflammation of the ocular surface. Other contributing factors include the use of high-water-content lenses, which have traditionally been reported to be associated with less patient comfort than lower-water-content lenses, potentially due to spoilation and deposition of the lipid layer. Symptoms of dryness are often cited as the primary reason patients drop out of contact lens wear.

Evaluate the tear volume, tear film structure and quality, tear film stability. The tear volume in prospective and current contact lens wearers can be assessed by observing the height of the lower lacrimal tear prism. Using optical coherence tomography, Chen et al. demonstrated that tear meniscus volumes in dry eye symptomatic wearers are lower than in asymptomatic wearers at baseline and during lens wear.

Tear film structure can be assessed clinically by observing the corneal surface in specular reflection or by using a tearscope. It has been observed by Young and Efron that the lipid layer was either absent or very thin on all hydrogel lenses rigid lenses, although there was a tendency of higher water content lenses to support a thin lipid layer. The aqueous layer was generally found to be thicker on lenses of high water content.

Tissue damage to the surface of the cornea and conjunctiva can occur during contact lens wear as a consequence of disruption of tear layer. Guillon and Maisa reported that lissamine green conjunctival staining could discriminate symptomatic from asymptomatic contact lens wearers.

An excess of a particular tear component coupled with compromised structural integrity of the tears leading to rapid tear break up and excessive surface drying, are intrinsic factors thought to be conductive to deposit formation on lens surface. Indeed there is some clinical evidence to support the notion that lens deposition is a particular problem in dry eye patients wearing contact lenses.

Symptoms of dryness are often cited as the primary reason patients drop out of contact lens wear. The lens induced changes cause increased osmolality of tears, change in pH of tears, composition of tears, tear film break up and the temperature profile of the tear film.

This can be treated by either changing the lens material, lens solution, rewetting drops, nutritional supplements and by control of evaporation and omega-6 fatty acids.

**Mucin Balls**

Mucin balls are spherical and translucent or opalescent bodies sandwiched between a CL and the cornea that can be observed within minutes after lens insertion. They are
Contact Lens-Induced Papillary Conjunctivitis

Contact lens–induced papillary conjunctivitis (CLPC) is an inflammation of the upper palpebral conjunctiva and is one of the main reasons for CL discontinuation. It is characterized by enlarged papillae (>0.3 mm), hyperemia, and mucus strands. Patients who have CLPC can be asymptomatic or experience acute ocular discomfort with complaints of itching, mucus or ropy discharge, lens awareness, and blurred vision, which are the results of increased front-surface lens deposits and excessive lens movement. Patients may be asymptomatic or experience foreign body sensation, especially on awakening. The lesions can be superficial, affecting only the first one to three layers of the epithelium or deep into the basement membrane, and stain with sodium fluorescein with no underlying infiltrates. Alternatively, the epithelium may become detached centrally but remain adherent at the border, representing an early stage of development. There is no mucopurulent discharge, and there may be localized limbal and bulbar conjunctival injection. Patients may be asymptomatic or experience foreign body sensation, especially on awakening if lenses are worn overnight, or sharp pain exists on lens removal. Although the aim of management is to reduce pain, prevent infection, and promote re-epithelialization, there is no consensus on how best to do so. In general, lens wear discontinuation, ocular lubricants in the form of drops, gels, or ointments, and prophylactic antibiotics are all believed to help with the healing process. Bandage CLs

Contact Lens-Induced Epithelial Erosions

Corneal epithelial erosions related to CL wear are epithelial defects with a wide range of clinical presentations. In general, they can be characterized as localized, well-circumscribed lesions that can be as small as 0.1 mm in diameter or encompass a much larger area of the cornea. They can present anywhere on the cornea, but 87.5% have been found inferiorly and, more commonly, near the vertical midline just below the pupil. The lesions can be superficial, affecting only the first one to three layers of the epithelium or deep into the basement membrane, and stain with sodium fluorescein with no underlying infiltrates. Alternatively, the epithelium may become detached centrally but remain adherent at the border, representing an early stage of development. There is no mucopurulent discharge, and there may be localized limbal and bulbar conjunctival injection. Patients may be asymptomatic or experience foreign body sensation, especially on awakening if lenses are worn overnight, or sharp pain exists on lens removal. Although the aim of management is to reduce pain, prevent infection, and promote re-epithelialization, there is no consensus on how best to do so. In general, lens wear discontinuation, ocular lubricants in the form of drops, gels, or ointments, and prophylactic antibiotics are all believed to help with the healing process. Bandage CLs

The obvious strategy for preventing mucin ball formation, should this be the aim of the clinician to re-fit the patient with a lens type that is not made from silicon hydrogel. This may create a dilemma because silicone hydrogel lenses are the only lens type indicated for extended wear. Therefore, optimizing lens fit, advising the patient to use lubricating drops before sleeping and after waking could minimize mucin ball formation.

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The cause of CLPC is still not well understood, but several factors have been suggested as possible causes. Since the advent of SiHy CLs and the report of two clinical presentations of CLPC, the paradigm has shifted to suggest that perhaps local CLPC is caused by mechanical trauma and general CLPC is a hypersensitivity reaction, caused by lens surface deposits, lens coatings, or solution.

CLPC has been associated with delayed tear clearance, which might increase the protein and inflammatory mediator concentrations in the tear film and contribute to the pathogenesis or aggravate the severity of CLPC. Of interest, MGD is common for patients with CLPC, but no association was found between MGD and delayed tear clearance. It has been proposed that a tear clearance test be incorporated in the eye examination for CL wearers with CLPC, as the treatment modality can be adjusted accordingly. In cases of decreased tear clearance, non-preserved steroids may be indicated. If tear clearance is normal, then changing the lens material may be recommended first, because mechanical trauma may be a more significant contributor compared with inflammation.

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are avoided in cases of CL-induced erosion to reduce the risk of infection.

Epithelial Microcysts
Small (15-50μm), irregular shaped inclusions usually found in the para-central to mid-peripheral zones of the cornea. Observation is best with retro-illumination. Microcysts show reversed illumination due to a suspected higher refractive index than the surrounding tissue.

The presence of epithelial microcysts in the corneal epithelium is a good clinical indicator of chronic hypoxia. Unlike disposable contact lenses, silicone hydrogel lenses do not induce an increase in epithelial microcysts when worn on extended wear basis. Microcysts can be a valuable “red flag,” indicating the patient needs a lens with higher oxygen transmissibility.

Don’t be alarmed if you observe an initial increase in microcysts after refitting a patient with a more oxygen-permeable contact lens. The additional oxygen will “flush out” microcysts from deeper epithelial layers over the first month or two after switching lens materials.

Corneal Oedema
Corneal edema, or swelling, occurs when there is an inadequate supply of oxygen reaching the cornea due to contact lens wear. Following sleep with lenses, the cornea showed evidence of gross stromal edema (striae and folds in the posterior cornea) that reduced or disappeared during the day depending on the level of edema finally reached.

Symptoms of corneal edema include blurred or foggy vision, seeing rainbows around lights, redness, and possibly irritation or pain.

Since oxygen starvation is the primary cause of contact lens-induced oedema, strategies are directed towards mechanisms for increasing corneal oxygen availability during lens wear. Changing to a higher Dk, reducing the lens thickness, increasing the edge lift, a smaller lens diameter will prove helpful in alleviating contact lens induced oedema.

Complications of corneal edema include corneal abrasion, a tight lens syndrome and corneal ulcers or infection.

Corneal Infiltrates and Ulcers
The development of a corneal ulcer unfortunately is a common complication of contact lens use. Soft contact lenses have a higher risk of corneal ulcer than rigid lenses, but all lenses have some risk. A corneal ulcer starts when a bacteria (or rarely a fungus or parasite) infects an area of breakdown in the corneal surface. The surface may break down, forming a small corneal abrasion, due to routine lens use.

Overwear of lenses, improper cleaning of lenses, extended wear use of lenses, and overly tight lenses may increase the risk of developing this surface breakdown. Normally, a corneal abrasion, even if tiny, is uncomfortable. However, a contact lens can act as a bandage on the eye masking symptoms, and some contact users develop a lack of sensitivity of the cornea.

Once an infection begins, most people experience severe symptoms. The eye typically becomes red and painful. There may be tearing or discharge and sensitivity to light. The vision may be variably blurred. The most important thing to do initially is to remove the contact lens. A corneal ulcer needs to be treated intensively with antibiotic eye drops, and often a culture of the infected cornea, or of the lens or lens case is performed. Frequent follow-up appointments will help you determine if the infection is being adequately treated with the antibiotics. Usually a week or two of antibiotic eye drops is needed, and contact lenses cannot be worn during this time.

A successfully treated corneal ulcer may still leave a scar, which could affect the vision.

It is important to avoid situations which can lead to corneal ulcer, such as over wear of lenses, poor disinfection techniques, swimming with contact lenses in, and ignoring symptoms of pain or redness.

When contact lens patients present with infiltrates, consider non-infectious causes, including extended wear, hypoxia, poor hygiene, case contamination, solution sensitivity and normal flora or bacterial toxins.

Solution toxicity and hypersensitivity have been identified as a potential cause of corneal infiltrates.

Hydrogen peroxide systems are a potential alternative in cases of hypersensitivity to multipurpose solutions. In fact, the lowest incidence of corneal infiltrative events was reported in silicone hydrogel wearers using hydrogen peroxide systems.

A prospective study looked at the risk of non-ulcerative contact lens complications and showed that there is a lower rate of complications in patients wearing daily disposable vs. planned replacement lenses; Silicone hydrogel lenses wear was associated with a two-fold risk for “sterile keratitis.” Other factors that increased risk for non-infectious infiltrates included occasional and habitual overnight wear, less than 10 years of wear, poor hand hygiene and smoking.

Endothelial Changes
Contact lenses may induce short- and long-term corneal endothelial changes.

The endothelial bleb response is a short-term, reversible change noted with contact lens wearers. One physiological
factor causing endothelial blebs and polymegathism is a local acidic pH change at the endothelium, which happens due to hypercapnia and hypoxia induced due to certain contact lens wear.

Long-term endothelial changes such as polymegathism (increased variation in cell size) and pleomorphism (a decrease in the frequency of hexagonal cells) have also been detected in polymethylmethacrylate, rigid gas permeable, and daily and extended wear soft contact lens patients. Differences in endothelial morphometry between the central and mid-peripheral regions of the cornea have also been noted in hard lens wearers34.

Morphologically, the endothelial cells of contact lens wearers showed greater variability in size and shape compared to controls. The mean endothelial cell size in contact lens wearers was smaller than that of controls. It has been found that the gas permeable lens wearers had more hexagonal and less pentagonal cells35.

From a clinical perspective, it is essential to take note of the presence of significant endothelial polymegathism and to take action to minimize the metabolic stress to the cornea. Strategies for alleviating contact lens induced hypercapnia and hypoxia include; fitting soft or rigid lenses made from materials of higher gas permeability, reducing lens thickness, sleeping in extended wear less frequently, and to take action to minimize the metabolic stress to the presence of significant endothelial polymegathism and pleomorphism. Contact lens wear.

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