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Contact Lenses and Associated Anterior Segment Disorders: Dry Eye Disease, Blepharitis, and Allergy

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The tear film is a dynamic structure consisting of three major components (lipid, aqueous, and mucin), which overlies the cornea and conjunctiva and is bounded by the lid margins. It is essential to maintain the cornea and conjunctiva in a normal state; it provides moisture, lubrication, and oxygen necessary for corneal and conjunctival epithelial respiration and forms a smooth surface interfacing with air to provide a clear image of incoming light to subserve vision. In addition, it serves as a pathway for movement of cytokines and other proteins secreted by the lacrimal glands, which act on the surface epithelium to direct and regulate normal cell proliferation, differentiation, maturation, and exfoliation to maintain the homeostatic state of the ocular surface and to facilitate cellular response to injury [1].

The ocular surface cells, lacrimal glands, and eyelids form a tightly regulated functional unit linked by a neural pathway from the sensory fibers of the ophthalmic division of the fifth cranial nerve, which innervate the ocular surface epithelium [2]. From these nerves afferent impulses run to the central nervous system from which efferent fibers course to the cornea, conjunctiva, lacrimal glands, meibomian glands of the eyelids, and the orbicularis muscle of the eyelids. Efferent nerve signals regulate lacrimal gland secretion of water, electrolytes, and over 300 immunologically active proteins. Many of these small molecular weight proteins are cytokines, which act on receptors on surface cells regulating cellular functions. In addition, efferent neural fibers also innervate the meibomian glands of the eyelid and the goblet cells

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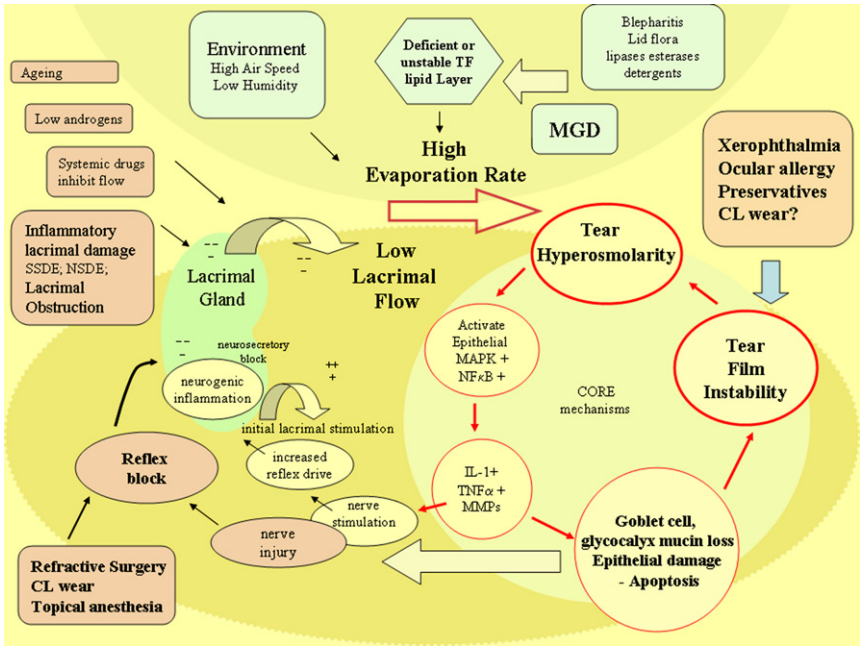
of the conjunctival. The presence of an intact neural pathway that mediates these functions is essential for the maintenance of a normal ocular surface.

The structure of tears is thought to be that of a two- or three-layered film consisting of an inner transmembrane mucin layer (MUC1 and MUC4) derived from the epithelial cells similar to the mucin in the bronchial linings. Overlying this is a thicker aqueous layer, the product of the lacrimal glands and possible transconjunctival fluid transport. Within this layer is a gradient of highly hydrated loose mucin (MUC5-AC), which forms a "blanket" protecting the ocular surface and stabilizing the tear film. Other mucin-soluble and gel-forming mucins include MUC7 and MUC16, whose function may be related to surface morphology [3]. The outermost covering of tears is a thin lipid layer, the product of the meibomian glands of the eyelid. This functions to limit evaporative loss of tears [4]. The interaction of these components contributes to the meta-stability of the tear film between blinks.

In the recent report of the International Dry Eye Workshop a new definition and classification system was issued [5–10].

> *Dry eye is a multifactorial disease of the tears and ocular surface that results in symptoms of discomfort, visual disturbance, and tear film instability with potential damage to the ocular surface. It is accompanied by increased osmolarity of the tear film and inflammation of the ocular surface.*

Two major etiopathogenic types of dry eye disease (DED) are recognized: an aqueous-deficient type in which there is a deficiency of the secretion of the lacrimal glands, and an evaporative type in which the meibomian glands of the



eyelid are dysfunctional resulting in a quantitative and qualitative change in secretion of the evaporation-sparing lipids of the tear film. In practice, about two thirds of patients present with both forms of the disease. There are a number of risk factors associated with the development of DED: age; female gender; a decrease in bioavailable androgen; systemic autoimmune disease, such as Sjögren's syndrome; collagen vascular disease, such as rheumatoid arthritis, lupus, and scleroderma; environmental stress including dry and windy climatic conditions; sustained video display terminal use; long air travel; neurologic sensory loss in the ocular surface; and decreased blinking.

Regardless of the initiating factor or group of factors, the final common expression of disease is breakdown in the homeostatic mechanisms operative at the tear-ocular surface interface resulting in instability of the tear film, an increase in the osmolarity of the tear film with damage to the ocular surface, and sensory and visual disturbances (Fig. 1).

Fig. 1. Mechanisms of dry eye [10]. The core mechanisms of dry eye are driven by tear hyperosmolarity and tear film instability. The cycle of events is shown on the right of the figure. Tear hyperosmolarity causes damage to the surface epithelium by activating a cascade of inflammatory events at the ocular surface and a release of inflammatory mediators into the tears. Epithelial damage involves cell death by apoptosis, a loss of goblet cells, and disturbance of mucin expression leading to tear film instability. This instability exacerbates ocular surface hyperosmolarity and completes the vicious circle. Tear film instability can be initiated without the prior occurrence of tear hyperosmolarity, by several etiologies including xerophthalmia, ocular allergy, topical preservative use, and contact lens wear. The epithelial injury caused by dry eye stimulates corneal nerve endings, leading to symptoms of discomfort; increased blinking; and, potentially, compensatory reflex lacrimal tear secretion. Loss of normal mucins at the ocular surface contributes to symptoms by increasing frictional resistance between the lids and globe. During this period, the high reflex input has been suggested as the basis of a neurogenic inflammation within the gland. The major causes of tear hyperosmolarity are reduced aqueous tear flow, resulting from lacrimal failure, or increased evaporation from the tear film. This is indicated by the arrow at the top-center of the figure. Increased evaporative loss is favored by environmental conditions of low humidity and high air flow and may be caused clinically, in particular, by meibomian gland dysfunction (MGD), which leads to an unstable tear film lipid layer. The quality of lid oil is modified by the action of esterases and lipases released by normal lid commensals, whose numbers are increased in blepharitis. Reduced aqueous tear flow is caused by impaired delivery of lacrimal fluid into the conjunctival sac. It is unclear whether this is a feature of normal ageing, but it may be induced by certain systemic drugs, such as antihistamines and antimuscarinic agents. The most common cause is inflammatory lacrimal damage, which is seen in autoimmune disorders, such as Sjögren's syndrome and in non-Sjögren dry eye (NSDE). Inflammation causes both tissue destruction and a potentially reversible neurosecretory block. A receptor block may also be caused by circulating antibodies to the M3 receptor. Inflammation is favored by low tissue androgen levels. Also, tear delivery may be obstructed by cicatricial conjunctival scarring or reduced by a loss of sensory reflex drive to the lacrimal gland from the ocular surface. Eventually, the chronic surface damage of dry eye leads to a fall in corneal sensitivity and a reduction of reflex tear secretion. Various etiologies may cause dry eye acting at least in part by the mechanism of reflex secretory block, including refractive surgery (LASIK dry eye); contact lens wear; and the chronic abuse of topical anesthetics. Individual etiologies often cause dry eye by several interacting mechanisms. Further details may be found in the text. CL, contact lens; IL, interleukin; MMP, matrix metalloproteases; SSDE, Sjogren syndrome dry eye; TF, tear film; TNF, tumor necrosis factor.

More severe forms of dry eye are associated with a systemic autoimmune disease (eg, Sjögren's syndrome, in which there is heavy lymphocytic cell infiltration with severe inflammation of the lacrimal glands, ocular surface, and associated structures, such as the meibomian glands of the eyelids). In the most severe cases vision-threatening complications, such as scleritis, corneal ulcers, and secondary bacterial infection, can occur. There is recent evidence that even in milder forms of aqueous tear deficiency, there is subclinical inflammation suggesting a commonality of disease processes [1].

Evaporative tear deficiency is most commonly associated with dysfunction of the meibomian glands of the eyelid. The term "blepharitis" is applied to various inflammatory conditions of the eyelids, some of which are infectious (eg, chalazia) and some noninfectious (eg, atopic blepharitis). The pathogenesis of a discrete category of blepharitis, meibomian gland dysfunction (posterior blepharitis, meibomitis), remains poorly explained. Meibomian gland dysfunction is, however, an extremely common condition; its prevalence increases with advancing age and is associated with the presence of androgen insufficiency and aqueous tear deficiency [11].

DED is widely prevalent. In its severe form, as keratoconjunctivitis sicca, it is part of a more systemic autoimmune process, Sjögren's syndrome, which is estimated to involve 4 million people in the United States. Epidemiologic studies have reported a DED prevalence of 13% to 15% in people 65 and older [12–14]. Recent studies estimate that over 9 million persons in the United States are afflicted with moderate to severe DED [15]. Based on these data and clinical studies, DED can be estimated to involve 40 to 60 million Americans. It is more common in women and involves large numbers of people from the third decade of life onward. This includes the prime age groups for contact lens wear.

Contact lenses represent a foreign body placed in the preocular tear film environment. Contact lenses have a number of effects on the ocular surface and the tear film. The effects of contact lenses on the cornea include hypoxia, a slight elevation in corneal temperature, microtrauma to the cornea, a reduction in corneal metabolic rate, a decrease in the mitotic rate of the epithelium, increased epithelial fragility, compromised junctional integrity, and an increase in lactate [16]. Despite these changes patients with a normal ocular surface accommodate to the presence of a contact lens with remarkable success.

In addition, contact lenses have been shown to have a number of effects on the tear film. Contact lenses disrupt the tear film by thinning and breakup of the film, increasing evaporative tear loss probably by a disruptive effect on the tear lipid layer [17,18]. Soft contact lenses have been shown to allow evaporation of fluid from corneal tissue, actually drawing fluid out of the cornea [19]. It is thought that only about 10% of the normal volume of tears is necessary to maintain the ocular surface. In a patient with an adequate volume of tears and normal meibomian gland secretion, the presence of a contact lens represents a tolerable stress on the tear film. In the absence of these normal adaptive

reservoirs, a contact lens can induce a clinically apparent dry eye state. The condition contact lens–induced dry eye is estimated to occur in 20% to 30% of soft contact lens wearers, and in over 80% of rigid contact lens wearers [20]. Both rigid and soft lenses create a thinned tear film at the lens edge; this is reported to affect adversely mucin spreading and lipid layer reformation. Moreover, the tear film overlying the lens is thinner than a normal tear film leading to more rapid breakup of the prelens tear film. The presence of a contact lens, rigid or soft, represents a stress to the tear film that in a predisposed patient can lead to contact lens–induced dry eye [21].

Recognizing marginal dry eye disease

Because patients who have pre-existing abnormalities of the tear film and ocular surface are at greater risk for contact lens failure, such as discomfort leading to discontinuance of contact lens wear and the development of contact lens–induced dry eye, it is important to screen candidates for contact lens wear before fitting. Screening tests generally identify subjects with abnormalities. Screening tests for dry eye syndrome can include the following, but they have been noted to have limited predictive values.

Dry eye questionnaires
Tear film breakup time
Schirmer test (and similar tests)
Ocular surface staining
Lid margin examination
Tear clearance test
Ocular Protection Index

Although tear osmolarity has been considered the gold standard for the diagnosis of dry eye, the lack of a readily available instrument for measuring this property of tears in a reproducible manner has left clinicians with a number of clinical tests that, taken together, can usually diagnose moderate to severe dry eye conditions. The diagnosis of early or mild dry eye can be more difficult because symptoms usually precede signs and more subtle measures are necessary. In this regard, the use of patient questionnaires has assumed importance in identifying dry eye subjects. These include an assessment of subjective complaints (eg, dryness, grittiness, sandiness, itching, burning, photophobia, and other complaints). In addition, frequency and intensity of symptoms, effect of environmental conditions, limitation of activities because of ocular symptoms, frequency of medications, symptoms of dry mouth, and other questions are included. Frequently used subjective instruments include the McMonnies Dry Eye Questionaire, the Ocular Surface Disease Index, the Dry Eye Questionnaire, and the NEI-VFQ. Although these have been criticized because of a lack of correlation with clinical signs of dry eye, they have power to identify early to moderate dry eye patients who lack many signs of more advanced cases. The use of one of these patient-administered

questionnaires by practitioners can be a very useful tool in recognizing patients who are at higher risk for developing contact lens intolerance. The Dry Eye Workshop report recognizes the potential role of tear osmolarity as a diagnostic tool of choice in DED and recent reports of a new tear collecting system and technology for measuring tear osmolarity on nanoliter samples of tears opens the way to a new clinically relevant method of diagnosing DED, which may change the diagnostic and disease management paradigm [8].

Tear film breakup time (BUT) is a global test for tear film instability [8]. In this test a small amount of 1% sodium fluorescein is instilled in the conjunctival cul-de-sac, and the patient is instructed to blink for 30 seconds and then to stare ahead without blinking. While the examiner is scanning the corneal surface with the broad beam and cobalt blue filter of the slit lamp, the appearance of the first randomly distributed dry spot is noted (usually with a stop watch or in a more sophisticated setting with a video camera with on-screen timer). BUT is usually measured three times in a row and averaged. In normal subjects the BUT is usually in excess of 10 seconds. Using the more sophisticated video recording technology, referent values of 7 seconds have been reported [22]. The test is exquisitely sensitive to the testing methods, such as the amount of fluorescein instilled, width of the interpalpebral fissure, intensity of the light source, and the patient's ability to cooperate and not blink prematurely. With practice, however, standard conditions of measurement can be achieved with reasonably reproducible results. To determine accurately a normal range of BUT under one's testing conditions, perform the test on a series of normal subjects. This test is a useful screening test.

The Schirmer test is the most widely used test to measure aqueous tear production because of its ease of performance, cost, and availability. It has been criticized for lack of reproducibility and discrimination in identifying dry eye. A commercial filter paper strip with a notch is bent and inserted over the lower lid margin at the junction of the middle and outer third of the lid. The patient is instructed to close the eye (both eyes are measured at once); the strip is removed at 5 minutes and the length of wetting of the strip from the notch is measured. Values less than 5 mm of wetting are considered suggestive of aqueous tear deficiency (ATD). This test can be performed after instillation of a topical anesthetic (which abolishes sensory input from the conjunctiva and cornea but not the lids and lashes). Most practitioners, however, consider a Schirmer test performed without anesthetic to be more predictive. The degree of stimulation with insertion of the strips and ambient test conditions can lead to variation in results but serially consistent values below 5 mm of wetting at 5 minutes are highly suggestive of ATD. A variant of this test is the phenol red thread test that uses a thread impregnated with phenol red (which undergoes a color change on contact with tears). Insertion of the thread is thought to cause less reflex tearing. Either of these tests should be part of a contact lens screening evaluation. One

should also realize that ocular allergy does not preclude the concomitant development of tear film abnormalities because many patients have intrinsic defects noted in metalloproteinases or because of use of oral antihistamines with their antimuscarinic binding [23].

Ocular surface staining is a commonly used test to measure damage to the ocular surface that is a hallmark of dry eye. There are three stains in use: (1) sodium fluorescein (1%); (2) rose Bengal (1%); and (3) lissamine green B. Fluorescein stains breaks in the corneal epithelium and is best seen using the cobalt blue filter. Rose Bengal stains epithelial cells that are unprotected by an intact mucin layer. Lissamine green B is thought to have staining patterns similar to rose Bengal but causes less irritation and takes slightly longer to become apparent, about 2 to 3 minutes after instillation. Rose Bengal and lissamine green B are more easily visualized on the conjunctival surface with a scleral background [24]. Although the presence of staining is an important sign of dry eye, it is a relatively late sign.

Examination of the lid margins is an important part of an evaluation for dry eye. As noted earlier, evaporative tear deficiency resulting from meibomian gland dysfunction is an extremely common condition and recognition can be subtle. Suggestive signs include increased vascularity of the lid margin and pouting or closure of the meibomian gland openings. Some of these changes are seen with aging and are not pathognomonic. Expression of meibom from the glands is a more reliable sign. With the fingernail pressed onto the lid about 1 mm from the glands openings, it is possible to express lipid from several glands above the fingernail. Normal secretion is clear; in meibomian gland dysfunction the secretion becomes cloudy or coagulated, like toothpaste. This alteration in lipid secretion is characteristic of meibomian gland dysfunction that causes excessive evaporative tear loss.

A newer measure of aqueous tear production is fluorescein tear clearance. In this test 5 μL of 1% fluorescein is instilled in the conjunctival sac. One-minute Schirmer tests are performed at 10-minute intervals. Persistent fluorescein staining of the strips indicates delayed tear turnover and, by inference, decreased aqueous tear production.

The Ocular Protection Index is a new concept based on measuring the BUT and the interblink interval (IBI) [25]. It is based on the idea that an early sign of dry eye is the breakup of the tear film before initiation of the next blink, exposing the ocular surface to drying. By measuring the IBI and BUT a ratio or index (BUT/IBI) can be created; this Ocular Protection Index is equal to or greater than 1 in normals and below 1 in dry eye patients. Although this concept is still new, it provides an easily performed test that should yield valuable information to identify dry eye patients.

Evaluation of the lids looking for abnormalities of lid structure, closure, and blink rate is essential in identifying poor candidates for contact lens wear. The tests listed are by no means exhaustive but rather provide a short list of practical measurements to identify potential contact lens problem patients.

Contact lens wear in patients with dry eye

It is possible for patients with mild to moderate dry eye successfully to wear contact lenses. To improve the chances for success it is necessary to modify the normal fitting and care process. In this regard supplementation of normal tears (and replacement of evaporative tear loss) is accomplished by the use of tear substitutes. Most artificial tear products in multiuse bottles contain preservatives to prevent contamination by microbes. These preservatives can be toxic to the ocular surface; their use in conjunction with soft contact lenses is not recommended. Preservative-free artificial tear can be used safely with soft and rigid lenses. In addition, the newer ''transiently preserved'' preparations are also compatible with contact lenses.

Although all contact lenses cause increased evaporative tear loss, certain types of soft lenses are thought to demonstrate less of a desiccating effect in patients with dry eye. It is recommended to fit patients with a hydrogel lens from Group I of the United States Food and Drug Administration lens categories. These lenses have less than 50% water content and are nonionic. This guideline is based on reports that higher water content lenses dehydrate more than lower water content ones.

A study investigated the use of using a hydrogel lens (Proclear) composed of omafilacon A combined with a synthetic analogue of the naturally occurring phospholipid, phosphatidylcholine. The resultant surface resembles that of the cell membrane of mammalian cells and is thought to be biomimetic. Initial studies reported that these lenses were resistant to dehydration and deposit formation [21]. A subsequent clinical trial demonstrated that the use of these lenses in patients with mild to moderate dry eyes resulted in a decrease in ocular staining, and an increase in comfort and wearing time [26]. Similar results have been reported with hioxofilocon contact lenses [27].

The silicone hydrogel lenses with low water content and high oxygen transmissibility are expected to show less dehydration than hydrogel lenses. One clinical study, however, did not demonstrate any difference in comfort and dryness between silicone hydrogel lenses and hydrogel lenses [28].

With careful management and patient compliance it is possible to achieve clinical success for contact lens wear in a motivated patient with dry eye. In patients with moderate to severe dry eye, those patients with Schirmer tests of 2 mm or less, more than mild ocular staining, or signs of clinically apparent surface inflammation, contact lens wear is contraindicated. The risk for serious, sight-threatening problems, such as infection, is too high. Contact lens–induced changes in tear flow, evaporative tear loss, the development of lens deposits that predispose to bacterial attachment, and the loss of antimicrobial ocular defense mechanisms in dry eye render the patient at risk for infection. The presence of a chronic infectious condition, such as blepharitis, is a contraindication to lens wear. Contact lens wear is contraindicated in all patients with Sjögren's syndrome (with the exception of special situations in which therapeutic contact lenses are used).

The use of therapeutic contact lenses in dry eye

Although the previous discussion has focused on identification of patients at risk for contact lens–induced dry eye and its attendant dangers, contact lenses have been used in patients with dry eye in certain clinical situations. Scleral lenses have been used for over 50 years to manage severely dry eyes. They reduce the frictional forces of the upper lid on the cornea and can act as a reservoir for instilled fluid. With the advent of newer materials with greater oxygen transmissibility, there has been renewed interest in both rigid scleral lenses and semiscleral soft lenses. The use of rigid scleral lenses is generally confined to severely desiccated eyes not amenable to other forms of therapy.

Hydrogel bandage lenses can be very useful in the management of a number of recalcitrant ocular surface disease states associated with dry eye. Because they provide a moist covering to the cornea and serve as an interface between the upper lid protecting the cornea from the frictional forces of the upper lid, they have found a place in the management of (1) filamentary keratitis; (2) mucin-deficient states, such as ocular pemphigoid, erythema multiforme, and chemical burns; (3) exposure keratitis; and (4) persistent epithelial defects [29]. Caution is urged, however, in their use. A small amount of lid movement is necessary to ensure that the lens does not become stuck to the surface. In addition, supplementation of tears is essential to maintain the moisture to facilitate healing. The use of prophylactic antiinfective medications is controversial because long-term use may encourage the emergence of resistant organisms. Because bandage lenses are frequently in place for extended periods and ocular surface defense mechanisms are compromised in dry eye, the presence of a contact lens (even a high D/k, highly oxygen-permeable) represents an increased risk for infection. The presence of chronic blepharitis (untreated) is a contraindication. Close monitoring of the patient after insertion and careful counseling to be alert for early signs of infection are essential to minimize the risks inherent in this form of therapy. Bandage lenses used with care represent an important adjunct in the management of difficult ocular surface disease problems.

Contact lenses of contemporary design and composition can be used in patients with mild to moderate dry eye with careful attention to identification of patients at higher risk for developing problems and appropriate modification of tear supplementation and wearing schedule. In certain difficult ocular surface disease states the use of contact lenses can be an important tool in successfully treating these conditions, but careful monitoring and patient counseling is essential to minimize the risk for infection.

The use of contact lenses in a patient with ocular allergy

Up to 40% of the population suffers from allergic rhinitis and 80% to 90% of these patients also have some form of ocular allergy [30]. It is common for patients with ocular allergy to present for contact lens evaluation to

correct ametropia. In addition, in certain conditions associated with allergy (eg, keratoconus and vernal conjunctivitis), contact lenses form an important therapeutic modality. It is important to recognize that certain features of ocular allergy alter the ocular response to contact lenses and care must be exercised to avoid complications. Ocular allergy can take different forms, including nonspecific allergic conjunctivitis, seasonal and perennial conjunctivitis, atopic conjunctivitis, vernal conjunctivitis, drug-induced allergic conjunctivitis, and contact lens–associated giant papillary conjunctivitis. This last condition develops in some contact lens wearers and an allergic response has been implicated in its pathogenesis.

Contact lenses and allergic reactions

In patients with various forms of allergic rhinoconjunctivitis, contact lenses play a dual role in instigating a progressive ocular condition, such as giant papillary conjunctivitis, or can be used in a treatment paradigm. The contact lens can act as a barrier or as an antigen depot, extending their exposure to the ocular surface. It is important to realize that the contact lens materials are in themselves inert plastics not capable of eliciting and allergic response. It has been demonstrated, however, that contact lenses are, within minutes of insertion, coated with a biofilm consisting of proteins and possibly lipid components [31]. This biofilm is an important adaptive phenomenon promoting comfort. The biofilm acts as a base for subsequent deposit formation consisting of denatured proteins, mucins, calcium, and lipids. A lens coated attracts bacteria that can contribute to increased surface inflammation. Antigens can bind directly to the coated surface.

Managing contact lens wear in the patient with ocular allergy

There are three key elements in successful contact lens wear in the presence of ocular allergy. First, in patients with seasonal allergies, avoid lens wear during these times of allergic response. As earlier studies of soft contact lenses tolerance in atopic versus nonatopic during the course of a year have shown, 58% of atopics had experienced symptoms compared with 33% of nonatopics ($P = .034$). Detection of eosinophils and neutrophils in the conjunctival scrapings at the first examination seemed to predict poor contact lens tolerance. History of an atopic condition increased fivefold the risk of experiencing various external eye symptoms during the prolonged use of contact lenses [32]. Similarly, in another study investigating whether underlying rhinoconjunctivitis contributed to contact lens intolerance, 76% of those with allergy exhibited intolerance compared with 60% of those with no allergy. Furthermore, the frequency of seasonal exacerbation of eye symptoms when using contact lenses was significantly higher in subjects with allergy compared with those without allergy (spring, 49% versus 19%, $P < .0001$; summer, 35% versus 20%, $P < .001$; fall, 27% versus 12%, $P < .0001$). The

chronic use of continuous-wear contact lenses in patients with allergic rhinoconjunctivitis seems to contribute to contact lenses intolerance [33]. Interestingly, comfort noted between the newer soft silicone and increased gas permeability reflected a higher satisfaction of comfort (56%) than rigid gas permeable lens (14%), whereas 63% of nonatopic and only 47% of atopic subjects described their lenses as very comfortable to wear [34].

Second, make sure that patients use a regular effective lens cleaning regimen. The use of enzymatic cleansers to remove deposits and limit further deposit formation is important in reducing allergy-enhancing events. An alternative strategy is the use of daily disposable lenses to achieve the same result. In a study evaluating the impact of daily disposable lenses versus patient's standard chronic wear lenses, 67% reported that the 1-day disposable lenses provided improved comfort compared with the lenses they wore before the study, compared with 18% agreeing that the new pair of habitual lenses provided improved comfort, suggesting that the use of 1-day disposable lenses may be an effective strategy for managing allergy-suffering contact lens wearers [35]. Certain lens materials have fewer tendencies to accumulate deposits, although the evidence for this is less than convincing. Because avoidance of significant lens deposits is a critical feature, the use of extended-wear lenses is contraindicated.

Third, the use of topical antiallergic medications while lenses are in place is not recommended. Although it is possible to use these agents after lens removal but if the condition is symptomatic enough to require pharmacologic treatment, a better course is to discontinue lens wear until the allergic condition is under control with topical agents. There are ongoing clinical investigations comparing two allergy drops for enhancing comfort and performance of contact lens wear (epinastine and olopatadine) (http://clinicaltrials.gov/show/NCT00489398, accessed October 21, 2007). In addition, there are actual studies to evaluate the efficacy and safety of an antiallergy drug, antihistamine, and mast stabilizing agent (ketotifen) with a contact lens compared with placebo in preventing ocular itching associated with allergic conjunctivitis. The primary outcome is ocular itching; conjunctival, ciliary, and episcleral redness; chemosis and mucus discharge; tearing; and lid swelling (http://clinicaltrials.gov/show/NCT00445874, accessed September 26, 2007). It is important to note that the use of topical vasoconstrictors has been associated with hypoxia of the ocular surface epithelium.

Summary of contact lens use in patient with ocular allergy

Contact lenses should be used with caution in patients with ocular allergy. In patients with seasonal allergy, avoid contact lens use during seasonal flare-ups. Patients with allergy need to have clean lenses with minimal deposit buildup; to this end, patients should use daily wear lenses with rigid disinfecting and cleaning techniques. Alternatively, they should use daily disposable lenses [36]. When such individuals wear contact lenses,

a special set of circumstances arises that increases the risk of ocular infection. The risk is greatest if the lenses are soft and provide for little tear exchange beneath their surface. Under such circumstances, limited tear flow allows for a greater buildup of lens deposits and metabolic wastes, while permitting increased tear evaporation from the lens surface [37–39].

Avoid the use of topical antiallergy agents while lenses are in place, in particular vasoconstrictor agents, until studies demonstrate that concomitant use with specific agents is not deleterious. Extended-wear contact lenses are contraindicated in patients with ocular allergy. In general, the use of contact lenses is contraindicated in patients with vernal conjunctivitis.

With careful attention to recognizing the patient with ocular allergy, regular monitoring, and patient compliance to lens care, successful contact lens wear can be achieved in most patients with ocular allergy [36].

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