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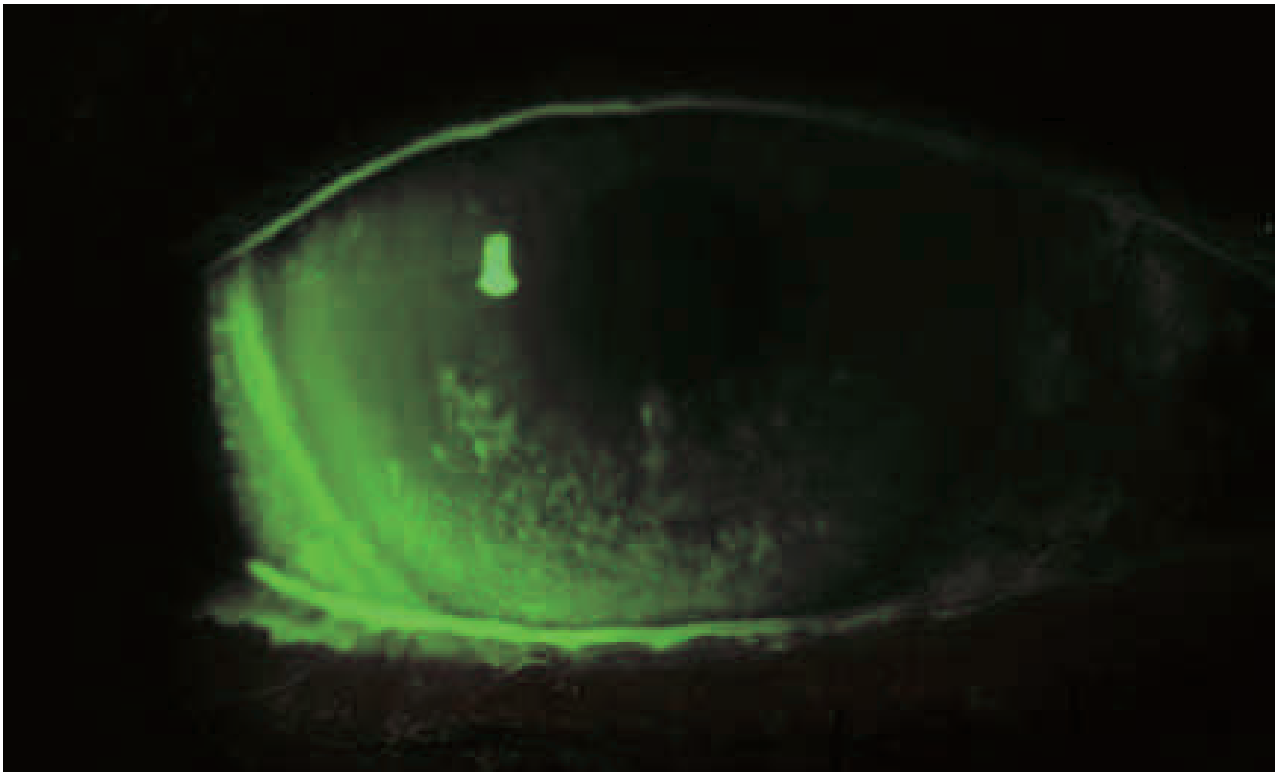

CLINICAL RESEARCH

Corneal Dystrophy Adds to the Frustration of a Dry Eye Patient

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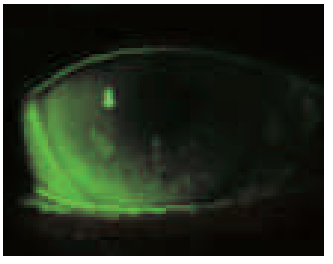

On the Cover

En face view of a cornea with punctate fluorescein staining consistent with dry eye disease (DED). DED is a common inflammatory disorder that needs to be managed effectively, all the more so in individuals undergoing ophthalmic surgery. In this issue, Karpecki et al. (p. 19) offer guidance on the peri-surgical management of DED, as well as best practice for collaborative management of surgical patients by optometrists and ophthalmologists. (Image courtesy of E. Bitton)

Shire

From **innovation**
to **inspiration**.

PRMCDA/CA//0264 November 2017

B. Ralph Chou, MSc, OD, FAAO
Editor-in-Chief

Having attended both the CAO Congress and the American Academy of Optometry Annual Meeting this year, just like I did 38 years ago when I entered practice, I can look back across this time to see how our profession's interests have developed and changed.

Canadian Optometry in 1979 was still essentially a drugless profession. While we were taught about ocular diagnostic and therapeutic drugs, the main emphasis in clinical training was in refraction, and prescribing and dispensing of optical corrections with a smattering of contact lens fitting and management, low vision care, binocular vision management and by today's standards, very basic ocular health assessment. Schiøtz tonometry was being supplanted by applanation and non-contact tonometry, the first autorefractors were just starting to appear, and glass spectacle lenses and contact lenses made with PMMA and HEMA dominated in the dispensary. Dilated fundus examinations were yet to become part of the standard of practice. Visual fields were painstakingly plotted with tangent screens and arc perimeters as the Goldmann perimeter made its debut.

Fast forward to this year. In most jurisdictions in North America, optometry more resembles medical ophthalmology (with maybe a couple of exceptions) and computerized technology can be found in all aspects of clinical care. Glass has mostly disappeared from the spectacle lens market, replaced by a wide variety of organic materials, while in the contact lens market HEMA and PMMA were long ago relegated to museum shelves. Wavefront technology has produced spectacle, contact and intraocular lens corrections that we could only dream of four decades ago. Technological advances such as OCT have revolutionized our ability to diagnose and manage many eye conditions. Computerized equipment has also improved our ability to assess visual fields and manage binocular vision conditions. A wide variety of drugs is available for optometric management of ocular diseases.

Our patients have benefitted greatly from our embrace of modern technology. However, the technology is not without its limitations. We must take advantage of the new testing equipment while understanding what it cannot do and what alternatives exist to cover the gaps. Only then can we be sure that the benefits to our patients are real. ●

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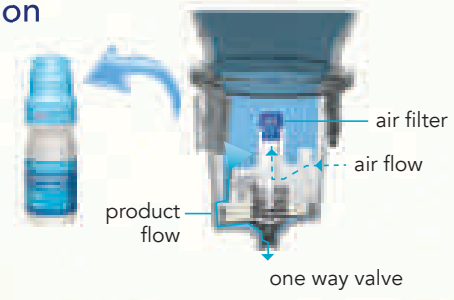

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BAYER

B. Ralph Chou, MSc, OD, FAAO
Rédacteur en chef

Après avoir participé cette année au Congrès de l'Association canadienne des optométristes (ACO) ainsi qu'au Congrès annuel de l'American Academy of Optometry, tout comme je l'ai fait il y a 38 ans quand j'ai commencé à exercer à titre de professionnel, je suis en mesure de voir l'évolution et le changement des champs d'intérêts de notre profession au fil du temps.

En 1979, les optométristes canadiens ne prescrivaient essentiellement aucun médicament. Bien qu'on nous enseignait ce qu'étaient les médicaments de diagnostic et de traitement oculaires, l'accent de la formation clinique était mis sur la réfraction, et sur la prescription et la distribution de corrections optiques, avec un peu d'ajustement et de gestion des lentilles cornéennes, de soins pour vision faible, de prise en charge de la vision binoculaire et, selon les normes d'aujourd'hui, une évaluation très sommaire de la santé oculovisuelle. La tonométrie de Schiøtz était supplantée par la tonométrie à aplanation et la tonométrie sans contact, les premiers autoréfracteurs commençaient tout juste à apparaître, et les lentilles de lunettes en verre et lentilles cornéennes avec PMMA et HEMA dominaient dans le dispensaire. Les examens du fond d'œil à pupilles dilatées ne faisaient pas encore partie des normes de pratique. Les champs visuels étaient laborieusement représentés à l'aide d'écrans tangents et d'arcs périmétriques, alors que le périmètre de Goldmann faisait ses débuts.

Revenons maintenant au présent. À peu près partout en Amérique du Nord, l'optométrie ressemble davantage à l'ophtalmologie médicale (peut-être à quelques exceptions près), et l'on retrouve la technologie informatique dans tous les aspects des soins cliniques. Le verre a essentiellement disparu du marché des lentilles de lunettes pour être remplacé par une vaste gamme de matières organiques, tandis que sur le marché des lentilles cornéennes, les HEMA et PMMA ont été relégués depuis longtemps aux rayons des musées. La technologie Wavefront a permis de produire des corrections au moyen de lentilles de lunettes, de lentilles cornéennes et de lentilles intraoculaires dont on ne pouvait que rêver il y a dix ans. Les avancées technologiques comme la tomographie par cohérence optique (OCT) ont révolutionné notre capacité de diagnostic et de prise en charge de nombreux problèmes oculovisuels. L'équipement informatisé a aussi amélioré notre capacité d'évaluer les champs visuels et de prendre en charge les problèmes de vision binoculaire. On dispose d'une vaste gamme de médicaments pour le traitement optométrique des maladies oculaires.

Nos patients ont grandement profité de notre adoption de la technologie moderne. Toutefois, la technologie n'est pas sans limites. Nous devons tirer parti du nouvel équipement de laboratoire tout en comprenant ce qu'il ne peut pas accomplir et les solutions qui existent pour combler les lacunes. C'est la seule façon de nous assurer que les avantages pour nos patients sont bien réels. ●

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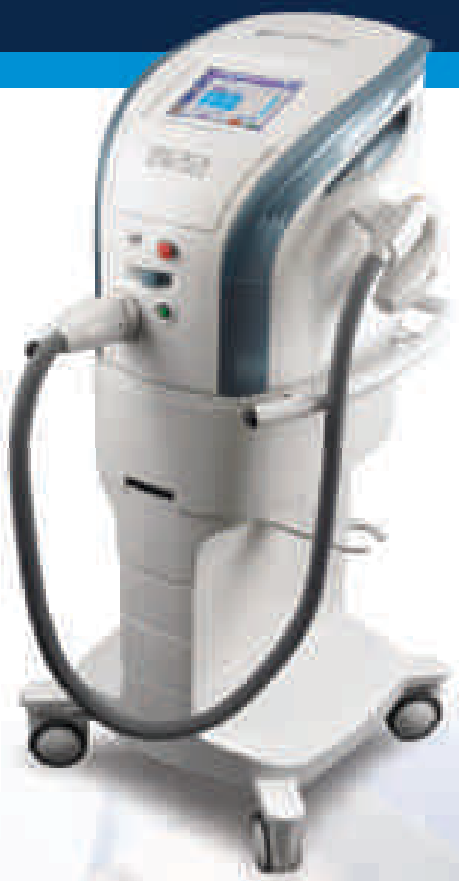

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Corneal Dystrophy Adds to the Frustration of a Dry Eye Patient

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Abstract

PURPOSE
This case report highlights how epithelial basement membrane dystrophy (EBMD), coupled with dry eye, can contribute to symptoms of unstable vision and discomfort. This report also reviews corneal dystrophies and offers eye care practitioners (ECPs) clinical pearls for identifying key features.

CASE REPORT
A 62-year-old Caucasian female presented for a dry eye evaluation due to fluctuating vision and longstanding ocular discomfort, despite ocular lubrication. Anterior segment examination revealed Meibomian gland dysfunction (MGD), upper lid margin staining (ULMS) and anterior blepharitis. The patient was unaware of a pre-existing EBMD and this lack of knowledge contributed to her frustration concerning her unstable vision, which she had solely attributed to her glasses. Management included warm compresses for MGD and targeted preservative-free artificial tears for ULMS and EBMD. Photographs were essential for educating the patient with respect to the irregularities of the ocular surface and its effect on vision. This provided a deeper understanding of the multifactorial nature of her symptoms.

CONCLUSION
Unstable and/or poor vision is among the main reasons why patients consult ECPs and it can be difficult to identify contributory factors. This report highlights that additional chair time may be warranted to educate patients on the multifactorial nature of dry eye and the complexities of corneal dystrophy.

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DISCLOSURES
None that are associated with this manuscript

KEYWORDS
corneal dystrophy, epithelial basement membrane dystrophy, dry eye

INTRODUCTION

Fluctuating vision can be very frustrating for a patient due to its transient and unpredictable nature, which can affect daily tasks, such as reading, driving, and computer use. Common factors that may contribute to fluctuating vision include ocular-surface anomalies (e.g., corneal dystrophy, dry eye), diabetes and hormonal changes (e.g., during pregnancy).[1] Poor vision may also be due to uncorrected refractive error, ocular media opacity (i.e., corneal, lenticular, or vitreal) and retinal conditions.

Dry eye (DE) disease is a widely prevalent condition,[2] which is often accompanied by symptoms of ocular discomfort and vision disturbances,[3] that affects many aspects of a patient's quality of life.[4-6] Fluctuating vision and ocular discomfort are major reasons why patients consult eye care practitioners (ECP).[2, 7]

The cornea needs to remain clear for proper vision. However, in some cases, alterations to any of the layers of the cornea can affect its transparency and ultimately its function. Corneal dystrophies are a group of genetic disorders that cause alterations to the cornea and affect its transparency.[8, 9] Corneal dystrophies arise from a progressive accumulation of abnormal material in any of the layers of the cornea, without inflammation, infection, or neovascularization.[1, 10, 11] Since most corneal dystrophies follow an autosomal dominant inheritance pattern, the examination of family members can be useful for confirming the diagnosis.[1, 10, 12, 13] These dystrophies are progressive in nature, bilateral, but not always symmetrical,[1, 9, 11, 12] and, depending on the layer of the cornea affected, may or may not lead to vision changes.[10, 11, 13]

Traditionally, corneal dystrophies are classified by the anatomical location of the opacity; i.e., anterior (epithelium/Bowman's layer), stromal or endothelial. While some are encountered more commonly than others, Table 1[12-17] summarizes corneal dystrophies according to their effect on vision, which may assist ECPs in differentiating among them.

Table 1: *Corneal Dystrophies* [6-11]

Tissue Affected	Dystrophy	Effect on vision
Anterior	Epithelial Basement Membrane Dystrophy (EBMD) (Map-Dot-Fingerprint)	Normal or reduced (sometimes impaired)
	Meesmann Dystrophy	Not usually affected but may rarely decrease
	Lisch Dystrophy	Sometimes impaired (20/25 to 20/40)
	Gelatinous droplike corneal dystrophy (Familial subepithelial corneal amyloidosis) *	Marked visual impairment (reduced in first decade)
	Reis-Bücklers Dystrophy	Progressive visual impairment, marked by 2nd - 3rd decade
	Thiel-Behnke dystrophy	Progressive visual impairment marked by 2nd - 3rd decade
Stromal	Lattice Dystrophy Type 1	Progressive visual impairment, marked by 3rd-4th decade
	Lattice Dystrophy Type 2	Vision usually normal until 6th-7th decade
	Granular Dystrophy	Vision good < 40 y, progressive visual impairment afterwards
	Macular Dystrophy	Severe visual impairment by 3rd-4th decade
	Schnyder Crystalline Dystrophy	Not usually affected (but might be occasionally)
Endothelial	Fuchs Dystrophy	Progressive visual impairment evolving in marked reduction (worse in the morning and improves during the day)
	Posterior Polymorphous Dystrophy	Rarely progressive visual impairment
	Congenital Hereditary Endothelial Dystrophy	Blurred vision (worse in the morning)

*May also be classified as stromal

This case report describes a symptomatic patient who was frustrated and unclear as to the source of her unstable vision and discomfort, which had several etiologies, including DE and corneal dystrophy.

CASE REPORT

A 62-year-old Caucasian female was referred to a DE clinic due to longstanding symptoms of fluctuating vision and DE. Her most recent eye exam was 5 months prior; a refractive change was noted and new glasses were prescribed. Her general health revealed a history of fibromyalgia, rheumatoid arthritis, hypertension, hypothyroidism and depression. Medication use included Diovan HCT® (Novartis) for her hypertension, Synthroid® (Abbvie) for her thyroid, Xanax® (Pfizer) for her depression, vitamins (E and C) and omega-3 supplements. Her ocular history revealed longstanding complaints of DE, fluctuating vision, pain, irritation and a gritty sensation in both eyes. The patient also reported dryness of the mouth, throat, and nose; she tested negative for Sjögren's syndrome. Additionally, macular drusen were noted and age-related macular degeneration (ARMD) was diagnosed, for which she takes vitamins (Vitalux®, ALCON) and is being followed by a retinal specialist. Mild nuclear sclerosis (grade 1) was noted in both eyes. The patient reported using artificial tears (Systane® ULTRA, ALCON) 8X/day and an ocular ointment at bedtime (Liposic® gel, Bausch + Lomb) to address her ocular discomfort. She remained unsatisfied with her vision despite her new glasses and continued to report ocular discomfort.

A comprehensive DE evaluation revealed severe symptoms (score of 87.5/100) using the Ocular Surface Disease Index (OSDI) questionnaire. Other tear tests were performed and the results are summarized in Table 2; most values were within normal limits. Distance acuities were similar to those reported at the annual eye exam (OD $6/7.5^{+2}$; OS $6/6^{+2}$; OU $6/6^{-1}$). The near acuity revealed less than optimal results (OD 0.8 M; OS 1.0 M at 40 cm using a near point card), which differed from the results at her check-up 5 months previously (OD 0.37; OS 0.50 M at 50 cm).

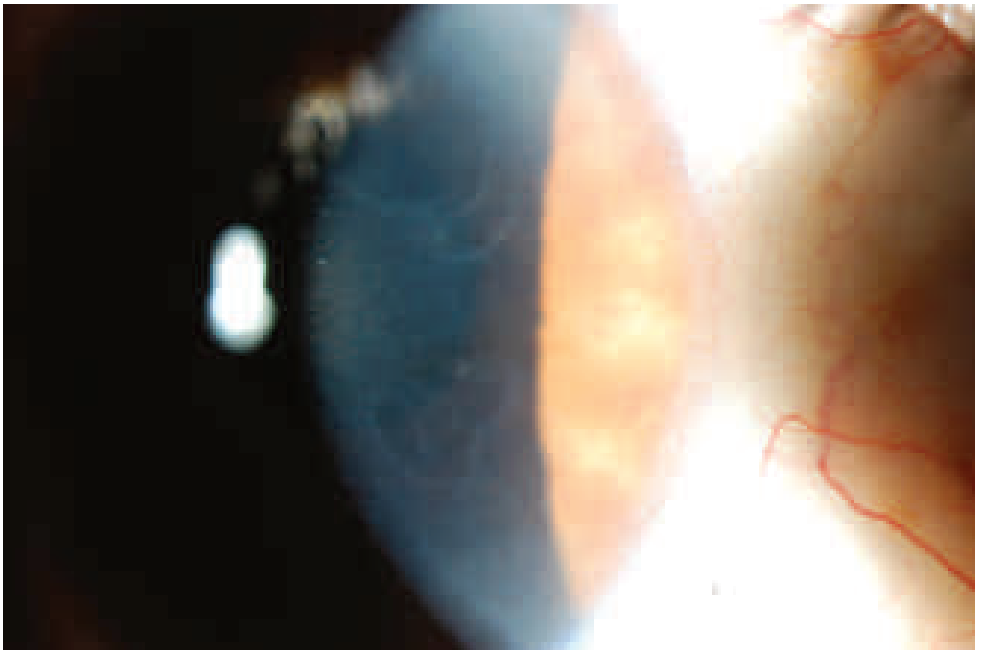
Table 2: *Clinical findings of DE exam*

Tests	Clinical Findings	
OSDI questionnaire	87.5/100	
Osmolarity (TearLab)	OD 288 mOsml/L	OS 291 mOsml/L
Cotton Thread Test	OD 34 mm/15 sec	OS 36 mm/15 sec
Tear meniscus height	0.2 mm OU	
Eyelid margin	Telangiectasia OU	
Lashes	Cylindrical dandruff OU	
Meibomian glands	Yellow, liquid secretions, non-linear with missing glands OU	
Corneal staining	No defects noted OU	
Bulbar Conjunctival Staining	No defects noted OU	
Palpebral Conjunctival Staining	ULMS <20% along the full length, OU	
Tear Break Up Time (TBUT)	OD 3 sec	OS 2 sec
Endothelial cell count	OD 2367 cells/mm^2	OS 2423 cells/mm^2

A detailed anterior segment evaluation revealed redness (telangiectasia) along the eyelid margin, clear gelatinous deposits at the base of a few lashes resembling cylindrical dandruff (CD) and several differently shaped translucent corneal opacities in both eyes. Epilation was performed on the lashes that had CD and a microscopic evaluation confirmed the presence of *Demodex folliculorum,* a common lash mite. Meibomian gland assessment revealed difficulty with expression along with yellow, liquid secretions. Meibography (Meiboscan, 5M Keratograph, Oculus) revealed non-linear and partially filled Meibomian glands in all four eyelids. Ocular staining revealed upper lid margin staining (ULMS) along the full length of the upper palpebral conjunctiva, with a 20% thickness profile (Table 2).

The corneal opacities varied with respect to both shape and size, and could be described as irregular gray geographic patches (resembling ground-glass) and clustered, whorl-like patterns resembling a fingerprint (Figs. 1 and 2). These were observed under white illumination with an oblique broad beam, but were more evident with fluorescein instillation (Fig. 3). No epithelial defects were noted in either eye. While both eyes presented opacities in the pupillary axis, the dystrophy was more advanced in the left eye, which partly contributed to the reduced near acuity. The corneal opacities were photodocumented to establish a baseline and to educate the patient.

Figure 1: *Map (below) and Fingerprint (above) corneal opacities apparent in the right eye*



Diagnosis included evaporative DE secondary to MGD, anterior blepharitis secondary to Demodex, and ULMS. Due to the clinical presentation of corneal opacities, a primary diagnosis of epithelial basement membrane dystrophy (EBMD) was established.

Management for MGD included daily warm compresses using a face towel for 5-10 minutes followed by ocular massage. Since the patient did not own a microwave, eyelid warming masks were not a feasible option. Consequently, patient education was important to explain how the warm face cloth needed to be alternated with another every 1-2 minutes to maintain heat on the eyelids.[18,19] She was encouraged to continue with omega-3 supplementation.

The patient was educated on EBMD, including its permanent nature and associated fluctuating vision and the possibility of recurrent corneal erosions (RCE). Despite this explanation, the patient was still convinced that her subpar vision was due to her new glasses. Management was aimed at decreasing the fluctuating vision, reestablishing a smooth refractive surface, and limiting friction between the lid and the corneal surface. Hence, a non-preserved artificial tear with sodium hyaluronate (I-Drop® Pur Gel, I-Med Pharma Inc.) was recommended at least 4X/day to both reduce friction (ULMS) and enhance the tear layer over the irregular ocular surface (EBMD).

Since the patient was distraught over the EBMD and the fact that her dissatisfaction with her vision may be more permanent than she had anticipated, a discussion about the anterior blepharitis secondary to Demodex was delayed for a follow-up visit 4 months later.

Figure 2: *Topographical map corneal opacities present centrally in the left eye*

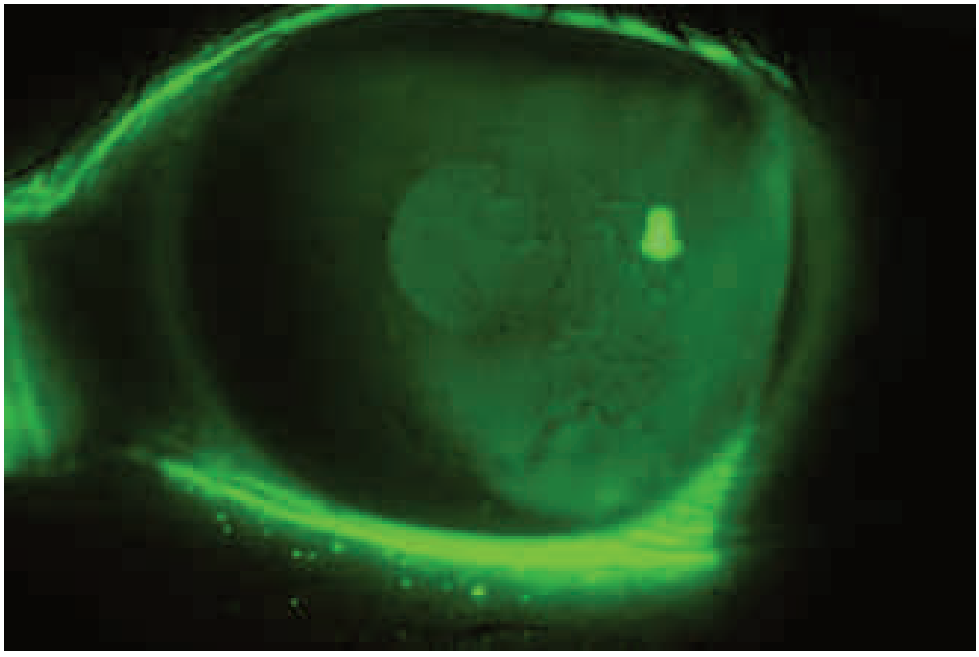
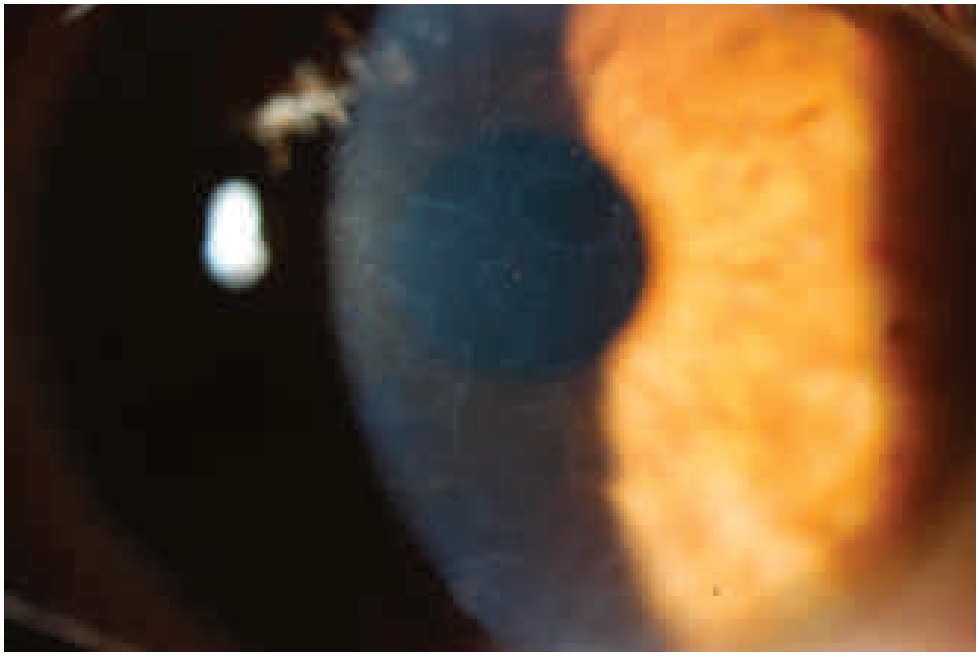


Figure 3: *Fluorescein staining highlights the geographic patterns of EBMD in the left eye*



The patient was properly counseled on the contributing etiologies of her symptoms, which included discomfort and fluctuating vision. Patient education included information on EBMD, dry eyes, cataracts, and ARMD. Although she was still disgruntled, the patient left with a better understanding of the causes of her fluctuating vision and ocular discomfort.

DISCUSSION

This case exemplifies how EBMD, coupled with DE, can contribute to a patient's symptoms of unstable vision and discomfort. Patients typically assume that all vision problems are correctable. However, in this case, the patient needed to be properly educated about the multifaceted etiology of her condition, which may not be completely resolvable. Patients need to be reminded that several components of the eye are responsible for creating optimal vision, from the tear film to the retina. Consequently, any disturbances along that path may contribute to poor/unstable vision. In this case, the tear film, cornea, lens and retina were compromised, and thus extra time was dedicated to educate the patient appropriately. The patient's complaints were focused on fluctuating blurred vision, irritation and pain, despite the lack of any apparent RCE and an updated refraction. A brief discussion of corneal dystrophies with particular attention to EBMD was necessary for the patient to understand its contribution to overall vision.

EBMD is the most common corneal dystrophy.[9, 16] Although EBMD can occur sporadically, there have been some cases of autosomal dominant inheritance.[9, 14, 20] EBMD is defined by a triad of characteristic corneal changes, notably grey geographic patches (referred to as 'Map'), gray-white round or oblong opacities ('Dot'), and curvilinear refractile clustered lines ('Fingerprint').[15, 21] Hence EBMD has also been described as Map-Dot-Fingerprint dystrophy to reflect the patterns observed on the cornea, and may present some or all of these three features.[20] Symptoms include blurry vision, grittiness, foreign body sensation, and pain (especially during RCE episodes).[20, 22, 23]

Histopathology of these opacities reveals thickening of the epithelial basement membrane with abnormal extensions into the overlying epithelium.[12, 20] With the use of fluorescein, these elevated areas appear as negative staining and contribute to decreased tear-film stability.[14, 24] Furthermore, these projections inhibit the normal surface migration of maturing epithelial cells, resulting in cysts containing cellular debris from the degenerating cells.[12] Epithelial cells have hemidesmosomes to reinforce their anchoring to the basement membrane. In EBMD, epithelial cells anterior to the abnormal basement membrane are unable to form hemidesmosomes, which causes poor adherence.[20] Due to this frail attachment, the epithelial layer can easily be separated, causing RCE in 10% of these patients; asymptomatic patients can quickly become severely symptomatic.[1, 9, 21]

When differentiating between corneal opacities, an ECP will consider age of onset, effect on vision and location and appearance of opacities to render a diagnosis. Age of onset is a poor parameter to distinguish between epithelial dystrophies as they all typically occur by the first and/or second decade of life.[12-14] EBMD, which is an exception, appears in early adulthood.[12, 14, 16] Although the present patient was an adult, the age of onset was unknown and therefore could not be used to help in the differential diagnosis.

The effect on vision can be used to distinguish between corneal dystrophies (Table 1). Reis-Bücklers and Thiel-Behnke dystrophies, for example, may be associated with a marked reduction in acuity, while EBMD and Meesmann dystrophy have the potential to impact vision.[12, 13] However, in this patient, fluctuating vision was a poor differentiating indicator because there were other contributing factors, such as DE, cataracts and ARMD. Therefore, in her case, anterior and posterior segment photography was warranted along with strict follow-up to best identify which condition will progressively affect her vision.

Symptoms of ocular discomfort and pain, which this patient reported, also may occur in DE and corneal dystrophies (with the presence of RCE). This patient had no visible RCE at the time of consultation, yet she reported pain, which may be linked to her ocular-surface dryness. Furthermore, the possibility of RCE was discussed, along with the associated abrupt onset of pain, which may occur and prompt consultation. Although RCEs are possible in any of the epithelial dystrophies,[13, 25] people may not consult an ECP due to the variability in pain sensation and discomfort that they may experience.

Consequently, age of onset, effect on vision, and pain are not reliable indicators for identifying a corneal pathology. As a result, the location and appearance of the opacities remain the principal factors in the diagnosis of epithelial dystrophies.[25] It is unlikely that an average practitioner would have clinical experience with the full scope of corneal dystrophies, unless in a corneal specialty practice. Hence, an atlas would be a useful resource for clarifying the clinical presentation of opacities. Table 3[1, 13, 14, 21, 25] provides some clinical pearls to associate characteristic features of corneal opacities with the related epithelial dystrophy. The present case represents a typical EBMD, with representative photographs, in that the clinical presentation included characteristic Map- and Fingerprint-like corneal opacities.

Table 3: *Clinical pearls in identification of epithelial dystrophies*[3,7,8,13,19]

Feature	Location	Dystrophy
Map (geographic-shaped opacities) Dot (putty-like opacities), Fingerprint (whorl-like clustered lines)	Epithelial/diffuse	EBMD
Epithelial vesicle of uniform shape and size	Epithelial/concentrated in the interpalpebral region	Meesmann
Feathery and/or flame-shaped opacities and optically empty microcysts	Epithelial/diffuse	Lisch
Fine reticular opacification (linear, ring-like, or alveolar patterns)	Subepithelial/most dense in central or mid-periphery (extreme periphery is spared)	Reis-Bücklers
Honeycomb patterned opacity	Subepithelial/most dense in central or mid-periphery (extreme periphery is spared)	Thiel-Behnke

EBMD was previously noted in the patient's chart, however the patient was unaware of the condition and its effects. In this case, the lack of knowledge could have contributed to the patient's frustration regarding her vision. Visualization of her opacities via digital photography was quite effective in solidifying her understanding of the causes of her poor vision. This further confirmed that photographs are valuable chair-side educational tools for patients. Typically, clinicians do not own anterior segment cameras. However, a smartphone can easily be propped up against the oculars of a slit lamp to facilitate anterior segment photography.[26]

In this patient, DE was evaporative (MGD), frictional (ULMS) and inflammatory (blepharitis). These conditions are all attracting interest in the optometric field and little is known about their pathophysiology and management. For now, treatment for this patient was limited to artificial tears and warm compresses, and an information sheet was provided to increase her understanding and compliance.

CONCLUSION

Visual impairment, whether permanent or not, is very disconcerting to patients. Consequently, an ECP needs to take time to educate their patient appropriately on contributory factors that can affect vision, such as corneal dystrophies. Although this patient initially presented with frustration regarding her vision and ocular discomfort, patient education allowed her to gain a better understanding of her conditions, especially with regard to EBMD and DE. The use of anterior segment photography proved to be a powerful tool for promoting the patient's understanding and hopefully improving compliance with the recommended management. ●

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Dry Eye Diseases and Ocular Surgery: Practical Guidelines for Canadian Eye Care Practitioners

for the Canadian Dry Eye Disease Consensus Panel

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ABSTRACT:

In 2014, the Canadian Dry Eye Disease Consensus Panel published Guidelines for screening, diagnosis and management of dry eye diseases (DED). These did not address the implications of DED for individuals who are being considered for or have recently undergone ocular surgery. DED is common in certain surgical cohorts, and the perisurgical setting poses specific challenges, both because surgery can complicate preexisting DED and because symptomatic and non-symptomatic DED place the patient at risk of poor surgical outcomes. The Consensus Panel has developed this Addendum to the 2014 Guidelines to offer guidance on DED care before and after ocular surgery.

SHORT TITLE:
Perisurgical Dry Eye Disease

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INTRODUCTION

Dry Eye Disease and the 2014 Canadian Guidelines

Dry eye disease (DED), also known as keratoconjunctivitis sicca, comprises a group of inflammatory ocular-surface diseases that, collectively, are ubiquitous[1, 2] in the general population. DED is highly prevalent in specific subpopulations, including adults over age 50,[1] women, people with long-standing diabetes,[3] and people who wear contact lenses.[4, 5] Rarer conditions associated with severe DED include Sjögren syndrome (SS),[6] Steven-Johnson syndrome, nocturnal lagophthalmos, exposure keratopathy, and graft-versus-host disease.[7, 8]

DED encompasses multiple conditions in which the precorneal tear film is scarce, unstable, inadequately distributed, or of abnormal composition. For instance, tears may be deficient in lipids (Meibomian gland dysfunction, a common cause of evaporative DED), water (e.g., in SS), or mucins (e.g., due to genetic deficiency or loss of conjunctival goblet cell function).[8] Tears in DED become hyperosmolar and may accumulate cytokines that trigger and perpetuate local inflammation. Hyperosmolar tears can damage the corneal and conjunctival epithelia, in part because they carry high levels of inflammatory mediators, such as the degradative enzyme matrix metalloproteinase-9.[9]

DED may present with any combination of ocular dryness, fatigue, redness, burning, itching or stinging pain, foreign-body sensation, and light sensitivity, as well as mucus filaments, eyelid irritation and crusting. Patients may report blurred or fluctuating vision and may experience reduced visual acuity and impaired functioning in visual tasks. Use of dyes for ocular-surface staining during routine exams often detects conjunctival and corneal abnormalities such as superficial punctate keratitis.[10] In extreme cases, DED can lead to corneal ulceration, neovascularization, permanent scarring, and irreversible visual loss.[11]

OCULAR NEUROPATHIC PAIN

Patients who suffer from neuropathic eye pain may describe their subjective experience in terms similar to those used for DED. Common pain descriptors used for both conditions include “burning,” “sharp,” and “gritty,” and both can cause light sensitivity. This similarity of presentation frequently leads to confusion and treatment dissatisfaction, particularly for individuals who develop persistent or intractable postsurgical pain.[24, 89]

The origins of neuropathic pain are obscure, but nerve damage from various sources may be a proximate cause.[23, 34, 90] Aberrant nerve regeneration and several other central and peripheral neurologic events[22] have been proposed to reinforce the neuropathy, causing the pain to become chronic.[24, 89]

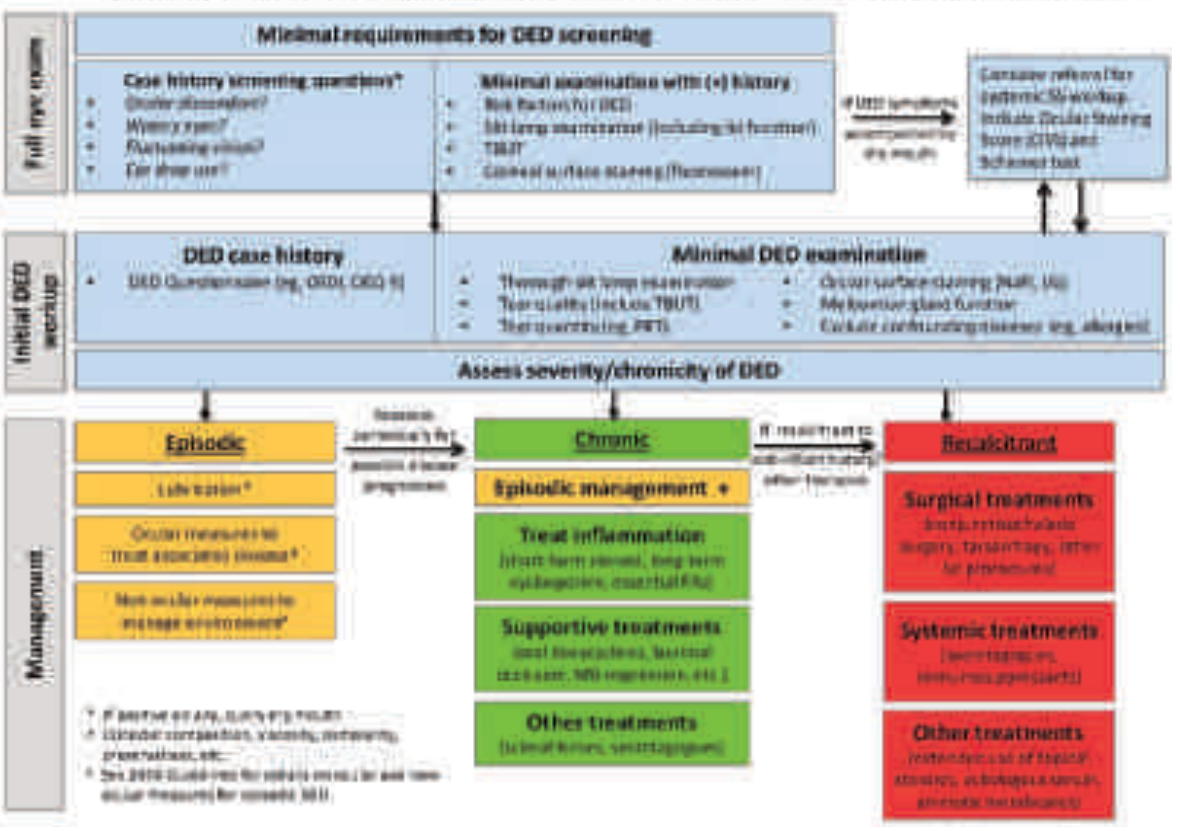
Eye pain may be identified for the first time as neuropathic following surgery. However, neuropathic ocular pain is also widely found in patients with no history of ocular surgery. Pain reported after surgery sometimes follows a pre-existing pattern that might have been misattributed to DED or other causes. A neuropathic origin should be suspected when ocular hypersensitivity, hyperalgesia (exaggerated pain response to suprathreshold noxious stimuli), or allodynia (pain in response to normally non-noxious stimuli) are not commensurate with objective signs of DED, such as corneal and conjunctival staining.

This possibility can be tested by instilling anesthetic drops, thus blunting nociceptive signals from corneal neurons.[22, 24] By definition, neuropathic pain originates in the brainstem or elsewhere in the central nervous system. Therefore, although this test is not diagnostic, the observation of pain that persists despite topical anesthesia is suggestive of neuropathy. Neuropathic pain is also reported to be poorly responsive to artificial tears, relative to physiologic (nociceptive) pain.[91]

Common comorbidities include general (non-ocular) neuropathic pain, as well as depression, anxiety, and sleep disturbance;[22, 92, 93] patients reporting otherwise-unexplained eye pain should be queried about these other conditions as well.

In 2014, the Canadian Dry Eye Disease Consensus Panel presented Guidelines on DED screening, diagnosis and management.[8] This guidance (Fig. 1) offered general principles for diagnosing and managing episodic, chronic, and recalcitrant DED and for monitoring the effectiveness of treatment. Based on these Guidelines, it is essential for all eye care professionals to screen for ocular-surface diseases and manage them appropriately. As discussed in the Guidelines, episodic DED can sometimes be managed with lubricating eye drops, eyelid hygiene, and/or modifications to the living and working environment. Conversely, chronic DED is an inflammatory disease that requires anti-inflammatory treatment.[8] Currently available options to manage ocular-surface inflammation include topical corticosteroids, which are generally reserved for short-term use, as well as essential fatty acids and cyclosporine 0.05% emulsion in castor oil (Restasis®, Allergan Inc.; throughout this Addendum, cyclosporine refers to this formulation).[8] Additional topical anti-inflammatory products, such as lifitegrast 5% and cyclosporine 0.1% in a cationic formulation, may soon reach the Canadian market for use in cases of DED.[12-17] To date, there have been no reports on the perisurgical use of these additional products.

Figure 1: *Diagnosis and management of DED, according to the 2014 Canadian Guidelines*[8]



Scope and Aims of this Addendum

The 2014 Guidelines did not address DED management in individuals undergoing ocular surgery. This topic is significant because of the variety of ways that uncontrolled DED can become problematic for patients undergoing procedures such as cataract surgery or refractive surgery. In addition, ocular surgeries of many types (Table 1) can induce ocular-surface inflammation, which results from direct trauma to the cornea, light toxicity from the surgical microscope, tear-film evaporation during surgery, and irritation due to topical anesthetics, surface antiseptic solutions, and preservative-containing eye drops.[18-21] As a result, surgery can precipitate *de novo* DED or exacerbate the condition in patients with pre-existing symptomatic or asymptomatic DED. Postsurgical ocular dryness and pain that present during the recovery phase are usually transient, but can persist in certain individuals. Chronic pain without significant ocular-surface disease ("pain without stain")[22] can also occur, and may be present before or after surgery; such neuropathic eye pain, resulting from lesions within the somatosensory nervous system,[23] is not readily distinguished from DED. This confusion poses a variety of clinical problems that can be frustrating for both patients and caregivers (see sidebar on *Ocular Neuropathic Pain*).[22, 24]

As discussed below, pre-existing ocular-surface disease complicates presurgical biometry, keratometry, topography and refraction, thus increasing a patient's risk of unsatisfactory visual correction and of refractive regression. DED can degrade the quality of vision (QoV) after surgery, leading to discomfort and dissatisfaction, even among patients whose visual acuity has been corrected effectively. Conversely, measures that restore the tear film, reduce tear osmolarity, or suppress corneal and conjunctival inflammation may improve postsurgical DED symptoms and visual outcomes. Thus, there are several compelling reasons to manage DED, both before and after surgery. Nevertheless, in surgical candidates and others, ocular-surface disease frequently goes unrecognized and untreated.[8, 25] It is crucial for eye care professionals to recognize DED signs and symptoms in surgical candidates, to begin treatment promptly, and wherever possible, to reverse ocular-surface inflammation before proceeding to surgery.

Table 1: *Ocular surgeries that may require DED diagnosis and management*

Type of surgery	Surgery induces or complicates DED?	DED complicates surgery?	DED highly prevalent in surgical cohort?	References
Refractive				
LASIK	Yes	Yes	Yes	This article
PRK	Yes	Yes	Yes	110
Cataract				
Phacoemulsification	Yes	Yes	Yes	This article
Corneal/conjunctival				
Penetrating keratoplasty	Yes	Yes	Yes	111-113
Endothelial keratoplasty	Yes	Yes	Yes	111-114
Pterygium excision	Yes	No	Yes	115
Conjunctivochalasis removal	No	Yes	Yes	116-119
Vitreo-retinal				
Vitrectomy	Yes	?	Yes	120, 121
Eyelid				
Blepharoplasty	Yes	No	Yes	122-124
Ptosis repair	Yes	?	No	122
Glaucoma				
Trabeculectomy	Yes	Yes	Yes	125, 126
Others				
Strabismus repair	Yes	No	No	127

Therefore, we developed this Addendum to the 2014 DED Guidelines for Optometrists specifically to address the perisurgical management of DED. The focus here is on cataract surgery, mainly phacoemulsification with intraocular lens (IOL) implantation and refractive procedures such as laser-assisted in situ keratomileusis (LASIK). Comorbid DED in glaucoma is discussed in the sidebar on *Dry Eye Disease in Glaucoma*.

DRY EYE DISEASE IN GLAUCOMA

DED is a common comorbidity with glaucoma, occurring in 15% to 59% of glaucoma patients.[94-99] This association can be explained at least in part by medicated eye drop polypharmacy, and specifically by the presence and duration of exposure to irritants found in medicated and non-medicated products, including active pharmaceutical ingredients and preservatives such as benzalkonium chloride (BAK).[99, 100]

Inflammation in response to BAK may be heightened by the presence of hyperosmolar tears,[101] as is common in DED. Moreover, chronic use of BAK-containing products can lead to increased tear-film osmolarity, and DED can complicate ophthalmic surgery, including procedures for glaucoma.[94] In patients using BAK-containing products, a preoperative course of topical steroids may decrease conjunctival inflammation and increase the rate of successful trabeculectomy; of course, it is essential to monitor intraocular pressure if steroids are used.[102, 103]

Preservative-free artificial tears (ATs) are associated with reduced DED symptoms[97] and should be used in preference to preserved ATs.[8] However, it is rarely feasible for patients with glaucoma to avoid the use of irritating topical medications, especially as the disease progresses and topical monotherapy must be abandoned.[100, 104, 105] For this reason, anti-inflammatory treatment may be considered, to ameliorate DED in patients requiring long-term exposure to topical glaucoma medications.[106] Whereas it has been generally assumed that controlling glaucoma is the primary goal when these two conditions co-exist, recent findings suggest that surface optimization in patients with comorbid glaucoma is compatible with reducing intraocular pressure.[107, 108]

Trabeculectomy and other surgeries for glaucoma temporarily exacerbate DED, but offer the prospect of long-term IOP control with greatly reduced need for topical glaucoma medications. Indeed, 40% of patients no longer require any topical glaucoma medications for up to 3 years post-trabeculectomy.[109] With the advent of less-invasive surgical procedures, it may be possible to allow for better control of IOP at an earlier point in the progression of glaucoma, thus reducing the chronic exposure to irritating topical medications. So-called micro-invasive glaucoma surgeries (MIGS) are *ab interno* procedures, meaning that they are carried out from inside the eye and cause minimal or no trauma to the conjunctiva. MIGS procedures are considered sufficiently low risk that their use can be justified even in individuals with mild to moderate glaucoma.[96] Although direct evidence is still lacking, MIGS procedures are therefore expected to carry less risk of inducing DED, compared to traditional surgery.

The general DED management approaches described in the Canadian Guidelines are considered to be applicable to patients requiring surgery. For this reason, this Addendum focuses on questions that are specific to perisurgical DED care, such as:

What evidence implicates uncontrolled DED in adverse post-surgical outcomes?
How should the presence of DED affect decisions on the timing or appropriateness of a procedure?
How should DED be managed before and after a procedure?
How should optometrists and ophthalmologists co-manage DED in individuals being considered for ocular surgery?

DED, VISUAL FUNCTIONING, AND OCULAR BIOMETRY

The precorneal tear film, the first refractive surface of the eye, functions best when mirror-smooth. The tear film is maintained by neuroendocrine mechanisms that regulate secretory function and the blink rate in response to shifting environmental stresses.[10, 26, 27] A healthy tear film is sufficiently thick, uniform, and balanced with appropriate components to protect the ocular surface from insult and to avoid optical aberrations between blinks.[28-31]

Abnormalities in the tear film, affecting either tear quantity or composition, can lead to aqueous-deficient or evaporative ocular-surface diseases. Surgical trauma compromises tear-film regulation, at least temporarily. For example, loss of tactile sensation at surgically denervated sites in the cornea impairs basal and reflex tearing and reduces the blink rate, leading to a compromised tear film and ocular surface while the damaged nerves regrow.[27, 32-34]

DED affects QoV without necessarily degrading visual acuity.[1, 29, 35] For instance, DED is associated with glare, impaired contrast sensitivity and symptoms of higher order aberrations (HOAs); patients experience fluctuating vision during the interval between successive blinks, due to a non-uniform and unstable tear film.[27] QoV is an important predictor of daily function, especially for tasks that place high demands on the visual system such as reading or driving.[36, 37]

In patients undergoing ocular surgery, visual disturbances related to DED may increase the risk of postsurgical patient dissatisfaction. For both refractive procedures and cataract surgery with IOL implantation, surgical planning requires accurate keratometry and/or topography. Uncontrolled DED can alter the shape of the cornea,[38] reducing the accuracy and precision of biometric findings. Tear hyperosmolarity is associated with greater statistical scatter in keratometric readings. Ocular-surface irregularity and instability reduce the precision of preoperative IOL and refractive calculations, increasing the risk of suboptimal refraction following surgery, especially when toric or multifocal implants are used.[39] In addition, even for individuals who achieve good postsurgical acuity, DED can reduce QoV over a period of weeks to months, and sometimes longer.[40]

Table 2 summarizes various goals for DED treatment in the general population and in candidates for ocular surgery.

Table 2: *Goals of DED management*

For all individuals with DED	For individuals with DED undergoing surgery
• To ameliorate eye discomfort and fatigue • To prevent corneal erosion and surface anomalies associated with ocular-surface disease and tear hyperosmolarity[3, 28, 32, 39] • To prevent optical aberrations that reduce visual quality (e.g., blurring, glare, loss of contrast sensitivity)[30, 35] • To improve performance and facility in demanding visual tasks (e.g., reading[36] and driving[37])	• To achieve more accurate and precise biometric/keratometric refractive measurements, allowing IOL power or LASIK surface parameters to be calculated more confidently • To improve postsurgical visual acuity and quality of vision • To prevent or minimize postsurgical DED[40]

The Growing Need for Collaborative Care for DED

Several demographic and societal trends have increased both the urgency and the burden of managing DED. First, both DED and ocular surgeries are increasingly common as the population ages. Second, the increasing reliance on and use of electronic devices can cause or exacerbate DED by decreasing the user's blink rate.[41, 42] People who use these devices require a high level of visual functioning and may seek surgical intervention specifically to improve their QoV. Third, ophthalmic technology itself has changed with the introduction of measurement approaches that offer unprecedented precision in ocular biometry but that rely on a healthy tear film if they are to be used optimally. Likewise, multifocal and toric IOLs offer the prospect of corrected near and distance vision, but they appear to be more sensitive than earlier-generation IOLs to both visual aberrations and errors in biometry.[43] This difference is intrinsic to the IOL technology, but dissatisfaction also results in part from higher expectations, including the desire for optimal visual functioning with minimal dependence on distance or reading glasses.

For all of these reasons, the growing need for diligent pre- and postsurgical DED care is placing increasing demands on caregivers' time. Efficient approaches, including collaborative DED management by optometrists and ophthalmologists, will be needed to meet these demands.

CATARACT SURGERY

If we extrapolate from 2014 data from a single province, approximately 450,000 phacoemulsification procedures may be carried out annually across Canada, making cataracts one of the most common reasons for ocular surgery.[44, 45] The demand for this surgery is projected to more than double by 2036.[46]

The current standard of care for cataract extraction is phacoemulsification followed by implantation of an IOL, which may be either monofocal or premium. The latter type of IOL includes a variety of designs that allow for correction of distance, reading, and intermediate vision, generally resulting in less dependence on glasses or contact

lenses. Depending on their design, premium IOLs may be prone to glare and halos, so it is important to minimize other visual disturbances in these patients, including aberrations related to DED.[47, 48] Interestingly, cataracts themselves can induce HOAs,[49, 50] and this effect on QoV is complicated by poorly controlled DED.[29, 30]

Pre-existing DED is common among individuals with cataracts, since some risk factors (notably advancing age, diabetes[3], and female sex[51]) predispose patients to both conditions.[52] However, DED commonly goes unrecognized and untreated in patients undergoing cataract surgery. Trattler et al. reported that, out of 136 American patients undergoing cataract surgery, while only 22% had a prior diagnosis of DED, a larger proportion showed objective signs of surface disease (tear break-up time (TBUT) ≤5 seconds, 63%; corneal staining, 77%) suggesting widespread underdiagnosis.[25] Interestingly, subjective symptoms of DED were less common, with only 31% reporting stinging and 41% reporting foreign-body sensation. These findings are consistent with previous reports suggesting that patient self-reporting is an unreliable screening tool for DED.[41, 53, 54]

Uncontrolled DED limits the accuracy of preoperative biometry, leading to errors in IOL power or placement.[41, 54] This effect has been clearly demonstrated using repeated readings in patients presenting for cataract surgery. Epitropoulos et al. reported that the mean difference between two successive keratometric readings was 0.28 D among individuals with hyperosmolar tears (n=100 eyes) versus 0.13 D among controls (n=50 eyes). Calculated IOL power differences were up to 5.5 D among 100 eyes with hyperosmolar tears, and the frequency of an IOL power difference ≥0.5 D was significantly higher with hyperosmolar versus normal tears (p=0.02). In addition, 17% of eyes with hyperosmolar tears but only 2% of eyes with normal tears showed a vector astigmatism difference ≥1.0 D (p=0.01) between readings.[39]

Onset of Symptoms of DED Following Cataract Surgery

Cataract surgery perturbs the ocular surface and induces intraocular and ocular-surface inflammation. In addition, the surgical procedure damages sensory and other neurons, and the resulting denervation reduces tactile and other sensation in the cornea.[40] *De novo* DED symptoms are common following phacoemulsification,[10, 27] but they are usually transient. Corneal hypoesthesia, tear-film instability, and other indicators of DED often resolve within 3 months, probably associated with the beginning of axonal regrowth.[18, 19] Corneal sensation gradually returns to near-preoperative levels over the course of 1 year.[40] In a small subset of patients, however, DED symptoms persist indefinitely.[20, 55] For instance, individuals with diabetes are at increased risk of severe and chronic postsurgical DED.[56]

Topical treatments should be applied consistently following surgery to limit the extent or duration of *de novo* DED.[52] Used alongside topical steroids, lubricants have been reported to improve symptoms of DED and visual functioning, relative to standard postsurgical topical care alone.[57, 58] Jee et al. directly compared the effects of preservative-free versus preserved steroid and lubricant eye drops after cataract surgery in 80 patients (80 eyes) with pre-existing DED. In this prospective, open-label study, patients received the preservative-free or preserved products 4 times daily for 1 month and twice daily thereafter. By Month 1 following surgery, subjects who received the preservative-free topical treatment reported significantly less severe symptoms compared to those who received preserved treatment ($p<0.05$). By Month 2, objective DED measures (staining, tear-film stability, inflammatory markers, and conjunctival goblet cells) were significantly improved with preservative-free treatment.[59]

Lubrication alone may be insufficient to manage the inflammation that drives chronic DED.[60-62] This has been shown most clearly in a randomized, multi-centre study of 233 Chinese adults with moderate to severe DED at baseline. Patients were randomized to twice-daily application of cyclosporine 0.05% or the emulsion that serves as its vehicle, with no other treatment permitted except for artificial tears. Whereas both groups experienced significant symptomatic improvement over baseline, significantly greater improvement was seen in corneal staining at 4 and 8 weeks (p=0.025 and 0.05, respectively) and in the Schirmer score at 4 weeks (p=0.035) with cyclosporine versus vehicle.[60] While no such vehicle-controlled study has been reported in a surgical setting, a prospective, contralaterally controlled study compared topical cyclosporine with saline in 32 patients undergoing bilateral phacoemulsification. In these patients, treatment with topical cyclosporine significantly improved tear-film stability and other measures of DED, relative to saline alone. While patient-reported DED intensity improved by the first month of treatment, clinical benefits became statistically significant by 2 months of cyclosporine treatment.[63]

Exacerbation of DED after Cataract Surgery

For patients with pre-existing chronic DED, anti-inflammatory treatment may improve postsurgical acuity and visual functioning.[47, 64] In a small, prospective, contralaterally controlled, randomized, double-masked study, Donnenfeld et al. compared lubricating eye drops (0.4% polyethylene glycol 400; 0.3% propylene glycol) to topical cyclosporine in patients undergoing bilateral phacoemulsification with multifocal IOL implantation. Of the 14 individuals studied, only 3 had been diagnosed as DED. However, because the mean baseline TBUT was low (approximately 6 seconds), others may have had undiagnosed or marginal DED. Treatment was initiated 1 month before surgery and maintained after the procedure. By Month 2 following surgery (i.e., after 3 months of topical treatment), uncorrected visual acuity was significantly better for eyes treated with cyclosporine than with lubricants alone (p=0.005). Contrast sensitivity was also improved with cyclosporine treatment, and there was a numeric trend toward greater tear stability at Month 2. Furthermore, corneal staining, which worsened from baseline in lubricant-treated eyes, improved significantly in cyclosporine-treated eyes (p=0.034 for the between-group difference at Month 2).[47]

Other studies have explored the postsurgical use of topical cyclosporine in cataract surgery.[63-65] It has been suggested that some treatment effects are seen within weeks of phacoemulsification[65] and other surgeries,[66] but these claims are difficult to evaluate, given that the established benefits of cyclosporine occur with longer-term use (≥3 months).[8, 54, 61]

REFRACTIVE SURGERY

LASIK and related photorefractive procedures are widely used to improve uncorrected visual acuity. These procedures generally lead to favorable outcomes and high patient satisfaction.[67, 68] However, postoperative dry eye is a possible complication of these procedures and a cause of discomfort, reduced vision, and overall dissatisfaction. As with cataract surgery (above), DED following LASIK is associated with the effects of surgical trauma on the ocular surface, including loss of sensation in the cornea[40] and suppression of blinking and both reflex and basal tearing.[69] Pre-existing DED is likely common and underdiagnosed, given that contact lens intolerance, which has been linked to DED, is a common motivator for patients requesting LASIK.[67]

Dry Eye Disease as a Factor in Patient Selection

Because refractive surgeries are elective, only individuals who have or can attain adequate ocular-surface health are considered candidates.[2, 32, 70, 71] Severe DED due to SS or other immune causes is usually considered a contraindication for surgical refractive treatment. However, some reports suggest that even these individuals may be candidates for LASIK if the condition is successfully managed before the procedure.[68, 72] To this end, pre-existing DED should be managed in a stepwise manner (Fig. 1), using therapies described in the 2014 Guidelines.[8] Notably, one retrospective study found that cyclosporine treatment for an average of 3.2 months (range 1–12 months) enabled those with mild DED to proceed with refractive surgery.[73]

Alternative photorefractive surgeries, including flapless procedures such as small-incision lenticule extraction (SMILE), appear to cause less nerve damage and less severe DED than does LASIK.[74, 75] Whether these newer procedures are preferable for patients at high risk of DED has not yet been established.[69, 72]

Transient versus Chronic DED Following LASIK

Following LASIK, tear-film instability and other DED signs and symptoms usually resolve spontaneously, but chronic postsurgical DED, persisting 6 to 12 months after LASIK, has been reported in 0.8% to 20% of patients.[70, 76] The risk of chronic post-LASIK DED increases with age and is greater in women than in men.[71, 73] It has also been suggested that hyperopic LASIK procedures carry a greater risk of chronic DED than does myopic LASIK.[73, 77, 78] Following hyperopic LASIK, patients with pre-existing DED and those who develop chronic DED appear to be at high risk of refractive regression.[70, 77]

In addition, pre-existing mild or subclinical DED appears to be a risk factor for chronic post-LASIK DED.[32] One prospective study followed 139 eyes undergoing LASIK. Subjects were excluded if they had a definitive diagnosis of DED, but could be included with isolated symptoms such as mild or moderate corneal staining or TBUT<5 seconds. In this population, presurgical corneal staining and low tear production were associated with DED persisting 1 year after LASIK.[76] Similarly, patients with reduced tear-film stability at baseline were at significantly greater risk of developing surface abnormalities over at least the first 6 months after LASIK.[79]

Use of topical cyclosporine following LASIK has been explored as a possible supplement to standard post-procedural care for patients with no prior diagnosis of DED. Peyman et al. reported that, in a series of 22 bilaterally

treated subjects, eyes receiving daily cyclosporine were significantly more likely to recover tactile sensitivity within 3 months following LASIK, relative to control eyes ($p \leq 0.011$).[80] The timing of this response is consistent with evidence that the clinical benefits of cyclosporine become significant after ≥3 months of treatment.[8, 61, 66]

Post-LASIK visual acuity is less satisfactory among patients who develop chronic DED relative to patients who have no such adverse effect.[70, 77, 78] In a large (n=565) retrospective analysis, Albietz et al. reported that refractive regression was associated with chronic DED (p=0.008 for DED at Month 12 vs no DED) and with pre-existing ocular-surface staining, reduced TBUT, and reduced corneal tactile sensation.[81] The authors noted that DED in their patients abated with persistent ocular-surface management, involving placement of lacrimal occlusion (punctal plugs), lid hygiene, and consistent use of lubricating eye drops. In eyes with evidence of post-LASIK DED, uncorrected visual acuity and DED signs and symptoms improved in parallel,[81] as has been reported outside the surgical setting.[82]

Taken together, these findings suggest that individuals at risk of post-LASIK DED benefit from topical treatments that restore the tear film and target inflammation.

Exacerbation of DED after LASIK

Salib et al. tested pre-LASIK DED treatment using cyclosporine versus unpreserved artificial tears in patients with a history of DED (n=21 [42 eyes]).[83] Those scheduled to undergo myopic LASIK began twice-daily treatment with the randomized drops 1 month before the procedure. For both of these treatments, corneal-surface staining and subjective DED symptoms improved by the time of the procedure. Topical treatment was maintained over the following 3 months. DED symptoms worsened transiently at the Week 1 observation, with faster recovery seen in the cyclosporine-treated eyes relative to eyes treated with artificial tears. From 3 to 12 months following LASIK, DED symptoms improved relative to the baseline evaluation in both treatment groups. Cyclosporine-treated eyes showed greater predictability in refraction over the 1-year observation period and were more likely to be within 0.5 D of their target spherical equivalent refraction. This difference was statistically significant by Month 3 (p=0.015).[83]

If cyclosporine is to be used in individuals being considered for LASIK, treatment should begin ≥1 month before the procedure and should be maintained for ≥3 months afterwards, to maximize the benefit when DED symptoms are most troublesome. Pretreatment may allow individuals who would not otherwise be considered for refractive surgery to undergo LASIK successfully,[32] and should be considered for those with evidence of mild or asymptomatic DED.[66, 80]

In addition to anti-inflammatory treatment,[66, 80, 83] pre- and post-LASIK therapeutic options include a broad range of options discussed in the 2014 Guidelines,[8] such as lubricating eye drops, scleral lenses, lacrimal occlusion, essential fatty acid supplementation,[84, 85] autologous serum, and oral doxycycline. Perisurgical data on most of these options are highly limited. In one small study of 12 individuals with residual refractive errors after LASIK whose uncorrected vision improved with the application of lubricating artificial tears, punctal plugs appeared to improve visual performance significantly ($p<0.0001$).[86] In addition, Di Pasquale et al. suggested that patients showing continued tear-film instability after ocular-surface inflammation has been addressed may benefit from an eye-warming procedure,[87] to stimulate Meibomian gland secretion and restore tear lipids.[8, 88] In an observational study on patients whose *de novo* DED persisted for 1 year after LASIK, the combination of punctal plugs, topical corticosteroids, and, where indicated, warm compresses, led to subjective symptomatic improvement in DED, as well as significant increases in tear lipid thickness and TBUT.[87]

COLLABORATIVE MANAGEMENT OF DED

Table 3 lists a series of goals for pre- and postoperative treatment of patients who are being considered for ocular surgery. The list is general, in that it could apply equally to cataract and refractive surgery and potentially to other procedures whose outcomes can be compromised by uncontrolled DED. Briefly, all patients who are referred for surgery consultation should be assessed carefully for symptoms and signs of ocular-surface disease. Patients with episodic or chronic DED should be treated prior to surgery with a goal of addressing both the signs and symptoms of DED and stabilizing the tear film and the ocular surface, to ensure that keratometric and topographic data are reliable and precise. Appropriate counseling during this treatment period is also critical to improve the patient's understanding of the condition and to manage expectations. After the procedure, ocular-surface disease may develop or worsen, at least transiently, despite the continued use of DED therapies initiated before surgery. The goal of care during this period should be to manage ocular discomfort and to restore a stable and healthy ocular surface.

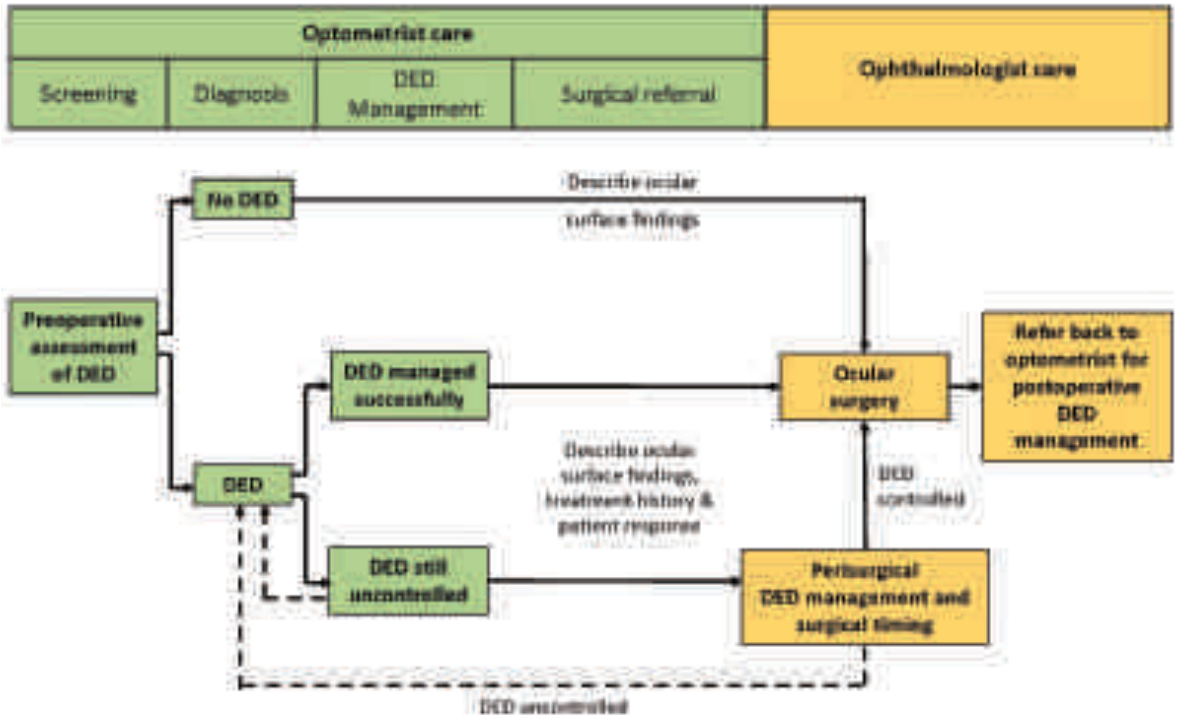
Table 3: *Goals for pre- and post-surgical management of DED*

In the preoperative setting, achieve:	In the postoperative setting:
1. Minimal DED signs on exam	1. Maintain presurgical treatment if ocular surface is stable
2. Minimal and controlled discomfort	2. Otherwise, intensify treatment until ocular surface is stable and returns to an adequate baseline
3. Stable, optimized tear film	
4. Stable keratometric readings (biometric and topographic)	
5. Stable manifest refraction	

While the locus of care before and after surgery may vary somewhat based on the circumstances, in general, optometrists are well-placed to deliver much of the DED care that patients will require before and after surgery. DED is often detected first while the patient is under an optometrist's care. Whether or not the patient is being considered for ocular surgery, the optometrist should assess the ocular surface and initiate appropriate treatment without delay. In all cases, the role of the tear film and ocular surface needs to be addressed with surgical candidates, to reinforce the need for good treatment adherence and to avoid postsurgical disappointment. If premium IOLs are being considered, the patient should be informed that these devices are particularly sensitive to ocular-surface disturbance.

Figure 2 shows a schema for the efficient co-management of patients requiring or requesting ocular surgery, who have been diagnosed with symptomatic or asymptomatic DED. In such cases, the optometrist should describe in a referral letter to the surgeon all ocular-surface findings, including subjective reports and objective evidence leading to this diagnosis. Ideally, the optometrist will manage the condition at this early point, both to improve the patient's immediate comfort and visual function and to streamline management of the surgery. The optometrist's correspondence should describe DED tests and treatments to date, as well as the outcome of these treatments. If appropriate, the optometrist may also recommend that the procedure be delayed to allow time to optimize the ocular surface.

Figure 2: *Proposed schema for the co-management of perisurgical DED*



The ophthalmologist will schedule surgery if the ocular surface is stable and healthy or will refer the patient back to the optometrist for ongoing or enhanced DED treatment. In some cases, the optometrist may request that the ophthalmologist assume care of the patient, including management of the ocular surface/cornea. Following surgery, it is generally appropriate for ongoing DED care to shift back to the optometrist, unless recalcitrant disease or complications manifest that require secondary or tertiary intervention. In a return report, the surgeon should describe the nature and outcomes of the procedure, as well as any changes that may have been made for postsurgical DED management, including changes in prescription and non-prescription topical treatments.

CONCLUSIONS

DED is a chronic inflammatory disorder that optometrists and ophthalmologists encounter on a routine basis. It should be assessed and appropriately treated in all patients. The principles of DED treatment are similar whether or not ocular surgery is being considered and should follow the recommendations of the 2014 *Canadian Dry Eye Disease Guidelines*.[8] However, DED management is particularly important before and after certain surgical procedures, specifically refractive and cataract surgery, because uncontrolled DED may place the patient at risk of less-than-optimal surgical outcomes and, conversely, surgery commonly induces or exacerbates DED. Surgery should therefore be delayed until the ocular surface has been stabilized and an adequate and appropriate tear film is restored, to the maximum extent possible. As outlined in Figure 1, anti-inflammatory treatment for patients with pre-existing chronic DED should be initiated before surgery and maintained for some months afterwards. Consensus recommendations for the management of perisurgical DED are shown in Table 4.

Table 4: *Consensus recommendations for the management of perisurgical DED*

Recommendation 1. For patients with suspected ocular neuropathic pain, it is important to identify associated conditions, such as non-ocular neuropathic pain, depression, anxiety, and sleep disorders.
Recommendation 2. Depending on frequency of use, preservative-free formulations of medicated and non-medicated topical products should be considered for use before and after ocular surgery.
Recommendation 3. Independent of self-reported eye discomfort, patients undergoing cataract surgery should be assessed for signs and symptoms of DED.
Recommendation 4. The ocular surface should be optimized prior to cataract surgery, to increase the accuracy and precision of preoperative biometry and to improve postoperative comfort and visual functioning.
Recommendation 5. Patients with pre-existing symptomatic or asymptomatic DED should be considered for treatment with anti-inflammatory agents prior to surgery, to prevent exacerbation of symptoms.
Recommendation 6. A dedicated DED assessment should be conducted as part of work-up in all patients being considered for refractive surgery.
Recommendation 7. Signs and symptoms of chronic DED, including mild DED, should be evaluated and managed in all candidates for ocular surgery.
Recommendation 8. Patients with ocular-surface staining, tear-film instability, or other signs of DED should be counselled about the risk of exacerbation of DED following ocular surgery.
Recommendation 9. Irrespective of any prior history of DED, patients undergoing ocular surgery should be counselled that DED symptoms can occur following the procedure.

Efficient perisurgical care for patients with DED requires that optometrists and ophthalmologists co-manage the condition and communicate effectively with one another and provide consistent messages to the patient about the ocular-surface findings, risks, and response to treatment. Checklists describing best practices for correspondence between the surgeon and the optometrist are provided in Supplementary Table 1.

Supplementary Table 1: *DED-related information to include in cross-referrals between optometrists and ophthalmologists*

	Optometrist to ophthalmologist	Ophthalmologist back to optometrist
Symptoms	• Sensation • Vision } *Indicate timeline*	• Sensation • Vision • Description of recent surgical procedure • Outcomes (*include postsurgical refraction if done*)
Signs/assessment	Findings • Symptom score (questionnaire) • Tear stability (NIBUT/TBUT) • Corneal (NaFl) and conjunctival staining (LG) • Meibomian gland function • Other testing (e.g., tear osmolarity)	
	Description of DED/ocular surface • Severity • Currently stable? (*if Yes, how long since stability achieved?*)	
Management	• Current and past treatments • Tolerability issues, if any • Adherence history	• Any postsurgical changes made to ocular-surface management, including non-prescription products? (*if Yes, specify products and dosing*)
Recommendations	• Should the patient be scheduled for the earliest possible surgery? • If No, what therapeutic milestones should be achieved before scheduling surgery?	• Instructions for postsurgical care • Next scheduled clinic visit *or* returning to optometrist's care *or* co-management • Patient's goals for vision

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Lignes directrices pratiques pour les professionnels canadiens des soins oculovisuels concernant la sécheresse oculaire et la chirurgie de l'œil

pour le Groupe de consensus canadien sur la sécheresse oculaire

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RÉSUMÉ :

En 2014, le Groupe de consensus canadien sur la sécheresse oculaire a publié un document intitulé Dépistage, diagnostic et prise en charge de la sécheresse oculaire : guide pratique à l'intention des optométristes canadiens. Ce guide pratique ne traitait pas des répercussions de la sécheresse oculaire chez les personnes en voie de subir une intervention chirurgicale de l'œil ou ayant récemment subi ce genre d'intervention. La sécheresse oculaire est courante dans certaines cohortes ayant subi une intervention chirurgicale, et le contexte périopératoire pose des problèmes précis; d'une part parce qu'une intervention chirurgicale peut compliquer une sécheresse oculaire préexistante et, d'autre part, parce la sécheresse oculaire symptomatique et asymptomatique expose le patient au risque d'obtenir des résultats chirurgicaux médiocres. Le groupe de consensus a élaboré cet addenda au guide pratique de 2014 pour offrir des conseils sur les soins relatifs à la sécheresse oculaire avant et après une intervention chirurgicale aux yeux.

TITRE ABRÉGÉ :

Sécheresse oculaire périopératoire

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MOTS CLÉS :

sécheresse oculaire, syndrome de l'œil sec, kératoconjonctivite sèche, phacoémulsification, LASIK, qualité de la vision, douleur neuropathique oculaire

INTRODUCTION

La sécheresse oculaire et le guide pratique à l'intention des optométristes canadiens de 2014

La sécheresse oculaire, aussi appelée kératoconjonctivite sèche, englobe un groupe de maladies inflammatoires de la surface oculaire qui, regroupées, sont omniprésentes[1, 2] dans l'ensemble de la population. La sécheresse oculaire est fortement répandue dans certaines sous-populations, notamment les adultes de plus de 50 ans[1], les femmes, les diabétiques de longue date[3], et les personnes qui portent des lentilles cornéennes.[4, 5] Les affections plus rares associées à une sécheresse oculaire grave sont notamment le syndrome de Sjögren (SS)[6], le syndrome de Stevens-Johnson, la lagophtalmie nocturne, la kératite lagophtalmique et la maladie du greffon contre l'hôte.[7, 8]

La sécheresse oculaire englobe de multiples affections dans lesquelles le film lacrymal précornéen est rare, instable, inadéquatement réparti ou anormalement composé. À titre d'exemple, les larmes peuvent avoir une carence en lipides (dysfonctionnement des glandes de Meibomius; une cause courante de la sécheresse oculaire par évaporation), en eau (p. ex., le syndrome de Sjögren), ou en mucines (p. ex., en raison d'une anomalie génétique ou d'une perte de la fonction des cellules caliciformes conjonctivales).[8] Chez les personnes atteintes de sécheresse oculaire, les larmes deviennent hyperosmolaires et peuvent accumuler des cytokines qui déclenchent et perpétuent l'inflammation locale. Les larmes hyperosmolaires peuvent endommager l'épithélium cornéen et conjonctival, en partie du fait qu'elles contiennent des niveaux élevés de médiateurs inflammatoires, comme la métalloprotéinase-9 de matrice enzymatique de dégradation.[9]

La sécheresse oculaire peut présenter toutes les combinaisons de symptômes suivants : sécheresse, fatigue, rougeur, sensation de brûlure, démangeaisons ou élancement des yeux, sensation de corps étranger, sensibilité à la lumière, filaments de mucus, irritation des paupières et croûtes sur le bord de la paupière. Les patients peuvent rapporter une vision floue ou des fluctuations de la vision, et constater une diminution de leur acuité visuelle et un dysfonctionnement des tâches visuelles. L'utilisation de colorants pour la coloration de la surface oculaire pendant les examens courants permet souvent de détecter les anomalies conjonctivales et cornéennes comme la kératite ponctuée superficielle.[10] Dans les cas extrêmes, la sécheresse oculaire peut mener à l'ulcération cornéenne, à la néovascularisation, à des lésions permanentes et à une perte de vision irréversible.[11]

ENCADRÉ LATÉRAL : DOULEUR NEUROPATHIQUE OCULAIRE

Les patients qui souffrent de douleur oculaire neuropathique peuvent décrire leur expérience subjective en termes semblables à ceux utilisés pour décrire la sécheresse oculaire. Les mots fréquemment utilisés pour décrire la douleur découlant de ces deux affections qui peuvent toutes deux causer une sensibilité à la lumière sont notamment « brûlure », « douleur aiguë » et « démangeaisons ». Cette similitude de présentation des affections entraîne souvent de la confusion et une insatisfaction par rapport au traitement, particulièrement chez ceux qui développent une douleur postopératoire persistante ou incurable.[24,89]

Les origines de la douleur neuropathique sont obscures, mais des dommages nerveux découlant de sources variées peuvent être une cause immédiate.[23,34,90] La régénération aberrante des nerfs et plusieurs autres événements neurologiques centraux et périphériques[22] ont été proposés pour expliquer la neuropathie; ils auraient entraîné la nature chronique de la douleur.[24,89]

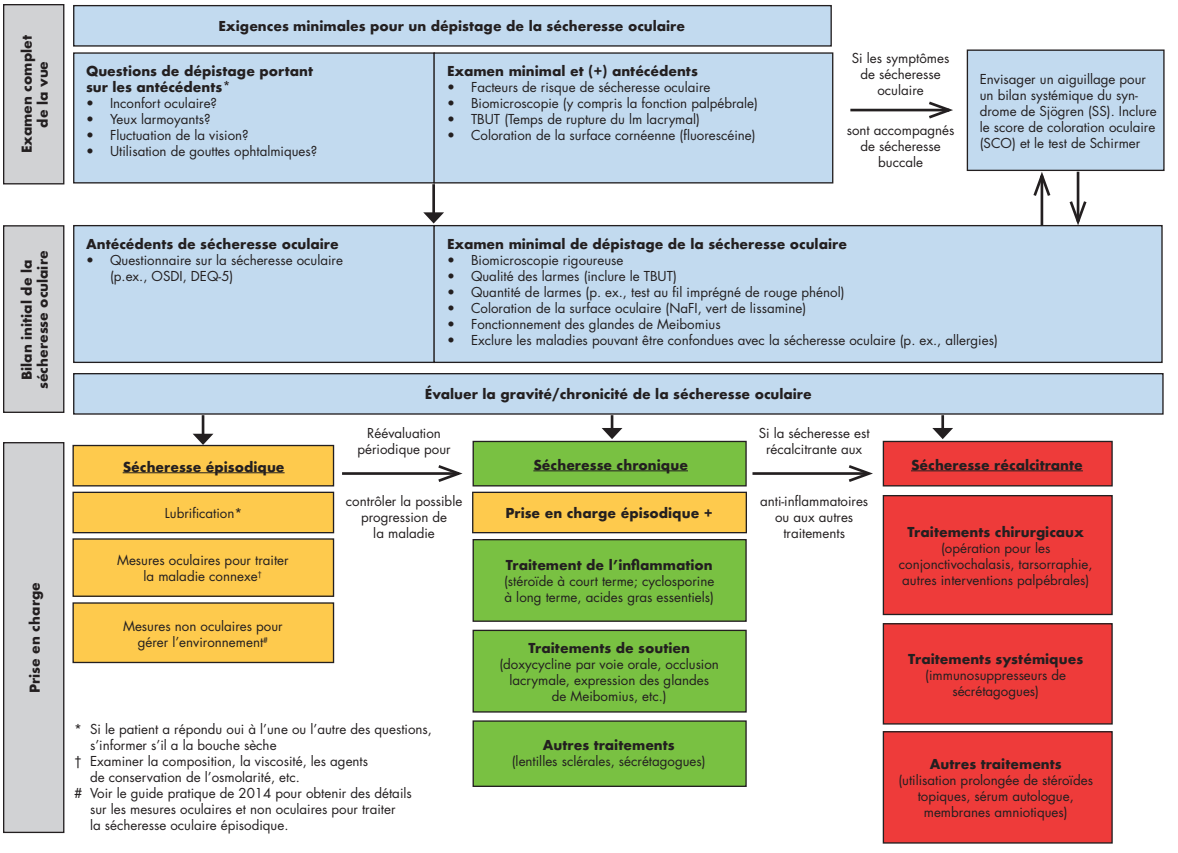
La douleur aux yeux peut être désignée pour la première fois comme étant neuropathique après l'intervention chirurgicale. Toutefois, la douleur oculaire neuropathique est aussi largement répandue chez les patients qui n'ont pas d'antécédents d'intervention chirurgicale oculaire. La douleur rapportée après l'intervention chirurgicale suit parfois un modèle préexistant qui peut avoir été fautivement attribué à la sécheresse oculaire ou à d'autres causes. On doit soupçonner une origine neuropathique lorsque l'hypersensibilité oculaire, l'hyperalgésie (réponse exagérée à la douleur lors d'un stimulus nociceptif supraliminaire), ou l'allodynie (douleur en réponse à un stimulus normalement non nociceptif) ne sont pas proportionnées aux signes objectifs de la sécheresse oculaire, comme la coloration cornéenne et conjonctivale.

Cette possibilité peut être évaluée en instillant des gouttes anesthésiques, émoussant ainsi les signaux nociceptifs des neurones de la cornée.[22,24] Par définition, la douleur neuropathique émane du tronc cérébral ou d'une autre région du système nerveux central. Par conséquent, bien que le test ne constitue pas un diagnostic, l'observation de la douleur qui persiste malgré l'anesthésie topique suggère qu'il s'agit de neuropathie. On a également signalé que la douleur neuropathique répondait mal aux larmes artificielles, en comparaison avec la douleur psychologique (nociceptive).[91]

Les comorbidités courantes sont notamment la douleur neuropathique générale (non oculaire), de même que la dépression, l'anxiété, et les troubles du sommeil;[22,92,93] les patients qui signalent une douleur oculaire autrement inexpliquée devraient être interrogés au sujet de ces autres affections également.

En 2014, le Groupe de consensus canadien sur la sécheresse oculaire a présenté le document intitulé Dépistage, diagnostic et prise en charge de la sécheresse oculaire : guide pratique à l'intention des optométristes canadiens.[8] Ce document d'orientation (fig. 1) offrait des principes généraux pour le diagnostic et la prise en charge de la sécheresse oculaire épisodique, chronique et récalcitrante, et pour la surveillance de l'efficacité du traitement. Selon ce guide pratique, il est essentiel que tous les professionnels des soins oculovisuels effectuent le dépistage des maladies de la surface oculaire et qu'ils prennent en charge ces maladies de manière adéquate. Comme il est exposé dans le guide pratique, la sécheresse oculaire épisodique peut parfois être prise en charge au moyen de gouttes ophtalmiques lubrifiantes, de l'hygiène des paupières et/ou de la modification des milieux de vie et de travail. En revanche, la sécheresse oculaire chronique est une maladie inflammatoire qui requiert un traitement anti-inflammatoire.[8] Les options accessibles à l'heure actuelle pour prendre en charge l'inflammation de la surface oculaire sont notamment les corticostéroïdes topiques, qui sont généralement réservés à l'utilisation à court terme, de même que les acides gras essentiels et l'émulsion de cyclosporine à 0,05 % dans l'huile de ricin (Restasis®, Allergan Inc.; tout au long du présent addenda, la cyclosporine renvoie à cette formulation).[8] Les autres produits anti-inflammatoires topiques, comme le lifitegrast à 5 % et la cyclosporine à 0,1 % dans une préparation cationique, pourraient bientôt être accessibles sur le marché canadien pour le traitement de la sécheresse oculaire.[12-17] À ce jour, il n'y a eu aucun rapport sur l'usage périopératoire de ces autres produits.

Figure 1. *Diagnostic et prise en charge de la sécheresse oculaire, selon le guide pratique à l'intention des optométristes canadiens de 2014*[8]



Portée et objectifs de cet addenda

Le guide pratique de 2014 ne traitait pas de la prise en charge de la sécheresse oculaire chez les personnes ayant recours à une intervention chirurgicale oculaire. Ce sujet est important étant donné les diverses façons dont la sécheresse oculaire non maîtrisée peut devenir un problème pour les patients ayant recours à une opération de la cataracte ou à une intervention de chirurgie réfractive. Par ailleurs, les interventions chirurgicales oculaires en tous genres (tableau 1) peuvent causer l'inflammation de la surface oculaire, laquelle résulte d'un traumatisme direct à la cornée, d'une légère toxicité du microscope chirurgical, de l'évaporation du film lacrymal pendant l'intervention chirurgicale, ou d'une irritation causée par les anesthésiques topiques, les solutions antiseptiques de surface et les gouttes ophtalmiques contenant des agents de conservation.[18-21] Par conséquent, une intervention chirurgicale peut précipiter le retour de la sécheresse oculaire ou exacerber l'affection chez les patients ayant une sécheresse oculaire symptomatique ou asymptomatique préexistante.

La sécheresse oculaire et la douleur postopératoire qui se manifestent pendant la convalescence sont habituellement temporaires, mais peuvent persister chez certaines personnes. Une douleur chronique sans maladie de la surface oculaire (« douleur sans tache »)[22] peut aussi se manifester, et peut être présente avant ou après l'intervention chirurgicale; ce genre de douleur oculaire neuropathique, résultant de lésions dans le système nerveux somatosensoriel,[23] ne se distingue pas aisément de la sécheresse oculaire. Cette confusion pose divers problèmes cliniques qui peuvent être frustrants pour les patients et les fournisseurs de soins (voir l'encadré latéral sur la douleur neuropathique oculaire).[22, 24]

Comme il en sera question plus loin, une maladie de la surface oculaire préexistante complique la biométrie préopératoire, la kératométrie, la topographie et la réfraction, augmentant ainsi le risque pour le patient d'une correction visuelle insatisfaisante et d'une régression réfractive. La sécheresse oculaire peut diminuer la qualité de la vision après une intervention chirurgicale, ce qui peut engendrer de l'inconfort et de l'insatisfaction, même chez les patients dont l'acuité visuelle a été corrigée efficacement. En revanche, les mesures qui rétablissent le film lacrymal, réduisent l'osmolarité des larmes, ou suppriment l'inflammation cornéenne et conjonctive peuvent améliorer les symptômes postopératoires et les résultats visuels. Par conséquent, il y a plusieurs raisons impérieuses de prendre en charge la sécheresse oculaire, autant avant qu'après une intervention chirurgicale. Néanmoins, chez les candidats à l'intervention chirurgicale et chez d'autres personnes, la maladie de la surface oculaire passe souvent inaperçue et n'est pas traitée.[8, 25]Il est essentiel que les professionnels des soins oculovisuels reconnaissent les signes et les symptômes de la sécheresse oculaire chez les candidats à une intervention chirurgicale pour commencer rapidement le traitement et, dans la mesure du possible, pour renverser l'inflammation de la surface oculaire avant de procéder à l'intervention chirurgicale.

Tableau 1. *Interventions chirurgicales de l'œil qui peuvent nécessiter un diagnostic et une prise en charge de la sécheresse oculaire (SO)*

Type d'intervention chirurgicale	Est-ce que l'intervention provoque ou complique la SO?	Est-ce que la SO complique l'intervention?	Est-ce que la SO est très courante dans la cohorte chirurgicale?	Références
Réfractive				
LASIK (kératomileusie in situ au laser)	Oui	Oui	Oui	Le présent article
PRK (photokératectomie réfractive)	Oui	Oui	Oui	110
Cataracte				
Phacoémulsification	Oui	Oui	Oui	Le présent article
Cornée/conjonctive				
Kératoplastie pénétrante	Oui	Oui	Oui	111-113
Kératoplastie endothéliale	Oui	Oui	Oui	111-114
Excision d'un ptérygion	Oui	Non	Oui	115
Enlèvement d'un conjonctivochalasis	Non	Oui	Oui	116-119
Vitréo rétinienne				
Vitrectomie	Oui	?	Oui	120, 121
Paupières				
Blépharoplastie	Oui	Non	Oui	122-124
Réparation du ptosis	Oui	?	Non	122
Glaucome				
Trabéculectomie	Oui	Oui	Oui	125, 126
Autres				
Correction du strabisme	Oui	Non	Non	127

Par conséquent, nous avons rédigé cet addenda au document Dépistage, diagnostic et prise en charge de la sécheresse oculaire : guide pratique à l'intention des optométristes canadiens de 2014 précisément pour assurer la prise en charge périopératoire de la sécheresse oculaire. L'accent est mis ici sur l'opération de la cataracte, principalement la phacoémulsification avec implantation d'une lentille intraoculaire et sur les procédures réfractives comme la kératomileusie in situ au laser (LASIK). On traite de la sécheresse oculaire comorbide chez les personnes atteintes de glaucome dans l'encadré latéral sur la sécheresse oculaire chez les personnes atteintes de glaucome.

ENCADRÉ LATÉRAL : SÉCHERESSE OCULAIRE ASSOCIÉE AU GLAUCOME

La sécheresse oculaire est une comorbidité courante du glaucome, qui se présente chez 15 % à 59 % des patients atteints de glaucome.[94-99] Cette association peut s'expliquer au moins en partie par la polypharmacie des gouttes ophtalmiques médicamenteuses, et plus précisément par la présence et la durée de l'exposition aux irritants qui se trouvent dans les produits médicamenteux et non médicamenteux, notamment les ingrédients pharmaceutiques actifs et les agents de conservation comme le chlorure de benzalkonium (BAK).[99,100]

L'inflammation résultant du BAK peut être accentuée par la présence de larmes hyperosmolaires,[101] qui sont courantes chez les personnes atteintes de sécheresse oculaire. En outre, l'utilisation chronique de produits contenant du BAK peut entraîner une augmentation de l'osmolarité du film lacrymal, et la sécheresse oculaire peut compliquer l'intervention chirurgicale en ophtalmologie, y compris les interventions de traitement du glaucome.[94] Chez les patients qui utilisent des produits contenant du BAK, un traitement préopératoire de stéroïdes topiques peut diminuer l'inflammation conjonctivale et augmenter le taux de succès des trabéculectomies; bien entendu, il est essentiel de surveiller la pression intraoculaire lors de l'utilisation de stéroïdes.[102,103]

Les larmes artificielles sans agent de conservation sont associées à la réduction des symptômes de sécheresse oculaire[97] et devraient être utilisées de préférence aux larmes artificielles avec agents de conservation.[8] Toutefois, il est rarement possible pour les patients atteints de glaucome d'éviter l'utilisation de médicaments topiques irritants, particulièrement lorsque la maladie évolue et qu'ils doivent abandonner la monothérapie topique.[100,104,105] Pour cette raison, on peut envisager un traitement anti-inflammatoire pour améliorer la sécheresse oculaire chez les patients qui devront s'exposer aux médicaments topiques contre le glaucome pendant une longue période.[106] Si l'on présume généralement que la maîtrise du glaucome est l'objectif principal lorsque ces deux affections coexistent, de récentes constatations suggèrent que l'optimisation de la surface chez les patients atteints d'un glaucome concomitant est compatible avec la réduction de la pression intraoculaire.[107,108]

La trabéculectomie et d'autres interventions chirurgicales pour traiter le glaucome exacerbent temporairement la sécheresse oculaire, mais offrent la possibilité d'une maîtrise à long terme de la pression intraoculaire en plus dépendance fortement réduite des médicaments topiques contre le glaucome. En effet, 40 % des patients n'ont plus besoin de médicaments topiques contre le glaucome pendant une période pouvant aller jusqu'à trois ans après la trabéculectomie.[109] Depuis l'émergence d'interventions chirurgicales moins invasives, il peut être possible de permettre une meilleure maîtrise de la pression intraoculaire plus tôt dans la progression du glaucome, réduisant ainsi l'exposition chronique aux médicaments topiques irritants. Ce qu'on appelle les interventions chirurgicales micro-invasives du glaucome (ICMIG) sont des interventions ab interno, ce qui signifie qu'elles sont effectuées à partir de l'intérieur de l'œil et qu'elles causent un trauma minimal ou nul à la conjonctive. Les ICMIG sont considérées comme présentant un risque assez faible pour que leur utilisation puisse être justifiée même chez les personnes atteintes de glaucome léger à modéré[96] Bien qu'on manque encore de données probantes directes, les ICMIG devraient par conséquent présenter moins de risque de provoquer la sécheresse oculaire en comparaison des interventions chirurgicales classiques.

Les approches générales de prise en charge de la sécheresse oculaire décrites dans le guide pratique à l'intention des optométristes canadiens s'appliquent aux patients qui ont besoin d'une intervention chirurgicale. C'est pour cette raison que le présent addenda est centré sur des questions propres aux soins périopératoires de la sécheresse oculaire, notamment les suivantes :

Quels éléments probants désignent la sécheresse oculaire non maîtrisée dans le cas de résultats postopératoires indésirables?

Quelle devrait être l'incidence de la présence de sécheresse oculaire dans la décision concernant le choix du moment ou la pertinence d'une intervention?

De quelle façon devrait-on prendre en charge la sécheresse oculaire avant et après une intervention?

Comment les optométristes et les ophtalmologistes devraient-ils prendre en charge conjointement la sécheresse oculaire chez les personnes retenues pour subir une intervention chirurgicale oculaire?

SÉCHERESSE OCULAIRE, FONCTIONNEMENT VISUEL ET BIOMÉTRIE OCULAIRE

Le film lacrymal précornéen, qui est la première surface réfractive de l'œil, fonctionne de façon optimale quand il est lisse comme un miroir. Le film lacrymal est entretenu par des mécanismes neuroendocriniens qui régulent la fonction sécrétoire et la fréquence de clignement des yeux en réponse aux stress environnementaux changeants.[10, 26, 27] Un film lacrymal sain est suffisamment épais, uniforme, et équilibré avec les composants appropriés pour protéger la surface oculaire des agressions et pour éviter les aberrations optiques entre les clignements.[28-31]

Des anomalies du film lacrymal, qui affectent soit la quantité ou la composition des larmes, peuvent mener à des maladies de la surface oculaire dues à la déficience aqueuse ou à l'évaporation. Un trauma chirurgical compromet la régulation du film lacrymal, au moins temporairement. À titre d'exemple, la perte de sensibilité tactile aux sites dénervés chirurgicalement dans la cornée nuit au larmoiement de base et par réflexe, en plus de réduire la fréquence du clignement des yeux, ce qui compromet le film lacrymal et la surface oculaire, pendant que les nerfs endommagés se reforment.[27, 32-34]

La sécheresse oculaire affecte la qualité de la vision sans nécessairement détériorer l'acuité visuelle.[1,29,35] À titre d'exemple, la sécheresse oculaire est associée à un éblouissement, à un dérèglement de la sensibilité différentielle et à des symptômes d'aberrations d'ordre supérieur; les patients ont une vision changeante durant l'intervalle entre les clignements successifs, en raison d'un film lacrymal instable qui n'est pas uniforme.[27] La qualité de la vision est un facteur prédictif important du fonctionnement quotidien, particulièrement pour les tâches qui sont très exigeantes envers le système visuel comme la lecture ou la conduite automobile.[36, 37]

Chez les patients qui subissent une intervention chirurgicale oculaire, les troubles de la vue liés à la sécheresse oculaire peuvent accroître le risque d'insatisfaction postopératoire du patient. La planification chirurgicale pour l'intervention de chirurgie réfractive et pour l'opération de la cataracte avec implantation d'une lentille intraoculaire nécessite une kératométrie et/ou une topographie exactes. Une sécheresse oculaire non maîtrisée peut altérer la forme de la cornée et[38] réduire ainsi l'exactitude et la précision des constatations biométriques. L'hyperosmolarité des larmes est associée à une plus grande dispersion statistique des mesures kératométriques. L'irrégularité et l'instabilité de la surface oculaire réduisent la précision des calculs préopératoires relatifs à la lentille intraoculaire et à la réfraction, ce qui accroît le risque de réfraction sous-optimale après l'intervention chirurgicale, particulièrement lors de l'utilisation d'implants toriques ou multifocaux.[39] Par ailleurs, même chez les personnes qui obtiennent une bonne acuité visuelle postopératoire, la sécheresse oculaire peut réduire la qualité de la vision au cours d'une période de quelques semaines ou de quelques mois, et parfois plus longtemps.[40]

Le tableau 2 résume les divers objectifs du traitement de la sécheresse oculaire dans l'ensemble de la population et chez les candidats à l'intervention de chirurgie oculaire.

Tableau 2. *Objectifs de la prise en charge de la sécheresse oculaire*

Pour l'ensemble des personnes atteintes de sécheresse oculaire	Pour les personnes atteintes de sécheresse oculaire qui subissent une intervention chirurgicale
• Améliorer l'inconfort et la fatigue oculaires • Prévenir l'érosion cornéenne et les anomalies de surface associées à la maladie de la surface oculaire et à l'hyperosmolarité des larmes[3,28,32,39] • Prévenir les aberrations optiques qui réduisent la qualité de la vue (p. ex., vision floue, éblouissement, perte de sensibilité aux contrastes)[30,35] • Améliorer la performance et la facilité de la demande des tâches visuelles (p. ex., la lecture[36] et la conduite[37])	• Obtenir des mesures réfractives de biométrie/kératométrie plus exactes et plus précises pour calculer avec plus d'assurance les paramètres de la puissance des lentilles intraoculaires ou de la surface LASIK • Améliorer l'acuité visuelle et la qualité de la vision postopératoire • Prévenir ou réduire au minimum la sécheresse oculaire postopératoire[40]

Le besoin croissant de soins concertés pour la sécheresse oculaire

Plusieurs tendances démographiques et sociales ont accru l'urgence et le fardeau de la prise en charge de la sécheresse oculaire. Premièrement, la sécheresse oculaire et les interventions chirurgicales aux yeux sont de plus en plus courantes à mesure que la population vieillit. Deuxièmement, la dépendance accrue à l'égard des dispositifs électroniques et leur utilisation croissante peuvent causer ou exacerber la sécheresse oculaire en réduisant la fréquence de clignement des yeux chez l'utilisateur.[41, 42] Les personnes qui utilisent ces dispositifs doivent avoir un haut niveau de fonctionnement visuel et peuvent souhaiter avoir recours à une intervention chirurgicale précisément pour améliorer la qualité de leur vision. Troisièmement, la technologie d'ophtalmologie elle-même a évolué avec l'introduction des techniques de mesure qui offrent une précision sans précédent en biométrie oculaire, mais qui dépendent d'un film sain pour une utilisation optimale. De ma même façon, les lentilles intraoculaires multifocales et toriques offrent la possibilité de corriger la vision de près et de loin, mais elles semblent plus sensibles aux aberrations visuelles et aux erreurs de biométrie que la génération précédente de lentilles intraoculaires.[43] Cette différence est inhérente à la technologie des lentilles intraoculaires, mais l'insatisfaction résulte aussi en partie d'attentes plus élevées, notamment du désir d'un fonctionnement visuel optimal et d'une dépendance minimale des lunettes pour la vision éloignée ou rapprochée.

Pour toutes ces raisons, le besoin croissant de soins diligents pour la sécheresse oculaire avant et après l'intervention chirurgicale exige de plus en plus d'heures des fournisseurs de soins. Des approches efficaces, dont la prise en charge concertée de la sécheresse oculaire par les optométristes et les ophtalmologistes, seront nécessaires pour répondre à ces demandes.

OPÉRATION DE LA CATARACTE

Si nous extrapolons les données de 2014 provenant d'une seule province, environ 450 000 interventions de phacoémulsification peuvent être exécutées chaque année à l'échelle du Canada, ce qui fait des cataractes la raison la plus fréquente d'avoir recours à une intervention de chirurgie oculaire.[44,45] La demande pour ce genre d'intervention devrait plus que doubler d'ici 2036.[46]

La norme actuelle de soins pour l'extraction de cataracte est la phacoémulsification suivie d'une implantation de lentille intraoculaire, qui peut être soit monofocale ou « Premium ». Ce dernier type de lentille intraoculaire comprend une variété de modèles qui permettent la correction de la vision éloignée, rapprochée et intermédiaire, ce qui permet généralement une moins grande dépendance à l'égard des lunettes ou des lentilles cornéennes. Selon le modèle, les lentilles intraoculaires Premium pourraient être sujettes à l'éblouissement et aux halos, il est donc important de réduire au minimum les autres perturbations visuelles chez ces patients, notamment les aberrations liées à la sécheresse oculaire.[47,48] Fait intéressant, les cataractes elles-mêmes peuvent provoquer des aberrations d'ordre supérieur,[49,50] et cet effet sur la qualité de la vision est compliqué par une sécheresse oculaire mal maîtrisée.[29, 30]

Une sécheresse oculaire préexistante est courante chez les personnes qui ont des cataractes, puisque certains facteurs de risque (nommément le vieillissement, le diabète[3], le fait d'être une femme[51]) prédisposent les patients à ces deux affections.[52] Toutefois, la sécheresse oculaire passe souvent inaperçue et n'est donc pas traitée chez les patients qui subissent une opération de la cataracte. Trattler et coll. ont déclaré que, sur 136 patients américains qui subissaient une opération de la cataracte, si seulement 22 % des patients avaient un diagnostic préalable de

sécheresse oculaire, une plus grande proportion de patients montraient des signes objectifs de maladie de la surface (temps de rupture du film lacrymal [TBUT] ≤5 secondes, 63 %; coloration cornéenne, 77 %), ce qui suggérait un sous-diagnostic généralisé.[25] Autre fait intéressant, les symptômes subjectifs de la sécheresse oculaire étaient moins courants, puisque seulement 31 % des répondants ont déclaré une sensation de brûlure, et 41 % ont déclaré sentir la présence d'un corps étranger. Ces constatations sont conformes aux rapports antérieurs qui suggèrent que l'autodéclaration des patients n'est pas un outil de dépistage fiable pour la sécheresse oculaire.[41,53,54]

La sécheresse oculaire non maîtrisée limite l'exactitude de la biométrie préopératoire et entraîne des erreurs sur le plan de la puissance ou du positionnement des lentilles intraoculaires.[41,54] Cet effet a été clairement démontré au moyen de données prélevées lors de lectures répétées chez les patients qui se présentent pour une opération de la cataracte. Epitropoulos et coll. ont rapporté que la différence moyenne entre deux lectures kératométriques successives était de 0,28 D chez les personnes qui ont des larmes hyperosmolaires (n=100 yeux) par rapport à 0,13 D dans le groupe de contrôle (n=50 yeux). Les différences de puissance calculées des lentilles intraoculaires atteignaient jusqu'à 5,5 D dans le groupe de 100 yeux ayant des larmes hyperosmolaires, et la fréquence d'une différence de puissance ≥0,5 D dans les lentilles intraoculaires était considérablement plus élevée pour les larmes hyperosmolaires que pour les larmes normales (p=0,02). Par ailleurs, 17 % des yeux qui ont des larmes hyperosmolaires, mais seulement 2 % des yeux qui ont des larmes normales affichaient une différence ≥1,0 D (p=0,01) du vecteur d'astigmatisme entre les lectures.[39]

Apparition des symptômes de la sécheresse oculaire après une opération de la cataracte

L'opération de la cataracte perturbe la surface oculaire et provoque l'inflammation intraoculaire et de la surface oculaire. Par ailleurs, l'intervention chirurgicale endommage les neurones sensoriels et les autres neurones, et l'énervation qui en résulte réduit la sensibilité tactile et autres sensibilités dans la cornée.[40] Le retour des symptômes de sécheresse oculaire est courant après la phacoémulsification,[10,27] mais ces symptômes sont habituellement temporaires. L'hypoesthésie cornéenne, l'instabilité du film lacrymal et d'autres indicateurs de la sécheresse oculaire se résorbent souvent dans une période de trois mois, laquelle coïncide probablement avec le début de la régénération axonale.[18,19] La sensibilité cornéenne revient graduellement à des niveaux quasi préopératoires sur une période d'un an.[40] Toutefois, chez un petit sous-ensemble de patients, les symptômes de la sécheresse oculaire persistent indéfiniment.[20,55] À titre d'exemple, les personnes atteintes de diabète présentent un risque accru de sécheresse oculaire postopératoire grave et chronique.[56]

Les traitements topiques doivent être appliqués de manière uniforme après l'intervention chirurgicale pour limiter l'étendue ou la durée du retour de la sécheresse oculaire.[52] Lorsqu'ils sont utilisés de concert avec les stéroïdes topiques, on a rapporté que les lubrifiants amélioraient les symptômes de sécheresse oculaire et le fonctionnement visuel, par rapport aux soins topiques postopératoires habituels administrés seuls.[57,58] Jee et coll. ont comparé directement les effets des gouttes ophtalmiques lubrifiantes et contenant des stéroïdes, sans agents de conservation et avec agents de conservation, après l'opération de la cataracte, chez 80 patients (80 yeux) atteints de sécheresse oculaire avant l'opération. Dans le cadre de cette étude prospective en mode libre, les patients ont reçu les produits sans agents de conservation ou les produits avec agents de conservation quatre fois par jour pendant un mois, et deux fois par jour par la suite. Après le premier mois suivant l'intervention chirurgicale, les sujets qui avaient reçu le traitement topique sans agents de conservation ont signalé des symptômes moins graves en comparaison des symptômes de ceux qui avaient reçu le traitement avec agents de conservation (p<0,05). Après le deuxième mois, les mesures objectives de la sécheresse oculaire (coloration, stabilité du film lacrymal, marqueurs inflammatoires et cellules à gobelet de la conjonctive) s'étaient considérablement améliorées grâce au traitement sans agents de conservation.[59]

La lubrification à elle seule pourrait être insuffisante pour soulager l'inflammation qui entraîne la sécheresse oculaire chronique.[60-62] Cela est ressorti le plus clairement dans une étude multicentrique randomisée auprès de 233 adultes chinois atteints de sécheresse oculaire modérée à grave au départ. Les patients ont été affectés par randomisation à l'un des deux groupes de l'étude et devaient faire l'application deux fois par jour des produits suivants : les patients de l'un des deux groupes ont appliqué la cyclosporine 0,05 % et les patients de l'autre groupe ont utilisé l'émulsion qui servait d'excipient; aucun autre traitement n'était autorisé à l'exception des larmes artificielles. Si les deux groupes ont constaté une importante amélioration des symptômes par rapport aux symptômes initiaux, une amélioration considérablement plus importante a été observée sur le plan de la coloration de la cornée après 4 et 8 semaines (p=0,025 et 0,05, respectivement) et dans le score de Schirmer après 4 semaines (p=0,035) dans le groupe de la cyclosporine par rapport au groupe de l'excipient.[60] Bien qu'aucune étude contrôlée par rapport à l'excipient de ce genre n'ait été rapportée dans un contexte chirurgical, une étude prospective contrôlée de manière controlatérale

a comparé la cyclosporine topique et la solution physiologique chez 32 patients subissant une phacoémulsification bilatérale. Chez ces patients, le traitement à la cyclosporine topique a considérablement amélioré la stabilité du film lacrymal et d'autres mesures de la sécheresse oculaire, par rapport à la solution physiologique utilisée seule. Si l'intensité de la sécheresse oculaire déclarée par les patients s'est améliorée après un premier mois de traitement, les avantages cliniques sont devenus statistiquement significatifs après deux mois de traitement à la cyclosporine.[63]

Exacerbation de la sécheresse oculaire après l'opération de la cataracte

Chez les patients atteints de sécheresse oculaire chronique préexistante, un traitement anti-inflammatoire peut améliorer l'acuité postopératoire et le fonctionnement visuel.[47, 64] Dans le cadre d'une petite étude prospective contrôlée de manière controlatérale, randomisée et en double aveugle, Donnenfeld et coll. ont comparé les gouttes ophtalmiques lubrifiantes (0,4 % de polyéthylèneglycol 400; 0,3 % de propylèneglycol) à la cyclosporine topique chez les patients qui se soumettent à une phacoémulsification bilatérale avec implantation de lentilles intraoculaires multifocales. Parmi les 14 participants à l'étude, seulement trois avaient un diagnostic de sécheresse oculaire. Toutefois, étant donné que le TBUT moyen au départ était faible (environ 6 secondes), d'autres participants avaient peut-être une sécheresse oculaire non diagnostiquée ou marginale. Le traitement a commencé un mois avant l'opération et a été maintenu après l'intervention. Deux mois après l'opération (c.àd. après trois mois de traitement topique), l'acuité visuelle non corrigée était considérablement meilleure chez les participants dont les yeux avaient été traités à la cyclosporine que chez les participants ayant reçu le traitement aux lubrifiants (p=0.005). La sensibilité aux contrastes a aussi été améliorée par le traitement à la cyclosporine, et il y avait une tendance numérique vers une plus grande stabilité lacrymale après deux mois. Par ailleurs, la coloration cornéenne, qui s'est aggravée par rapport à la coloration initiale dans les yeux traités au lubrifiant, s'est améliorée considérablement dans le groupe des yeux traités à la cyclosporine (p=0,034 pour la différence entre les groupes au deuxième mois).[47]

D'autres études ont exploré l'utilisation postopératoire de la cyclosporine topique après l'opération de la cataracte.[63-65] Il a été suggéré que certains effets de traitement sont observables dans les semaines suivant la phacoémulsification[65] et d'autres interventions chirurgicales,[66] mais ces allégations sont difficiles à évaluer, étant donné que les avantages établis de la cyclosporine se produisent avec une utilisation de plus longue durée (≥3 mois).[8,54,61]

CHIRURGIE RÉFRACTIVE

L'intervention LASIK et d'autres interventions photoréfractives connexes sont largement utilisées pour améliorer l'acuité visuelle non corrigée. Ces interventions produisent généralement des résultats favorables et une forte satisfaction chez les patients.[67,68] Toutefois, la sécheresse oculaire postopératoire est une complication possible de ces interventions qui peut causer de l'inconfort, une baisse de la vue et une insatisfaction générale. Comme dans le cas de l'opération de la cataracte (plus haut), la sécheresse oculaire après l'intervention LASIK est associée aux effets du trauma chirurgical sur la surface oculaire, notamment la perte de sensibilité dans la cornée[40] et l'inhibition du clignement des yeux et du larmoiement réflexe et de base.[69] La sécheresse oculaire préexistante est probablement courante et sous-diagnostiquée, étant donné que l'intolérance aux lentilles cornéennes, qui a été associée à la sécheresse oculaire, est un facteur courant qui incite les patients à demander l'intervention LASIK.[67]

Le facteur de la sécheresse oculaire dans la sélection des patients

Puisque les interventions chirurgicales réfractives sont des opérations chirurgicales non urgentes, seules les personnes qui ont ou qui peuvent avoir une santé adéquate de la surface oculaire sont considérés comme de bons candidats.[2,32,70,71] La sécheresse oculaire grave causée par le syndrome de Sjögren ou d'autres problèmes immunitaires est habituellement considérée comme une contre-indication pour le traitement de chirurgie réfractive. Toutefois, certains rapports suggèrent que même ces personnes pourraient subir l'intervention LASIK si l'affection est prise en charge avec succès avant l'intervention.[68,72] Pour ce faire, la sécheresse oculaire préexistante doit être prise en charge de manière progressive (fig. 1), à l'aide des traitements décrits dans le guide pratique de 2014.[8] Fait à remarquer, une étude rétrospective a révélé que le traitement à la cyclosporine pendant une moyenne de 3,2 mois (plage de 1 à 12 mois) permettait aux personnes atteintes de sécheresse oculaire légère d'avoir recours à la chirurgie réfractive.[73]

Les autres interventions chirurgicales photoréfractives, y compris les interventions sans découpe de volet cornéen comme la technique SMILE (small-incision lenticule extraction), semblent causer moins de lésions aux nerfs et une sécheresse oculaire moins grave que la technique LASIK.[74,75] Il n'a pas encore été déterminé si ces nouvelles interventions sont préférables pour les patients à risque élevé de sécheresse oculaire.[69,72]

Sécheresse oculaire transitoire et sécheresse oculaire chronique après l'intervention LASIK
Après l'intervention LASIK, l'instabilité du film lacrymal et d'autres signes et symptômes de sécheresse oculaire se résorbent habituellement de façon spontanée, mais une sécheresse oculaire postopératoire chronique, qui persiste pendant 6 à 12 mois après l'intervention LASIK, a été signalée chez 0,8 % à 20 % des patients.[70,76] Le risque de sécheresse oculaire chronique après l'intervention LASIK augmente avec l'âge et est plus élevé chez les femmes que chez les hommes.[71,73] On a également suggéré que les interventions LASIK pour l'hypermétropie comportent un plus grand risque de sécheresse oculaire chronique que les interventions LASIK pour la myopie.[73,77,78] Après l'intervention LASIK pour l'hypermétropie, les patients atteints de sécheresse oculaire préexistante et ceux qui ont développé une sécheresse oculaire chronique semblent présenter un risque plus élevé de développer une régression réfractive.[70,77]

Par ailleurs, la sécheresse oculaire légère préexistante ou infraclinique semble être un facteur de risque pour la sécheresse oculaire chronique après l'invention LASIK.[32] Une étude prospective a suivi 139 yeux soumis à l'intervention LASIK. Les sujets étaient exclus de l'étude s'ils avaient un diagnostic définitif de sécheresse oculaire, mais ils pouvaient être inclus dans l'étude s'ils n'avaient que des symptômes isolés comme une coloration légère ou modérée de la cornée ou un TBUT inférieur à 5 secondes. Dans cette population, une coloration cornéenne préopératoire et une faible production de larmes étaient associées avec une sécheresse oculaire persistante un an après l'intervention LASIK.[76] De la même façon, les patients dont la stabilité du film lacrymal était réduite au départ étaient considérablement plus à risque de développer des anomalies de surface au moins au cours des six derniers mois après l'intervention LASIK.[79]

L'utilisation de la cyclosporine topique suivant l'intervention LASIK a été étudiée comme supplément possible aux soins normalisés après l'intervention pour les patients qui n'ont pas de diagnostic préalable de sécheresse oculaire. Peyman et coll. ont signalé que, dans une série de 22 sujets traités bilatéralement, les yeux qui ont reçu de la cyclosporine tous les jours étaient considérablement plus susceptibles de recouvrer la sensibilité tactile dans une période de trois mois suivant l'intervention LASIK par rapport aux yeux du groupe de contrôle ($p \leq 0{,}011$).[80] Le délai de cette réaction est conforme aux éléments probants qui indiquent que les avantages cliniques de la cyclosporine deviennent plus significatifs après un traitement de 3 mois ou plus.[8,61,66]

L'acuité visuelle après l'intervention LASIK est moins satisfaisante chez les patients qui développent une sécheresse chronique que chez les patients qui n'ont pas cet effet indésirable.[70,77,78] Lors d'une vaste (n=565) analyse rétrospective, Albietz et coll. ont signalé que la régression réfractive était associée à la sécheresse oculaire chronique ($p=0{,}008$ pour la sécheresse oculaire au 12e mois par rapport à aucune sécheresse oculaire) et à la coloration préexistante de la surface oculaire, mais à un TBUT réduit et à une sensibilité tactile réduite de la cornée.[81] Les auteurs ont fait remarquer que la sécheresse oculaire chez leurs patients décroissait grâce à une prise en charge persistante de la surface oculaire, notamment l'installation d'une occlusion lacrymale (bouchons méatiques), à l'hygiène des paupières, et à l'utilisation constante de gouttes ophtalmiques lubrifiantes. Dans les yeux où l'on pouvait constater une sécheresse oculaire après l'intervention LASIK, l'acuité visuelle non corrigée et les signes et symptômes de sécheresse oculaire se sont améliorés parallèlement[81], comme il a été déclaré en dehors du contexte clinique.[82]

Dans l'ensemble, ces constatations semblent indiquer que les personnes à risque de sécheresse oculaire après l'intervention LASIK bénéficient de traitements topiques qui rétablissent le film lacrymal et ciblent l'inflammation.

Exacerbation de la sécheresse oculaire après l'intervention LASIK
Salib et coll. ont mis à l'essai le traitement de la sécheresse oculaire avant l'intervention LASIK au moyen de la cyclosporine par rapport à des larmes artificielles sans agents de conservation chez les patients qui ont des antécédents de sécheresse oculaire (n=21 [42 yeux]).[83] Les patients devant subir une intervention LASIK pour la myopie ont commencé un traitement aux gouttes randomisées deux fois par jour, un mois avant l'intervention. Les deux traitements ont permis d'améliorer la coloration de la surface de la cornée et les symptômes subjectifs de la sécheresse oculaire avant l'intervention. Le traitement topique a été maintenu pendant les trois mois suivant l'intervention. Les symptômes de la sécheresse oculaire se sont aggravés de façon passagère pendant la première semaine d'observation, alors que l'on constatait un rétablissement plus rapide dans le groupe des yeux traités à la cyclosporine par rapport au groupe des yeux traités aux larmes artificielles. De 3 à 12 mois après l'intervention LASIK, les symptômes de la sécheresse oculaire se sont améliorés par rapport à l'évaluation initiale dans les deux groupes de traitement. Les yeux traités à la cyclosporine affichaient une plus grande prévisibilité en réfraction au cours de la période d'observation d'un an et étaient plus susceptibles de se situer à 0,5 D de leur réfraction d'équivalent sphérique ciblée. Cette différence était statistiquement significative au troisième mois ($p=0{,}015$).[83]

Si l'on prévoit utiliser la cyclosporine chez les personnes retenues pour l'intervention LASIK, le traitement devrait commencer un mois ou plus avant l'intervention et se poursuivre pendant trois mois ou plus après l'intervention, en vue d'optimiser les bienfaits lorsque les symptômes de la sécheresse oculaire sont plus incommodants. Le traitement préalable peut permettre aux personnes qui ne seraient pas admissibles autrement à la chirurgie réfractive de subir une intervention LASIK réussie,[32] et devrait être envisagé pour les personnes qui manifestent une sécheresse oculaire légère ou asymptomatique.[66,80]

En plus du traitement anti-inflammatoire,[66,80,83] les options thérapeutiques avant et après l'intervention LASIK englobent une vaste gamme d'options exposées dans le guide pratique de 2014,[8] comme les gouttes ophtalmiques lubrifiantes, les lentilles sclérales, l'occlusion lacrymale, la supplémentation en acides gras essentiels,[84,85] le sérum autologue et la doxycycline par voie orale. Les données périopératoires sur la plupart de ces options sont très limitées. Dans une petite étude portant sur 12 personnes qui présentaient des erreurs réfractives résiduelles après l'intervention LASIK et dont la vision non corrigée s'est améliorée avec l'application de larmes artificielles lubrifiantes, les bouchons méatiques semblaient améliorer considérablement la performance visuelle ($p<0,0001$).[86] Par ailleurs, Di Pasquale et coll. ont suggéré que les patients qui présentent une instabilité persistante du film lacrymal après le traitement de l'inflammation de la surface oculaire peuvent bénéficier d'un traitement qui consiste à réchauffer l'œil[87] pour stimuler la sécrétion des glandes de Meibomius et rétablir les lipides des larmes.[8,88] Dans le cadre d'une étude observationnelle auprès de patients dont la sécheresse oculaire récidivante a persisté pendant un an après l'intervention LASIK, la combinaison de bouchons méatiques, de corticostéroïdes topiques et, s'il y a lieu, de compresses chaudes, a donné lieu à une amélioration subjective des symptômes de la sécheresse oculaire, de même qu'à des augmentations importantes de l'épaisseur de la couche lipidique du film lacrymal et du TBUT.[87]

PRISE EN CHARGE CONCERTÉE DE LA SÉCHERESSE OCULAIRE

Le tableau 3 contient la liste d'une série d'objectifs pour le traitement préopératoire et postopératoire des patients admissibles à l'intervention de chirurgie oculaire. La liste est générale, en ce sens qu'elle pourrait s'appliquer également à l'opération de cataracte et à la chirurgie réfractive, et potentiellement à d'autres interventions dont les résultats peuvent être compromis par une sécheresse oculaire non maîtrisée. En bref, tous les patients qui sont aiguillés vers une consultation en chirurgie devraient être évalués soigneusement pour déceler les signes et symptômes de maladies de la surface oculaire. Les patients atteints de sécheresse oculaire épisodique ou chronique devraient être traités avant l'intervention chirurgicale dans le but de traiter les signes et les symptômes de la sécheresse oculaire et de stabiliser le film lacrymal et la surface oculaire, pour faire en sorte que les données kératométriques et topographiques soient fiables et précises. Un counseling approprié pendant cette période de traitement est aussi essentiel pour améliorer la compréhension du patient de l'affection et pour gérer les attentes. Après l'intervention, une maladie de la surface oculaire peut se développer ou s'aggraver, au moins temporairement, malgré le recours continu aux traitements de la sécheresse oculaire commencés avant l'intervention chirurgicale. L'objectif des soins pendant cette période devrait être de gérer l'inconfort oculaire et de rétablir une surface oculaire stable et saine.

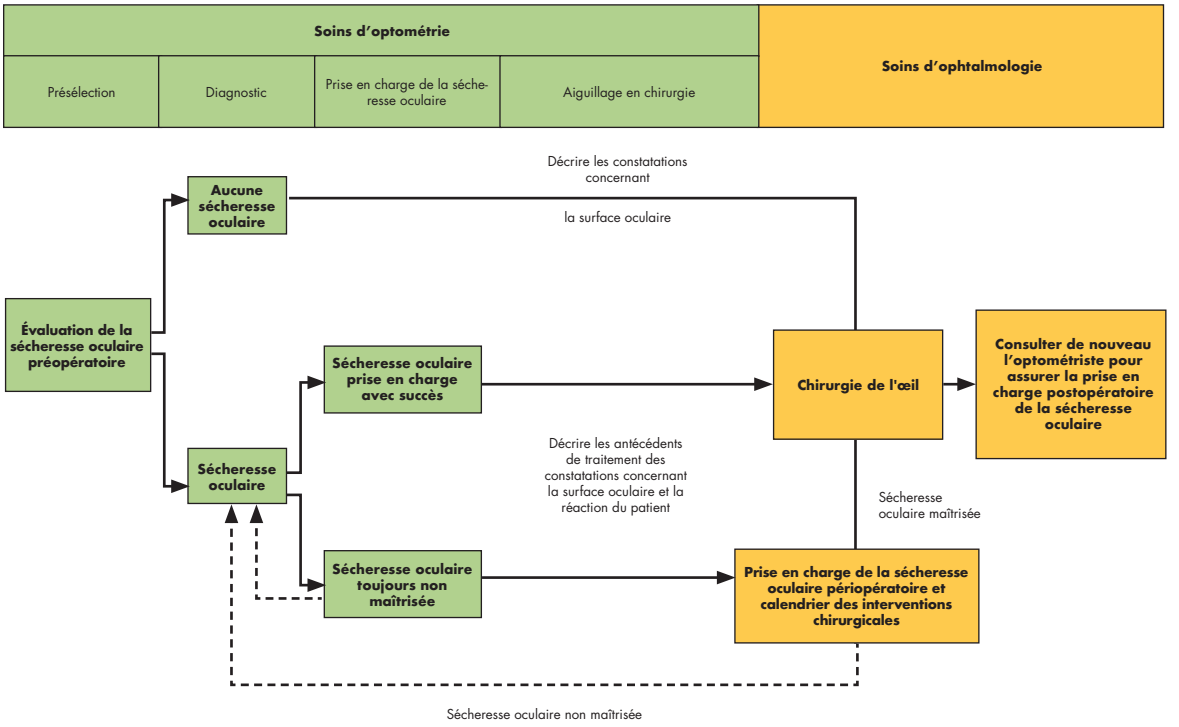
Tableau 3. *Objectifs pour la prise en charge préopératoire et postopératoire de la sécheresse oculaire*

Dans le contexte préopératoire, atteindre :	Dans le contexte postopératoire :
1. Des signes minimaux de sécheresse oculaire à l'examen	1. Maintenir un traitement périopératoire si la surface oculaire est stable
2. Un inconfort minimal et maîtrisé	2. Autrement, intensifier le traitement jusqu'à ce que la surface oculaire soit stable et qu'elle revienne à un niveau initial adéquat
3. Un fil lacrymal stable et optimisé	
4. Des mesures kératométriques stables (biométrie et topographie)	
5. Une réfraction manifeste stable	

Si le lieu des soins avant et après l'intervention chirurgicale peut varier quelque peu en fonction des circonstances, en règle générale, les optométristes sont bien placés pour dispenser une grande partie des soins pour la sécheresse oculaire dont ont besoin les patients avant et après l'intervention. La sécheresse oculaire est souvent détectée pour la première fois lorsque les patients reçoivent des soins optométriques. Que le patient soit admissible ou non à l'intervention chirurgicale oculaire, l'optométriste devrait évaluer la surface oculaire et commencer le traitement approprié sans délai. Dans tous les cas, il faut discuter du rôle du film lacrymal et de la surface oculaire avec les candidats à l'intervention chirurgicale pour renforcer la nécessité de bien respecter le traitement et pour éviter la déception après l'opération. Si l'on envisage les lentilles intraoculaires Premium, il faut aviser le patient que ces dispositifs sont particulièrement sensibles aux perturbations de la surface oculaire.

La figure 2 présente un diagramme pour la prise en charge conjointe des patients qui ont besoin d'une intervention chirurgicale oculaire ou qui en font la demande, et qui ont reçu un diagnostic de sécheresse oculaire symptomatique ou asymptomatique. En pareil cas, l'optométriste devrait décrire dans une demande de consultation à l'intention du chirurgien, toutes les constatations concernant la surface oculaire, y compris les rapports subjectifs et les données objectives qui ont mené au diagnostic. Idéalement, l'optométriste prendra en charge l'affection à ce stade précoce, à la fois pour améliorer le confort immédiat du patient et sa fonction visuelle et pour simplifier la prise en charge de l'intervention chirurgicale. La correspondance de l'optométriste devrait décrire les tests effectués et les traitements administrés à ce jour concernant la sécheresse oculaire, de même que les résultats de ces traitements. S'il y a lieu, l'optométriste peut aussi recommander que l'intervention soit retardée pour permettre l'optimisation de la surface oculaire.

Figure 2. *Diagramme proposé pour la prise en charge conjointe de la sécheresse oculaire périopératoire*



L'ophtalmologiste planifiera l'intervention chirurgicale si la surface oculaire est stable et saine ou il renverra le patient consulter l'optométriste en vue qu'il reçoive un traitement continu ou amélioré contre la sécheresse oculaire. Dans certains cas, l'optométriste peut demander que l'ophtalmologiste se charge des soins au patient, notamment en ce qui concerne la surface oculaire ou la cornée. Après l'intervention, il est généralement approprié que les soins continus pour la sécheresse oculaire soient repris en charge par l'optométriste, à moins que la maladie soit récalcitrante ou que des complications nécessitant une intervention secondaire ou tertiaire ne se manifestent. Dans son

rapport, le chirurgien devrait décrire la nature et les résultats de l'intervention, de même que tous les changements qui peuvent avoir été faits dans le cadre de la prise en charge postopératoire de la sécheresse oculaire, notamment les changements aux traitements topiques sur ordonnance et en vente libre.

CONCLUSIONS

La sécheresse oculaire est un trouble inflammatoire chronique que les optométristes et les ophtalmologistes côtoient régulièrement. Elle doit être évaluée et traitée adéquatement chez tous les patients. Les principes du traitement de la sécheresse oculaire sont semblables, que l'on envisage ou non l'intervention chirurgicale oculaire, et devraient suivre les recommandations du document *Dépistage, diagnostic et prise en charge de la sécheresse oculaire : guide pratique à l'intention des optométristes canadiens* de 2014.[8] Toutefois, la prise en charge de la sécheresse oculaire est particulièrement importante avant et après certaines interventions chirurgicales, précisément les opérations de chirurgie réfractive et de la cataracte, parce que la sécheresse oculaire non maîtrisée peut placer le patient à risque d'obtenir des résultats chirurgicaux moins qu'optimaux et, à l'inverse, l'intervention chirurgicale provoque ou exacerbe fréquemment la sécheresse oculaire. Par conséquent, il faut retarder l'intervention chirurgicale jusqu'à ce que la surface oculaire se stabilise et qu'un film lacrymal adéquat et approprié soit rétabli, dans toute la mesure du possible. Comme l'illustre la figure 1, le traitement anti-inflammatoire pour les patients atteints de sécheresse oculaire chronique préexistante devrait commencer avant l'intervention chirurgicale et se poursuivre quelques mois après l'intervention. Les recommandations consensuelles pour la prise en charge de la sécheresse oculaire périopératoire figurent au tableau 4.

Tableau 4. *Recommandations consensuelles pour la prise en charge de la sécheresse oculaire périopératoire*

Recommandation nº 1 : Dans le cas des patients chez qui l'on soupçonne la présence d'une douleur neuropathique oculaire, il est important de cerner les affections connexes, comme la douleur neuropathique non oculaire, la dépression, l'anxiété et les troubles du sommeil.
Recommandation nº 2 : Selon la fréquence d'utilisation, les formulations sans agents de conservations de produits topiques médicamenteux et non médicamenteux devraient être envisagées pour un traitement avant et après une intervention chirurgicale oculaire.
Recommandation nº 3 : Indépendamment de l'inconfort oculaire autodéclaré, les patients qui subissent une opération de la cataracte devraient être évalués pour la détection de signes et symptômes de la sécheresse oculaire.
Recommandation nº 4 : La surface oculaire devrait être optimisée avant l'opération de la cataracte, afin d'augmenter l'exactitude et la précision de la biométrie préopératoire et d'améliorer le confort postopératoire et le fonctionnement visuel.
Recommandation nº 5 : Les patients affectés d'une sécheresse oculaire symptomatique ou asymptomatique préexistante devraient faire l'objet d'un traitement aux agents anti-inflammatoires avant l'intervention chirurgicale pour prévenir l'exacerbation des symptômes.
Recommandation nº 6 : Il faut effectuer une évaluation vouée à la sécheresse oculaire dans le cadre du bilan de santé de tous les patients retenus pour l'intervention chirurgicale réfractive.
Recommandation nº 7 : Les signes et les symptômes de la sécheresse oculaire chronique, y compris de la sécheresse oculaire légère, devraient être évalués et pris en charge chez tous les candidats à l'intervention chirurgicale oculaire.
Recommandation nº 8 : Les patients qui présentent une coloration de la surface oculaire, une instabilité du film lacrymal ou d'autres signes de sécheresse oculaire devraient être informés des risques d'exacerbation de la sécheresse oculaire après l'intervention chirurgicale.
Recommandation nº 9 : Indépendamment des antécédents de sécheresse oculaire, les patients qui subissent une intervention chirurgicale oculaire devraient être informés que des symptômes de sécheresse oculaire peuvent se manifester après l'intervention.

Pour que les soins périopératoires pour les patients atteints de sécheresse oculaire soient efficients, les optométristes et les ophtalmologistes doivent prendre en charge l'affection conjointement, communiquer entre eux de façon efficace et transmettre des messages uniformes au patient concernant les constatations relatives à la surface oculaire, les risques et la réponse au traitement. Le tableau 1 supplémentaire contient des listes de vérification décrivant les pratiques exemplaires en matière d'échanges entre les chirurgiens et les optométristes.

Tableau 1 supplémentaire : *Renseignements relatifs à la sécheresse oculaire à inclure dans les aiguillages entre les optométristes et les ophtalmologistes*

	Optometrist to ophthalmologist	Ophthalmologist back to optometrist
Symptômes	• Sensation • Vision } *Préciser le calendrier*	• Sensation • Vision • Description d'intervention chirurgicale récente • Résultats (inclure la réfraction postopératoire si elle a été effectuée)
Signes/évaluation	Constatations • Score symptomatique (questionnaire) • Stabilité lacrymale (NIBUT/TBUT) • Coloration cornéenne (NaFl) et conjonctivale (LG) • Fonctionnement des glandes de Meibomius • Autres tests (p. ex., osmolarité du film lacrymal)	
	Description de la sécheresse oculaire/surface oculaire • Gravité • Actuellement stable? *(si oui, depuis combien de temps a-t-on obtenu la stabilité?)*	
Prise en charge	• Traitements actuels et antérieurs • Problèmes de tolérance, s'il y a lieu • Antécédents d'adhésion	• Modifications postopératoires apportées à la prise en charge de la surface oculaire, y compris les produits sans ordonnance? *(Si la réponse est oui, veuillez préciser les produits et le dosage)*
Recommandations	• Le patient devrait-il subir une intervention chirurgicale le plus tôt possible? • Si la réponse est non, quelle étape de traitement doit être atteinte avant de planifier l'intervention chirurgicale?	• Instructions pour les soins postopératoires • Prochaine visite prévue à la clinique ou retour aux soins de l'optométriste ou prise en charge conjointe • Objectifs du patient concernant sa vision

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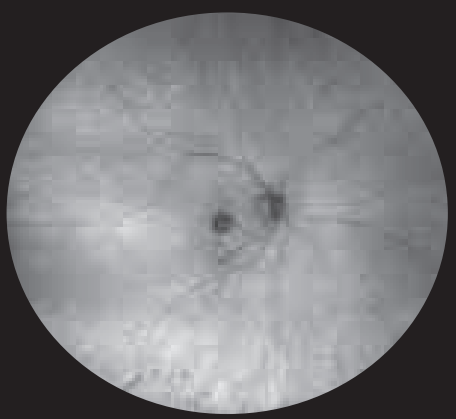

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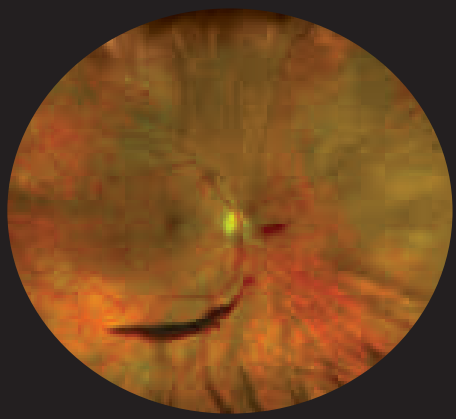

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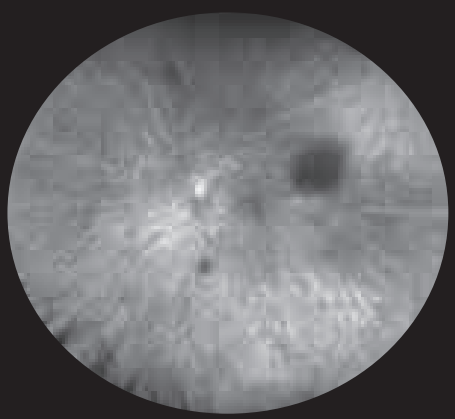

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Evaluating Various Building Types for Optometry Tenants

Dale Willerton **Jeff Grandfield**

The Lease Coach are Commercial Lease Consultants who work exclusively for tenants. Dale and Jeff are professional speakers and co-authors of Negotiating Commercial Leases & Renewals FOR DUMMIES (Wiley, 2013). Got a leasing question? Need help with your new lease or renewal? Call 1-800-738-9202, e-mail DaleWillerton@TheLeaseCoach.com or visit www.TheLeaseCoach.com.

Whether you are opening your first optometry clinic or considering moving your operations to another location, one of the first things to remember is that not all buildings and/or properties are created equally. Although the fundamentals of negotiating commercial leases are the same regardless of where you have your business, each type of property has unique aspects that should be considered. You should have the mindset that there is no such thing as a "perfect property" for you; the challenge is to lease a commercial space with the most advantageous criteria that you can find.

Our book *Negotiating Commercial Leases & Renewals FOR DUMMIES* describes several different types of locations, as summarized below:

OFFICE BUILDINGS

Office tenants can choose from simple, one-storey buildings to downtown high-rise properties. The variety of office buildings and their locations is quite extreme. Some office buildings are so large they're almost communities unto themselves, with their own food and service tenants. These office buildings may be linked to other properties via pedestrian walkways. Typically, downtown office properties are more expensive to lease, and the operating costs are typically higher as well.

A major factor to consider when determining where to lease office space is parking – both its availability and cost. Your patients may not be willing or able to interrupt their appointment with you to run outside and add more money to a parking meter. Also, don't overlook public transportation for your patients who don't drive but still need to visit you. Another consideration is elevator access — if you lease office space on a higher floor and plan to remain open for business on evenings and/or weekends, you should ensure that the building's elevator isn't shut down during that period.

For a smaller office building in an outlying area, you may be allowed to have a sign on the building or property to identify your clinic, but this isn't common for buildings downtown. The building and property amenities can vary greatly. With larger, high-rise properties, tenants will often share common washrooms and nightly janitorial services. The presence of a café tenant inside the building is an amenity that can provide greater comfort (and even productivity) to all business owners and their staffs. If someone accompanies a patient to an appointment at your clinic, they would likely be more comfortable in a café enjoying a cup of coffee than sitting in a typical waiting room.

RETAIL STRIP PLAZAS

A retail strip plaza may or may not have anchor tenants. Anchor tenants are large, well-known, heavily-trafficked businesses. In contrast, unanchored strip plazas contain only small "Mom-and-Pop"-type stores. Sometimes, several strip plazas are clustered together. Neighbourhood plazas typically have a well-rounded mix of tenants, but it's not uncommon to see just four or five tenants in a small strip plaza. Larger plazas are typically owned by larger landlords – with or without local management. Typical units in strip plazas are around 1,200 square feet, depending on the depth of the property. Most tenants in a strip plaza want at least 18 to 20 feet of *frontage* (or width). This allows for maximum exposure. If the property is 60 feet deep, then a unit with 20 feet of frontage would measure 1,200 square feet.

The type of strip mall or plaza you select for your clinic and its location will determine how far you can expect your patients to travel to your place of business. Consumers like to be able to do multiple tasks when running errands – they want to get their eyes checked, grab some groceries, pick up the dry cleaning, and so on, without having to move their car.

Not all units in a strip mall are the same. *End caps* (or end units) are the most valuable units for lease (and often the most expensive for a tenant to lease). Some advantages of end cap units are their proximity to the road and the fact that they have both front and side windows. End cap units often have more parking adjacent to the building (so your visiting patients will not have to endlessly circle the lot looking for a parking space). Furthermore, end cap units often have business signage on two sides of the building – this can be extremely desirable as it increases your visibility.

CONDOS

To clarify, a condo is not a type of building – it's a type of ownership. When landlords turn their property into condominiums, they are creating individual ownership opportunities. Condos can apply to any type of building, but not to just a portion of the building. A building is either all condos or none. This may not be self-evident if the landlord turns their property into condominiums, sells some units, and keeps others for themselves.

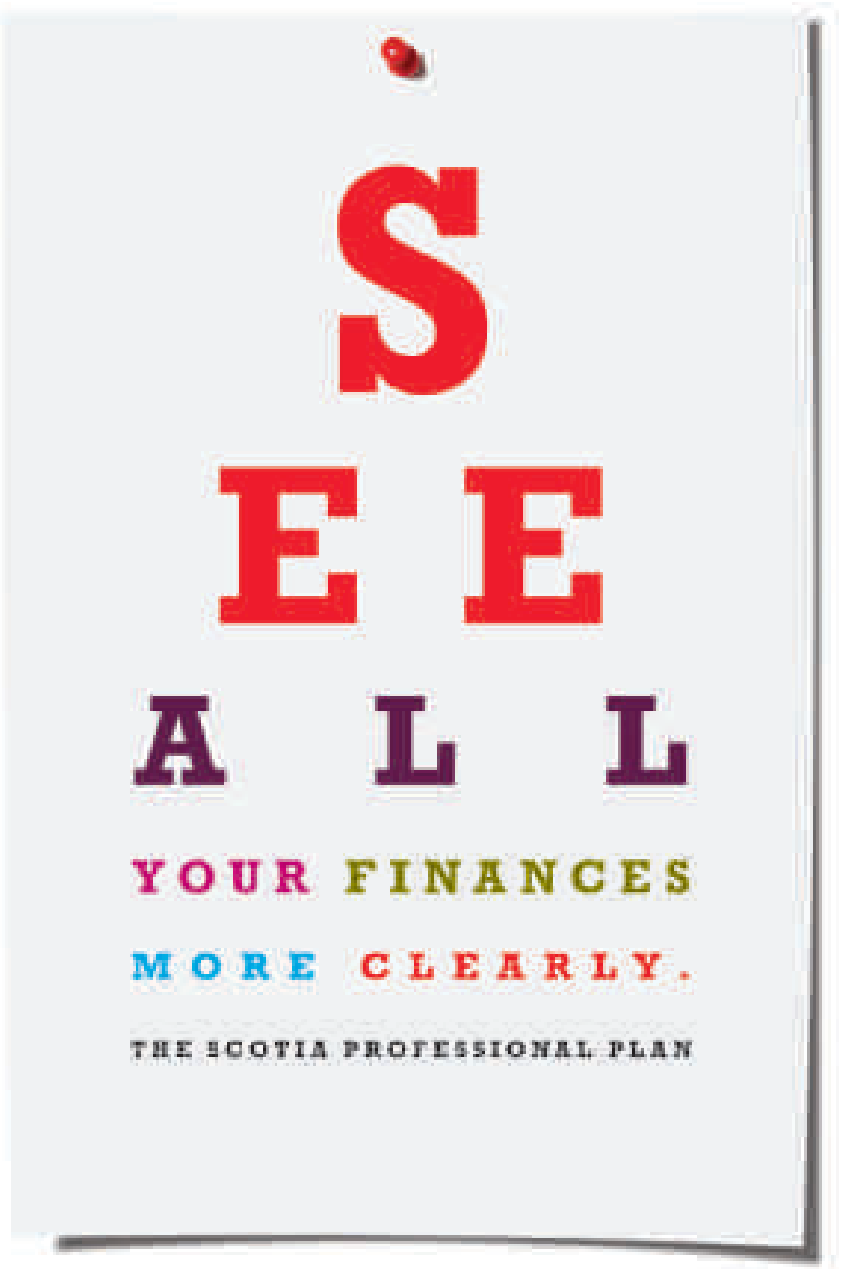
Owning your own condo unit can be very appealing for several reasons:

- You can create equity, which you can use for other business ventures and/or projects.
- The value of the condo or property can appreciate, which can make you wealthier.
- You have greater control over what happens to the building, and you run your own business.
- You can enjoy stability – you can better make long-range plans without worrying whether your landlord will raise your rent.

An important point to keep in mind if you are considering buying property for your clinic is to not buy a location that you wouldn't want to lease.

Optometry clinics can benefit by being in the right location. If your clinic fails due to a poor location, it won't matter whether you lease or own your building. ●

For a copy of our free CD, Leasing Do's & Don'ts for Commercial Tenants, please e-mail your request to JeffGrandfield@TheLeaseCoach.com.



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