

Ocular surface optimization before cataract surgery

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Access this article online

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DOI:

10.4103/sjopt.sjopt_190_21

Abstract:

The evolution of refractive cataract surgery has increased patient expectations for visual outcomes following cataract surgery. Precise biometry and keratometry are critical for accurate intraocular lens (IOL) selection and favorable surgical outcomes. In patients with the ocular surface disease and corneal pathologies, preoperative measurements can often be erroneous, leading to postoperative refractive surprises and dissatisfied patients. Conditions such as dry eye disease, epithelial basement membrane dystrophy, Salzmann's nodular dystrophy, and pterygia need to be addressed thoroughly before performing cataract surgery to optimize the ocular surface, obtain high-quality preoperative measurements, and ultimately determine the appropriate IOLs. In this review, the various ocular surface pathologies affecting cataract surgery outcomes and options for treatment are discussed and the importance of optimization of the ocular surface before cataract surgery is reviewed.

Keywords:

Cataract surgery, corneal pathologies, intraocular lens calculations, refractive outcomes

INTRODUCTION

Cataract surgery is one of the most commonly performed surgical procedures in ophthalmology; however, the variables that are considered during the preoperative planning process are numerous and require utmost precision. A favorable surgical outcome is dependent on accurate keratometry, axial length, lens thickness, and anterior chamber depth measurements as well as new generation intraocular lens (IOL) formulas to determine the correct implant choice. While a good surgeon can perform safe cataract surgery, a great surgeon strives to not only perform a safe surgery but also a surgery that achieves the best possible visual outcome for their patient. As such, eye surgeons today are no longer just cataract surgeons, but refractive surgeons. Thankfully, an understanding of ocular surface disease (OSD) combined with technological advances to address surface irregularities have greatly improved the accuracy of preoperative measurements and have allowed surgeons to perform high-quality refractive cataract surgery.

Corneal surface irregularities and pathologies can lead to inaccurate IOL power measurements,^[1,2] axis calculations for toric IOLs,^[3] and wavefront analyses.^[4] In this review, we will discuss the effects of ocular surface pathologies on preoperative lens calculations and consequently, cataract surgery outcomes.

DRY EYE DISEASE AND CATARACTS

Dry eye disease (DED), as defined by TFOS DEWS II, "is a multifactorial disease of the ocular surface characterized by a loss of homeostasis of the tear film, and accompanied by ocular symptoms, in which tear film instability and hyperosmolarity, ocular surface inflammation and damage, and neurosensory abnormalities play etiological roles."^[5] It is understood that the incidence of DED is underestimated due to the varying presentations of the disease and lack of standard diagnostic criteria. However, it has been suggested that up to 35% of all adults^[6] suffer from DED. Unsurprisingly, the incidence of dry eye before cataract surgery tends to be higher than the general population as both dry eye and the need for cataract surgery, increase with age.^[7]

A variety of studies have tried to understand the incidence of DED among patients having cataract surgery. Gupta *et al.*^[8] analyzed a cohort

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How to cite this article: Venkateswaran N, Luna RD, Gupta PK. Ocular surface optimization before cataract surgery. Saudi J Ophthalmol 2022;36:142-8.

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Submitted: 09-Aug-2021

Revised: 27-Dec-2021

Accepted: 10-Feb-2022

Published: 29-Aug-2022

of 120 patients at two tertiary care centers and used tear osmolarity testing, Matrix metalloproteinase-9 (MMP-9) levels, and the Symptom Assessment in Dry Eye questionnaire,^[9] to determine DED status. They found abnormal tear osmolarity in approximately 57% of patients, abnormal MMP-9 in 63% of patients, and 47% of patients had positive corneal staining, while 7% had epithelial basement membrane dystrophy (EBMD). Interestingly, 46% of patients denied having any dry eye symptoms, yet 85% of this cohort had an abnormal tear osmolarity or MMP-9 test result while 38% of the cohort were positive for both of these tests. Trattler *et al.*^[10] looked at a population of 136 patients at 9 clinical sites in the United States and Canada. Clinicians graded DED based on tear break-up time (TBUT) and corneal staining. They found that 63% of patients had a TBUT <5 sec, 77% of eyes had positive corneal staining, and 50% of eyes had positive central corneal staining. Taken together, these two studies suggest two things: First, that the incidence of dry eye is approximately 50%–70% based on the diagnostic criteria, and second, that DED is likely underestimated in the cataract surgery population because patients can be asymptomatic but still have evidence of clinically significant DED on clinical testing.

The consequence of DED is its effect on preoperative cataract surgery planning. Epitropoulos *et al.*^[3] looked at a cohort of 100 hyperosmolar (>316 mOsm/L) and 50 normal eyes (<308 mOsm/L) using the Tearlab Osmolarity system and found that the hyperosmolar group had a statistically significant higher variability in keratometry readings on biometry than the normal group. The hyperosmolar group additionally had a higher percentage with 1D or greater difference in measured astigmatism as well as a higher percentage of eyes with an IOL power difference of more than 0.5D. While a diopter of astigmatism or half a diopter of power can easily be corrected with glasses, this level of error may not be acceptable for patients who have high preoperative expectations^[11] or who are receiving a presbyopia-correction IOL. Interestingly, when the groups were sorted by self-reported dry eye symptoms rather than measured osmolarity, these differences disappeared. This corroborates the fact that symptoms of DED do not necessarily correspond with clinical measures of dry eye.

Evaluation of dry eye disease

The American Society of Cataract and Refractive Surgery developed an algorithm to evaluate OSD. The goal of this tool is to help surgeons diagnose and treat OSD before surgery.^[12] The algorithm starts with a preoperative visit where the patient has had, at minimum, a 2 week holiday from contact lenses. At the preoperative visit, the patient has the standard preoperative refractive surgery testing (keratometry, topography, biometry, etc.) followed by an OSD screen. The OSD screen is similar to the components presented in the Gupta *et al.* study which include a short symptom questionnaire, tear osmolarity testing and MMP-9 testing. A positive screen for OSD then leads to an extensive clinical exam of the lids and ocular surface followed by a determination if the patient has signs and symptoms associated with a neuropathic cornea. The surgeon determines

whether these symptoms are visually significant. Depending on the outcome, eyelid and or corneal surface surgery is performed or the patient is observed while on topical dry eye treatment. The algorithm was only recently published in 2019 so feedback on its utility is promising but limited.

Treatment for dry eye disease

After the diagnosis of DED is made, there are a variety of available treatment options that can be utilized in both the preoperative and postoperative periods to maintain a healthy ocular surface. One framework for understanding DED is based on the Lacrimal Functional Unit (LFU)^[13] which includes the ocular surface (cornea, conjunctiva, and meibomian glands), the main and accessory lacrimal glands, and the neural network that controls gland secretion. Treatment is targeted at restoring homeostasis to the LFU.

The least invasive, and most commonly used treatments, including over-the-counter artificial tears and lubricating ointments. These interventions target the cornea to restore barrier function, supplement poor lacrimal gland secretion, and compensate for tear film instability, tear hyperosmolarity, or excessive tear film evaporation by mimicking tear osmolarity. These treatments are generally well tolerated except when a sensitivity develops in response to preservatives such as benzalkonium chloride which can produce toxic epithelial damage and inflammation.^[14] As such, preservative-free versions of tears and ointments are often recommended if patients require frequent usage. Punctal occlusion is another nonprescription alternative for dry eye treatment where a semi-permanent silicone or collagen plug is placed in the upper or lower puncta. Punctal plugs treat dry eyes by increasing the time that tears remain on the ocular surface. Of note, while studies have demonstrated the benefits of punctal plugs,^[15] a recent Cochrane review of punctal occlusion has found that the results of this intervention are equivocal, possibly due to researcher bias as well as imprecise data measurement.^[16]

Prescription options for DED treatment include cyclosporine (0.05% -Restasis or 0.09% -Cequa) or lifitegrast (Xiidra). Cyclosporine is an immunomodulatory therapy that binds cyclophilin D and prevents the opening of the mitochondrial permeability transition (MPT) pore. The MPT pore is thought to be an early step in the apoptosis cascade and apoptosis of the ocular surface epithelium has been highly associated with dry eye.^[7] There is evidence that cyclosporine can be an effective treatment for OSD through the mechanism described previously or by increasing mucus secretion by increasing conjunctival goblet cells.^[17] However, cyclosporine can cause burning and stinging on instillation which may decrease patient compliance and the efficacy of the treatment.^[18] Lifitegrast blocks the binding of intercellular adhesion molecule-1 to lymphocyte function-associated antigen-1 on T cell surfaces which inhibit both T cell activation and proinflammatory cytokine release, both of which are associated with dry eye.^[19,20] This treatment has been shown

to decrease dry eye symptoms^[21] but can cause irritation and blurred vision with use.^[22]

While the interventions described thus far focus on targeting tear quantity, quality, and stability on the corneal surface, eyelid thermal pulsation procedures target the meibomian glands. Meibomian gland dysfunction arises from hyperkeratinization of ductal epithelium leading to duct obstruction as well as increased viscosity of the meibum. Thermal pulsation entails warming the eyelids approximately 40°–42° Celsius with the goal of facilitating the expression of the meibum from the glands. There are a variety of thermal pulsation options including LipiFlow (Johnson and Johnson), iLux (Alcon), and TearCare (Sight Sciences). Studies have shown improvement in dry eye and irritation symptoms, increases in TBUT,^[23] and improved meibomian gland secretion after these therapies.^[24]

Finally, intense pulsed light therapy (IPL) uses a nonlaser light source to produce wavelengths from 500 nm to 1200 nm that are absorbed by hemoglobin and travel to the skin surface. This leads to coagulation and thrombosis of surface blood vessels, decreased abnormal blood vessel growth in the meibomian glands, greater ease of oil secretion, and destruction of inflammatory mediators. Studies have shown decreased redness, vascularity, and increased meibum viscosity with IPL treatment.^[25] While this technology has been extensively used for dermatologic purposes, it is relatively new to the field of ophthalmology and further studies are needed to understand its efficacy.

Advanced treatment of dry eye disease

There are other, more advanced, treatments for dry eye disease including amniotic membrane, punctal cautery, serum tears, scleral lenses, and PROSE lenses. Amniotic membranes can be placed on the ocular surface for one to 2 weeks to protect the cornea and decrease ocular surface inflammation. Cryopreserved or freeze-dried versions of amniotic membranes are available and can be used according to a patient's ocular surface status and anatomy. Punctal cautery can be performed for permanent closure of two or all four puncta in patients with refractory DED in whom there is punctal scarring or abnormal anatomy causing punctal plugs to constantly dislodge. Autologous serum tears (AST) mimic the complex composition of natural tears including water, lipids, salts, proteins, and hydrocarbons and are created from the supernatant of the patient's blood. Prior work has shown improvement in dry eye symptoms after the use of these tears.^[26-29] Similarly, platelet-rich plasma and plasma rich in growth factors are alternative hemoderivative tear formulations that contain platelet derivatives along with higher concentrations of growth factors and anti-inflammatory cytokines that can facilitate ocular surface restoration and healing. Scleral lenses are another alternative to dry eye treatment. They are large-diameter gas permeable lenses that sit on the sclera and create a space, or vault, over the cornea. Users insert a sterile solution into the lens before wearing it and the solution is held in place between the cornea and the lens creating a "liquid

bandage."^[30] Finally, customized PROSE prosthetic devices can be used that are created on a patient-by-patient basis with a custom-designed vault to treat severe OSD.

Autoimmune disease and dry eye disease

DED in the context of underlying autoimmune disease presents unique challenges for cataract surgeons due to the prevalence of DED among this patient population. The most common autoimmune diseases with OSD manifestations include rheumatoid arthritis (RA), Sjogren's syndrome, systemic lupus erythematosus (SLE), and Graves' disease. The prevalence of DED among patients with RA ranges from 18% to 90%, with severe symptoms reported in approximately 50% of RA patients.^[31,32] DED associated with RA as well as Sjogren's syndrome, is most likely due to an aqueous deficient state of the ocular surface.^[33]

Prior literature has shown that Sjogren's patients present with worse clinical dry eye parameters, including corneal staining and Schirmer's testing than non-Sjogren's dry eye patients.^[34] However, corneal staining among Sjogren's patients was found to resolve with appropriate dry eye treatment escalation.^[34] Keratoconjunctivitis sicca (KCS) affects approximately 25% of patients with SLE.^[35] The cause KCS is not completely understood but is thought to be related to cellular infiltrate of the major and accessory lacrimal glands leading to dysfunctional tear production.^[36] Patients with DED from Graves' disease suffer from both lacrimal gland dysfunction as well as evaporative dry eye disease from impaired eyelid closure.^[37]

There are no established differences in the treatment algorithm to optimize the ocular surface of patients with the autoimmune disease compared to DED patients without the auto-immune disease. However, understanding the ocular manifestations of a patient's autoimmune disease as well as the predominant type of DED that the patient suffers FROM (please change spelling) will help the clinician select the appropriate treatment modalities before cataract surgery.

Refractive surgery and dry eye disease

A common complication after refractive surgery is DED. The etiology of DED after refractive surgery is multifactorial. Among patients who have undergone laser *in-situ* keratomileusis (LASIK), DED is thought to be caused by iatrogenic corneal nerve damage from LASIK flap creation which disrupts the interaction between the afferent sensory nerves of the ocular surface and the efferent autonomic nerves to the lacrimal gland that modulate tear composition and secretion.^[38] There is also damage and loss of goblet cells due to the section device used intra-operatively for flap creation. Tear film changes have also been noted among postrefractive patients, specifically reduced tear secretion, increased tear instability, and increased tear osmolarity.^[38] Before performing cataract surgery, it is crucial that DED is diagnosed in patients who have a history of refractive surgery. A thorough history of symptoms and a complete examination should be performed before cataract surgery including TBUT, tear meniscus

measurements, Schirmer's testing, MMP-9 testing, tear osmolarity testing, and ocular surface staining. Patients should then be treated aggressively to ensure adequate lubrication of the ocular surface before preoperative biometry measurements.

Corneal transplantation and dry eye disease

DED associated with keratoplasty is a unique disease entity due as it is caused by both an impairment in corneal sensory innervation and a disruption of the normal tear film. The disruption of the tear film after keratoplasty can be secondary to a decrease in tear volume^[39] as well as concurrent decrease in tear film stability.^[40] Ocular surface dysfunction is most common after a full-thickness or anterior penetrating keratoplasty as compared to after an endothelial keratopathy. Aside from DED, surgeons also need to ensure that patients with prior keratoplasty do not suffer from recurrent epithelial breakdown from underlying corneal nerve dysfunction^[41] which can impact vision. Optimization of the ocular surface postkeratoplasty is critical to obtain stable measurements for cataract surgery, especially if a toric IOL is being considered to debulk postoperative astigmatism.

Combination of treatments for dry eye disease or ocular surface disease and measurements for cataract surgery

If a patient is seen in the clinic and diagnosed with DED or OSD, these conditions should be treated and the patient re-evaluated in approximately 4–6 weeks to assess for clinical improvement. A combination of the above-discussed approaches can be implemented for the adequate treatment depending on the severity of disease.

Patients are encouraged to remain out of soft contact lenses for approximately 1 week and out of rigid gas permeable contact lenses for several weeks (approximately 1 week/decade of contact lens wear) before any preoperative imaging to avoid any errors in keratometry. In patients with severe DED or OSD, longer contact lens holidays may be beneficial to rehabilitate the ocular surface. Obtaining multiple sets of measurements after treatment is critical. The authors typically obtain two serial sets of measurements spaced 2–4 weeks apart to verify the reproducibility of measurements, especially for premium IOL selection such as toric, trifocal, or extended-depth of focus IOLs. Measurements on topography, tomography, and keratometric values on biometry should be compared to ensure consistency. Clarity of corneal measurement points and mires as well as marked deviations from standard deviation values on the raw data should be assessed to verify accuracy of data acquisition. Inconsistencies in measurements per eye or between both eyes should prompt repeat measurements and clinical evaluation for ongoing surface disease. Oftentimes, marked changes in measured keratometry and biometry can be seen before and after DED treatment.

OTHER CORNEAL PATHOLOGIES

Preoperative lens calculations for cataract surgery require a smooth corneal refractive surface. Any irregularities including Salzmann's nodular degeneration or EBMD can

render preoperative keratometric and biometry measurements inaccurate.

Salzmann's nodular degeneration

Salzmann nodular degeneration (SND) lesions are blue, white, or grey corneal opacities that develop on the cornea. They occur most often in the peripheral cornea as compared to centrally and are typically found in the superior quadrants. SND lesions occur are situated between Bowman's layer and the epithelium causing thinning of the overlying epithelium.^[42] There can also be deposition of extracellular matrix in the nodule, stromal scarring, activated fibroblasts, and inflammatory B and T cell lymphocytes.^[43] The cause of SND is unknown however mechanical disruption from trauma or chronic corneal irritation may predispose patients to deposition of extracellular material and SND formation.^[43]

SND has been reported to have multiple effects on corneal topography. They not only create local corneal irregularities, but also their elevation accentuates flattening in other areas of the cornea [Figure 1a and b]. When the SND is located paracentrally, it can cause the pooling of tears and flatten of the cornea.^[44] Excessive epiphora provoked by SND has been anecdotally noted as another challenge in obtaining keratometry measurements.^[45]

Epithelial basement membrane dystrophy

Among the anterior corneal dystrophies, EBMD is the most common and occurs in 2%–6% of the population. EBMD can have many presentations including subepithelial fingerprint lines, geographic maps, epithelial microcysts or dots, and bleb

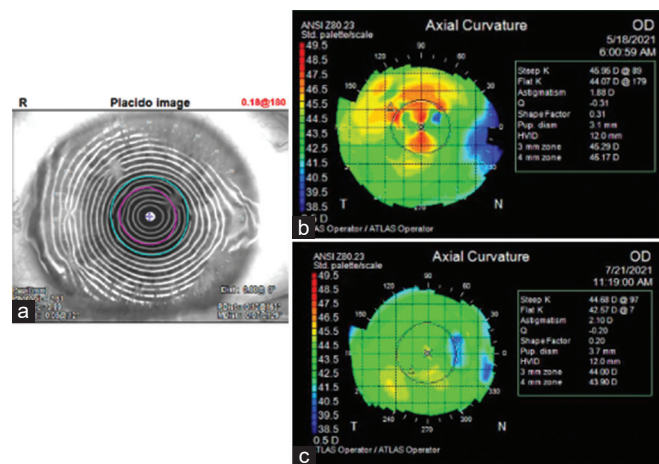


Figure 1: Effect of treatment of Salzmann's nodular degeneration. (a) Placido disc image showing disrupted mires nasally, temporally and centrally due to presence of multiple Salzmann's nodules in a patient with a visually significant cataract. (b) Topography showing irregular astigmatism with multiple areas of central steepening in areas of Salzmann's nodules. (c) Topography 1 month after superficial keratectomy with mitomycin C application and placement of amniotic membrane for Salzmann's nodular degeneration. Normalization of corneal architecture can be observed. This patient's uncorrected vision was 20/25 after superficial keratectomy. She is holding off on cataract surgery at this time given the marked improvement in vision after the superficial keratectomy

patterns.^[46] The corneal surface irregularity caused by EMBD leads to irregular astigmatism, higher-order aberrations, and visual disturbances [Figure 2a-c]. On optical coherence tomography imaging, EMBD appears as irregular thickened epithelial basement membrane due to pathologic duplication of the basement membrane.^[47] As a result of the abnormal corneal surface, preoperative biometry can be significantly affected and case reports of refractive surprise after cataract surgery due to unrecognized EMBD have been published.^[48]

Corneal irregularities and keratometry

A study from Goerlitz-Jessen *et al.*^[1] compared biometry measurements for patients with EMBD and SND before and after intervention for these corneal surface irregularities. The study showed that in eyes with EMBD, there was a statistically significant difference in K values before and after the intervention. Of the 26 eyes with EMBD, 81% had changes in the spherical IOL power closest to the spherical equivalent of 0 after treatment. Finally, the suggested toric lens power changed in 66% of eyes after treatment.

Similarly, for patients with SND, there were statistically significant differences in K values before and after treatment. Of the 13 patients with SND, 85% had changes in the spherical IOL power closest to the spherical equivalent of 0 after treatment and the toric lens power changed in 91% of eyes after treatment. Notably, the mean cylinder power change for toric IOLs was 1.5D. The results of this study show the significant impact of corneal surface irregularities on biometry measurements and the importance of recognizing and treating the irregularities before cataract surgery to ensure the best surgical outcome.

Pterygia

Pterygia are fibrovascular growths of the bulbar conjunctiva. While they are generally benign, uncontrolled growth can lead to both cosmetic and visual impairments for patients. Of note, there are a variety of treatment options for pterygium including conjunctival autografts, amniotic membranes, and the use of mitomycin C to prevent recurrence.^[49]

Generally, pterygia induce with the rule astigmatism.^[50] The effect of pterygium on the magnitude of astigmatism is demonstrated in a study that looked at refractive outcomes of patients who received simultaneous pterygium extraction and cataract surgery. The authors found that there was a significant myopic shift postoperatively as a result of the removal of the pterygium, especially when the pterygium was large.^[51] As a result, pterygium removal and cataract surgery is typically staged procedure to ensure the accuracy of the biometric calculations and lens selection. Interestingly, various groups have shown a somewhat predictable change in K values after pterygium removal. This suggests that when the appropriate correction is applied to biometric measurements taken before pterygium removal, cataract surgery can be performed with pterygium removal with a good refractive outcome.^[51-53]

Treatment and measurements for cataract surgery in patients with epithelial basement membrane dystrophy, Salzmann nodular degeneration and pterygia

If a patient is seen in clinic and diagnosed with EMBD, SND or pterygia, these conditions should be evaluated and addressed before cataract surgery.

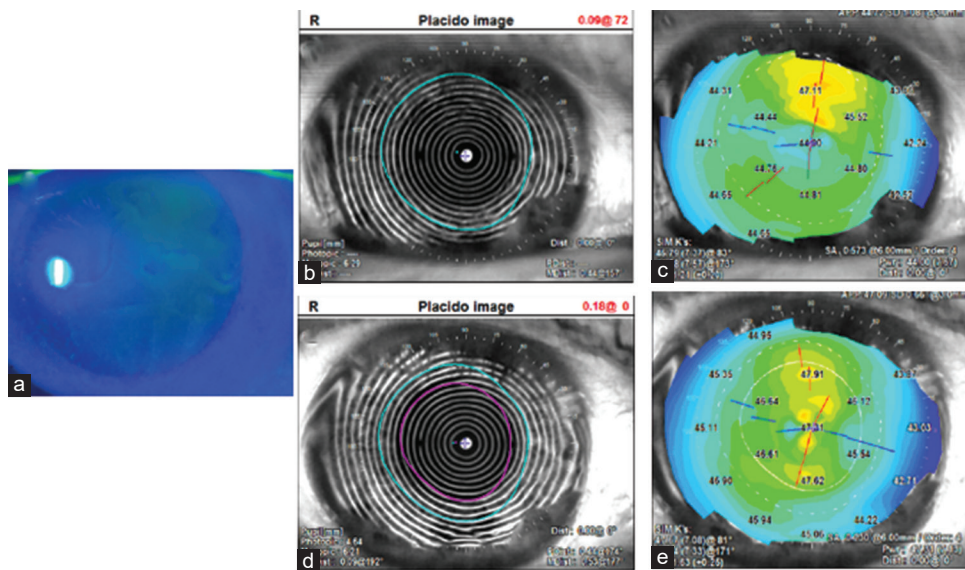


Figure 2: Effect of treatment of epithelial basement membrane dystrophy. (a) Clinical photo of a cornea stained with fluorescein showing the irregular negative staining characteristic of epithelial basement membrane dystrophy in a patient with a visually significant cataract. (b) Placido disc image displaying subtle disruption of mires in the paracentral cornea. (c) Topography shows irregular superior steepening in the area affected by epithelial basement membrane dystrophy. (d) Placido disc image 3 months after superficial keratectomy with amniotic membrane showing normalization of mires. (e) Topography 3 months after superficial keratectomy with amniotic membrane shows improvement of corneal architecture and more regular corneal astigmatism. This patient underwent cataract surgery with implantation of a toric intraocular lens with a 20/20 uncorrected vision postoperatively

In patients with EBMD, the location and severity of epithelial irregularity should be noted. If the irregular epithelium is within the central 4 mm of the cornea and causing fluctuations in vision and/or irregular astigmatism on topography, patients should be advised to undergo a superficial keratectomy with a bandage contact lens or amniotic membrane placement to normalize the epithelial surface. For patients with recurrent corneal erosions in the setting of EBMD, the stromal surface can be smoothed with a diamond burr or phototherapeutic keratectomy. The authors typically wait for 6–8 weeks for the corneal epithelium to stabilize. Repeat examinations, refractions, biometry, and topographies should be performed until stable and reproducible measurements are obtained [Figure 2d and 2e].

Similarly, in patients with SND causing blurred vision or visual fluctuations, superficial keratectomy with peeling of the Salzmann's nodules, and selective mitomycin C application and placement of a bandage contact lens or amniotic membrane should be performed to rehabilitate the ocular surface. The authors again wait 6–8 weeks for the corneal epithelium to remodel and stabilize. Serial clinical exams, refractions, biometry, and topographies should be performed until stable and reproducible measurements are obtained, at which point cataract surgery can be considered. In rare scenarios, remodeling of the ocular surface after superficial keratectomy can improve a patient's vision so much so that cataract surgery can be delayed [Figure 1c].

Finally, in patients with pterygia inducing significant astigmatism, pterygium excision should be performed before cataract surgery. The authors typically wait for 8–12 weeks for the corneal epithelium and ocular surface to heal before repeating biometry for and performing cataract surgery.

With all corneal pathologies, patients must be adequately counseled about the staged nature of procedures and the time investment these procedures require. In certain cases, epithelial remodeling can occur up to 3 or 4 months after a superficial keratectomy, and patients should be counseled that they may need to wait several weeks until cataract surgery. Some patients may opt to forego corneal procedures in effort to have more expedient cataract surgery and these patients should be counseled on the risks of refractive misses and the need for glasses postoperatively. Ultimately, thorough informed consent is critical to ensuring patients make the right choice when choosing which procedures are best suited for them.

CONCLUSION

While cataract surgery is one of the most common surgical procedures performed in ophthalmology, the variables that go into preoperative planning are numerous and precise. As demonstrated in this review article, a variety of factors including DED, OSD, and corneal pathologies can adversely impact preoperative biometric and keratometric data. Thankfully, an understanding of corneal surface disease combined with technological advances to address surface

irregularities allows surgeons to not only perform safe cataract surgeries but also surgeries with the best possible refractive outcomes.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

- Goerlitz-Jessen MF, Gupta PK, Kim T. Impact of epithelial basement membrane dystrophy and Salzmann nodular degeneration on biometry measurements. *J Cataract Refract Surg* 2019;45:1119-23.
- Röggla V, Leydolt C, Schartmüller D, Schwarzenbacher L, Meyer E, Abela-Formanek C, *et al.* Influence of artificial tears on keratometric measurements in cataract patients. *Am J Ophthalmol* 2021;221:1-8.
- Epitropoulos AT, Matossian C, Berdy GJ, Malhotra RP, Potvin R. Effect of tear osmolarity on repeatability of keratometry for cataract surgery planning. *J Cataract Refract Surg* 2015;41:1672-7.
- Montés-Micó R, Alió JL, Muñoz G, Pérez-Santonja JJ, Charman WN. Postblink changes in total and corneal ocular aberrations. *Ophthalmology* 2004;111:758-67.
- Craig JP, Nichols KK, Akpek EK, Caffery B, Dua HS, Joo CK, *et al.* TFOS DEWS II definition and classification report. *Ocul Surf* 2017;15:276-83.
- Hashemi H, Khabazkhoob M, Kheirkhah A, Emamian MH, Mehravaran S, Shariati M, *et al.* Prevalence of dry eye syndrome in an adult population. *Clin Exp Ophthalmol* 2014;42:242-8.
- de Paiva CS. Effects of aging in dry eye. *Int Ophthalmol Clin* 2017;57:47-64.
- Gupta PK, Drinkwater OJ, VanDusen KW, Brissette AR, Starr CE. Prevalence of ocular surface dysfunction in patients presenting for cataract surgery evaluation. *J Cataract Refract Surg* 2018;44:1090-6.
- Schaumberg DA, Gulati A, Mathers WD, Clinch T, Lemp MA, Nelson JD, *et al.* Development and validation of a short global dry eye symptom index. *Ocul Surf* 2007;5:50-7.
- Trattler WB, Majmudar PA, Donnenfeld ED, McDonald MB, Stonecipher KG, Goldberg DF. The Prospective Health Assessment of Cataract Patients' Ocular Surface (PHACO) study: the effect of dry eye. *Clin Ophthalmol* 2017;11:1423-30.
- Chen Z, Lin X, Qu B, Gao W, Zuo Y, Peng W, *et al.* Preoperative expectations and postoperative outcomes of visual functioning among cataract patients in urban Southern China. *PLoS One* 2017;12:e0169844.
- Starr CE, Gupta PK, Farid M, Beckman KA, Chan CC, Yeu E, *et al.* An algorithm for the preoperative diagnosis and treatment of ocular surface disorders. *J Cataract Refract Surg* 2019;45:669-84.
- Stern ME, Gao J, Siemasko KF, Beuerman RW, Pflugfelder SC. The role of the lacrimal functional unit in the pathophysiology of dry eye. *Exp Eye Res* 2004;78:409-16.
- Zhang X, Vimalin Jeyalatha M, Qu Y, He X, Ou S, Bu J, *et al.* Dry eye management: Targeting the ocular surface microenvironment. *Int J Mol Sci* 2017;18:1398.
- Fiscella RG. Understanding dry eye disease: A managed care perspective. *Am J Manag Care* 2011;17 Suppl 16:S432-9.
- Ervin AM, Law A, Pucker AD. Punctal occlusion for dry eye syndrome. *Cochrane Database Syst Rev* 2017;6:CD006775.
- De Paiva CS, Raince JK, McClellan AJ, Shanmugam KP, Pangelinan SB, Volpe EA, *et al.* Homeostatic control of conjunctival mucosal goblet cells by NKT-derived IL-13. *Mucosal Immunol* 2011;4:397-408.
- Kim EC, Choi JS, Joo CK. A comparison of vitamin A and cyclosporine a 0.05% eye drops for treatment of dry eye syndrome. *Am J Ophthalmol* 2009;147:206-13.e3.
- Holland EJ, Luchs J, Karpecki PM, Nichols KK, Jackson MA, Sall K, *et al.* Lifitegrast for the treatment of dry eye disease: Results of a phase III, randomized, double-masked, placebo-controlled trial (OPUS-3). *Ophthalmology* 2017;124:53-60.

20. Lollett IV, Galor A. Dry eye syndrome: Developments and lifitegrast in perspective. *Clin Ophthalmol* 2018;12:125-39.
21. Sheppard JD, Torkildsen GL, Lonsdale JD, D'Ambrosio FA Jr, McLaurin EB, Eiferman RA, *et al.* Lifitegrast ophthalmic solution 5.0% for treatment of dry eye disease: Results of the OPUS-1 phase 3 study. *Ophthalmology* 2014;121:475-83.
22. Tauber J, Karpecki P, Latkany R, Luchs J, Martel J, Sall K, *et al.* Lifitegrast ophthalmic solution 5.0% versus placebo for treatment of dry eye disease: Results of the randomized phase III OPUS-2 study. *Ophthalmology* 2015;122:2423-31.
23. Greiner JV. A single LipiFlow® thermal pulsation system treatment improves meibomian gland function and reduces dry eye symptoms for 9 months. *Curr Eye Res* 2012;37:272-8.
24. Finis D, Hayajneh J, König C, Borrelli M, Schrader S, Geerling G. Evaluation of an automated thermodynamic treatment (LipiFlow®) system for meibomian gland dysfunction: A prospective, randomized, observer-masked trial. *Ocul Surf* 2014;12:146-54.
25. Gupta PK, Vora GK, Matossian C, Kim M, Stinnett S. Outcomes of intense pulsed light therapy for treatment of evaporative dry eye disease. *Can J Ophthalmol* 2016;51:249-53.
26. Chiang CC, Lin JM, Chen WL, Tsai YY. Allogeneic serum eye drops for the treatment of severe dry eye in patients with chronic graft-versus-host disease. *Cornea* 2007;26:861-3.
27. Matsumoto Y, Dogru M, Goto E, Ohashi Y, Kojima T, Ishida R, *et al.* Autologous serum application in the treatment of neurotrophic keratopathy. *Ophthalmology* 2004;111:1115-20.
28. Pan Q, Angelina A, Marrone M, Stark WJ, Akpek EK. Autologous serum eye drops for dry eye. *Cochrane Database Syst Rev* 2017;2:CD009327.
29. Geerling G, MacLennan S, Hartwig D. Autologous serum eye drops for ocular surface disorders. *Br J Ophthalmol* 2004;88:1467-74.
30. Bavinger JC, DeLoss K, Mian SI. Scleral lens use in dry eye syndrome. *Curr Opin Ophthalmol* 2015;26:319-24.
31. Zlatanović G, Veselinović D, Cekić S, Zivković M, Dorđević-Jocić J, Zlatanović M. Ocular manifestation of rheumatoid arthritis-different forms and frequency. *Bosn J Basic Med Sci* 2010;10:323-7.
32. Lemp MA. Dry eye (Keratoconjunctivitis Sicca), rheumatoid arthritis, and Sjögren's syndrome. *Am J Ophthalmol* 2005;140:898-9.
33. Nelson JD, Craig JP, Akpek EK, Azar DT, Belmonte C, Bron AJ, *et al.* TFOS DEWS II introduction. *Ocul Surf* 2017;15:269-75.
34. Cui D, Mathews P, Li G, VanCourt S, Akpek E. Outcomes of Sjögren's versus non-Sjögren's related dry eye in a longitudinal, tertiary clinic-based sample. *PLoS One* 2021;16:e0261241.
35. Read RW. Clinical mini-review: Systemic lupus erythematosus and the eye. *Ocul Immunol Inflamm* 2004;12:87-99.
36. Shoughy SS, Tabbara KF. Ocular findings in systemic lupus erythematosus. *Saudi J Ophthalmol* 2016;30:117-21.
37. Selter JH, Gire AI, Sikder S. The relationship between Graves' ophthalmopathy and dry eye syndrome. *Clin Ophthalmol* 2015;9:57-62.
38. Shtein RM. Post-LASIK dry eye. *Expert Rev Ophthalmol* 2011;6:575-82.
39. Hara S, Kojima T, Dogru M, Uchino Y, Goto E, Matsumoto Y, *et al.* The impact of tear functions on visual outcome following keratoplasty in eyes with keratoconus. *Graefes Arch Clin Exp Ophthalmol* 2013;251:1763-70.
40. Lin X, Xu B, Sun Y, Zhong J, Huang W, Yuan J. Comparison of deep anterior lamellar keratoplasty and penetrating keratoplasty with respect to postoperative corneal sensitivity and tear film function. *Graefes Arch Clin Exp Ophthalmol* 2014;252:1779-87.
41. Miller DD, Hasan SA, Simmons NL, Stewart MW. Recurrent corneal erosion: A comprehensive review. *Clin Ophthalmol* 2019;13:325-35.
42. Hurmeric V, Yoo SH, Karp CL, Galor A, Vajzovic L, Wang J, *et al.* *In vivo* morphologic characteristics of Salzmann nodular degeneration with ultra-high-resolution optical coherence tomography. *Am J Ophthalmol* 2011;151:248-56.e2.
43. Yoon KC, Park YG. Recurrent Salzmann's nodular degeneration. *Jpn J Ophthalmol* 2003;47:401-4.
44. Koch DD. Impact of Salzmann's lesions on corneal curvature. *J Cataract Refract Surg* 1995;21:111-2.
45. Paranjpe V, Galor A, Monsalve P, Dubovy SR, Karp CL. Salzmann nodular degeneration: Prevalence, impact, and management strategies. *Clin Ophthalmol* 2019;13:1305-14.
46. Buffault J, Zéboulon P, Liang H, Chiche A, Luzu J, Robin M, *et al.* Assessment of corneal epithelial thickness mapping in epithelial basement membrane dystrophy. *PLoS One* 2020;15:e0239124.
47. El Sanharawi M, Sandali O, Basli E, Bouheraoua N, Ameline B, Goemaere I, *et al.* Fourier-domain optical coherence tomography imaging in corneal epithelial basement membrane dystrophy: A structural analysis. *Am J Ophthalmol* 2015;159:755-63.
48. Ho VW, Stanojic N, O'Brart NA, O'Brart DP. Refractive surprise after routine cataract surgery with multifocal IOLs attributable to corneal epithelial basement membrane dystrophy. *J Cataract Refract Surg* 2019;45:685-9.
49. Janson BJ, Sikder S. Surgical management of pterygium. *Ocul Surf* 2014;12:112-9.
50. Avisar R, Loya N, Yassar Y, Weinberger D. Pterygium-induced corneal astigmatism. *Isr Med Assoc J* 2000;2:14-5.
51. Kamiya K, Shimizu K, Iijima K, Shoji N, Kobashi H. Predictability of intraocular lens power calculation after simultaneous pterygium excision and cataract surgery. *Medicine (Baltimore)* 2015;94:e2232.
52. Koc M, Uzel MM, Aydemir E, Yavrum F, Kosekahya P, Yilmazbaş P. Pterygium size and effect on intraocular lens power calculation. *J Cataract Refract Surg* 2016;42:1620-5.
53. Takahashi S, Manabe S, Ota N, Hayashi K. Prediction of corneal curvature radius after pterygium surgery using anterior segment optical coherence tomography. *Jpn J Ophthalmol* 2019;63:145-50.