Optimization of the Ocular Surface Through Treatment of Ocular Surface Disease Before Ophthalmic Surgery: A Narrative Review

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ABSTRACT

Ocular surface disease commonly exists in individuals requiring ophthalmic surgery and may compromise the structure and function of ocular surface components. Ophthalmic surgery may further affect the ocular surface by injuring the epithelium and sensory nerves, disrupting the tear film, or causing local inflammation. Medical management of ocular surface disease prior to ophthalmic surgery aids in reducing inflammation, resolving infection, improving epithelial pathology, stabilizing the tear film, and easing patient symptoms, promoting positive long-term outcomes and minimizing the incidence of postoperative complications. This review summarizes frequently encountered ocular surface diseases and available preoperative medical management options, discusses common ophthalmic surgeries and their effects on the ocular surface, examines potential postoperative complications, and defines recommendations for postoperative ocular surface maintenance.

Keywords: Cataract surgery; Dry eye disease; Ocular surface disease; Ocular surface optimization; Ophthalmic surgery; Refractive surgery

Key Summary Points

Individuals requiring ophthalmic surgery commonly have pre-existing ocular surface diseases due to age or chronic use of topical ophthalmic medications, which may compromise the structure and function of ocular surface components.

Ophthalmic surgeries may further compromise the ocular surface by injuring the ocular epithelium and nerves, disrupting the tear film, and inducing inflammation.

Prior to ophthalmic surgery, appropriate medical management should be instituted for extant ocular surface diseases to reduce inflammation, treat infection, improve epithelial lesions, and stabilize the tear film.

Preoperative ocular surface optimization promotes positive surgical outcomes, improves patient satisfaction, and minimizes postoperative complications.
INTRODUCTION

The ocular surface comprises the lacrimal, accessory lacrimal, and meibomian glands, eyelids, eyelashes, cornea, conjunctiva, and tear film [1]. These structures are interconnected through the continuity of ocular surface epithelia, sensory and motor innervation, and immune, endocrine, and vascular systems [2]. Ocular surface health is vital to maintaining normal vision and may be affected by multiple disease processes. Due to the interrelation of ocular surface components, associated diseases commonly occur in a continuum where one pathology may promote or perpetuate another [1].

Ocular surface disease is more common in elderly individuals, a population that also frequently requires ophthalmic surgery [3]. Pre-existing ocular surface disease poses surgical challenges including chronic inflammation, acquired structural abnormalities, and decreased corneal clarity [4]. Additionally, surgery may further compromise the ocular surface secondary to mechanical injury of the epithelium and nerves, disturbance of normal tear film, and local inflammation [5]. Surgical procedures performed in eyes with pre-existing ocular surface disease may worsen the disease process and/or affect optimal vision, in addition to other potential adverse outcomes [4]. Whenever possible, optimizing the ocular surface prior to surgery by treating ocular surface disease may improve surgical outcomes, increase postoperative patient satisfaction, and decrease postoperative complications [4, 6].

This review provides an overview of common pre-existing ocular surface diseases found in ophthalmic surgery patients and available medical treatments, as well as frequently performed ophthalmic surgeries that may affect the ocular surface; we also define postoperative complications that may result with failure to optimize the preoperative ocular surface. It is based on previously conducted studies and does not contain any new studies with human participants or animals performed by either of the authors.

COMMON PRE-EXISTING OCULAR SURFACE DISEASES IN OPHTHALMIC SURGERY CANDIDATES

Demodex Infestation

Demodex is a common microscopic skin mite; eyelash follicle infestation is estimated to occur in 41% of adults, with increased prevalence in immunocompromised patients [7, 8]. Demodex folliculorum occupies the eyelash follicle and D. brevis inhabits eyelash sebaceous and meibomian glands [9]. Infestation may be asymptomatic or cause cylindrical eyelash dandruff, ocular discomfort, and visual disturbance [10, 11]. Demodex infestation may promote blepharitis, conjunctivitis, and meibomian gland dysfunction (MGD), but its pathogenicity remains controversial [10]. In patients undergoing ophthalmic surgeries such as cataract surgery, demodicosis may affect postoperative tear production and tear film homeostasis [12].

Blepharitis

Blepharitis is a chronic inflammatory disorder associated with eyelid redness and irritation; anterior blepharitis affects the lid margin, lashes, and eyelash follicles, and posterior blepharitis mainly affects the meibomian glands [13]. In a 2009 study, blepharitis prevalence ranged from 37 to 47% of eye care patients [14]. Lifelong management is suggested to reduce symptoms, minimize ocular damage, and prevent visual impairment [13]. Common sequelae include abnormal tear film and ocular inflammation, promoting conjunctivitis, keratitis, or dry eye disease (DED) [13]. Additionally, pre-existing blepharitis may be associated with postoperative endophthalmitis in patients undergoing ophthalmic surgery [13, 15].

Meibomian Gland Dysfunction

The meibomian glands open at the eyelid margin and produce meibum, a lipid-based secretion that stabilizes the tear film [16].
Meibomian gland dysfunction is a chronic abnormality involving decreased or irregular meibum secretion due to terminal duct obstruction and/or altered glandular function [16]. Its reported prevalence varies from 3.5 to 69% and appears higher in Asian populations [16]. Proposed etiologic factors include blepharitis, *Demodex* infestation, DED, and contact lens wear [16]. Symptoms include ocular irritation and vision fluctuation [16]. In MGD, insufficient or abnormal meibum causes tear film hyperosmolarity with subsequent increased tear evaporation, promoting blepharitis, DED, and ocular surface damage [16]. As the primary contributor to evaporative DED, MGD is considered the leading cause of DED [1]. Meibomian gland dysfunction has been found in over 50% of patients presenting for cataract surgery; detection and management of this condition prior to ophthalmic surgery is important to optimize surgical outcomes [17].

**Allergic Conjunctivitis**

Allergic conjunctivitis is a chronic condition, often beginning in childhood and exacerbated by environmental allergens [18]. Estimated prevalence is up to 40% in the US [19]. Symptoms include conjunctival injection, eyelid edema, and excessive lacrimation; allergic conjunctivitis also commonly occurs with allergic rhinitis [18]. Chronic allergic conjunctivitis may contribute to MGD, DED, ocular surface damage or structural changes, and reduced visual function [18]. Allergic conjunctivitis has been associated with increased risk of postoperative refractive surgery complications, including corneal haze, myopic regression, and diffuse lamellar keratitis [20, 21].

**Dry Eye Disease**

Dry eye disease is a chronic, multifactorial disease characterized by loss of tear film homeostasis with an estimated prevalence of 5 to 33% in US adults [1, 22]. Ocular disorders, such as blepharitis and MGD, as well as numerous systemic diseases, such as Sjögren syndrome, diabetes mellitus, and autoimmune conditions like rheumatoid arthritis and systemic lupus erythematosus, can contribute to the development of DED [2]. These conditions may impair the normal function of the cornea, conjunctiva, lacrimal glands, or meibomian glands, which secrete and regulate tear film components [1]. Dry eye disease has been traditionally classified into aqueous deficient and evaporative subtypes, but recent research suggests that in most cases, these etiologies co-exist or occur as a continuum [1]. The resultant qualitative and/or quantitative tear deficiency initiates a vicious cycle of ocular surface inflammation and damage that may affect vision and cause symptoms of ocular discomfort [1, 2]. Pre-existing DED has been shown to negatively impact refractive surgical outcomes and may convey increased risk of postoperative infections and surgical complications [23].

**Epithelial Basement Membrane Dystrophy**

Epithelial basement membrane dystrophy (EBMD) is an often degenerative disorder in which abnormal corneal basal laminar material causes development of an irregular epithelium prone to recurring erosions [24]. It is estimated to occur in < 2% of the population [25]. Most patients are asymptomatic; symptoms include ocular pain, epiphora, and decreased visual acuity [24, 26]. Preoperative testing for EBMD is imperative in patients undergoing ophthalmic surgery, as the disorder may significantly alter biometry measurements, increase the risk of postoperative wound healing complications, and induce irregular astigmatism [27, 28].

**Salzmann’s Nodular Degeneration**

Salzmann’s nodular degeneration (SND) is a rare degenerative disease occurring most often in middle-aged women; it may be idiopathic or associated with past corneal inflammation [29]. It involves formation of bluish-gray corneal nodules or sheets, which are cellular accumulations of extracellular matrix between thinned corneal epithelium and Bowman’s layer [30]. Symptoms include ocular discomfort and visual disturbance [29]. Nodules can impede vision,
induce corneal astigmatism, and disrupt the tear film [29]. Additionally, SND can significantly alter preoperative biometry measurements in patients undergoing cataract surgery [28].

PREOPERATIVE OPTIMIZATION OF THE OCULAR SURFACE

Each unique ocular surface disease may ultimately affect the ocular surface similarly. Shared potential outcomes include inflammation and damage to ocular structures, patient discomfort, and compromised vision. The appropriate medical management should be implemented in affected patients to decrease likelihood of these outcomes.

Effective medical management of ocular surface diseases involves following established treatment guidelines for each condition. Many therapeutics exist, including topical and systemic medications, procedural therapies, and lifestyle adjustments. As ocular surface diseases are often multifactorial, multifaceted therapy plans to address singular conditions are frequently necessary. Medical treatment of allergic conjunctivitis, for example, may require topical antihistamines to address the underlying etiology, topical anti-inflammatory agents such as corticosteroids to treat severe inflammation, topical lubricants to provide symptom relief, and daily eyelid cleansing to reduce allergen exposure [18].

Additionally, ocular surface diseases commonly occur in continuum, necessitating comprehensive treatment protocols to address each component [31]. For example, blepharitis, MGD, and DED are often interrelated: posterior blepharitis impairs meibomian gland function and abnormal meibomian secretion may cause tear film alterations, resulting in evaporative DED [1, 13]. To address all involved etiologies, a therapeutic approach in affected patients may call for topical lubricants, topical or oral antibiotics, and/or topical anti-inflammatory agents like cyclosporine; procedural therapies like meibomian gland thermal pulsation and expression; and lifestyle adjustments like implementing routine eyelid cleansing and/or warm compressing.

Overall goals of preoperative ocular surface disease management should include reducing inflammation, resolving infection, improving epithelial pathology, stabilizing the tear film, and easing symptoms before ophthalmic surgery, which increases likelihood of positive outcomes [31]. Medical treatment options for common ocular surface diseases are summarized in Table 1.

COMMON OPHTHALMIC SURGERIES AND THEIR EFFECTS ON THE OCULAR SURFACE

Prior to ophthalmic surgery, preoperative assessments are necessary to evaluate ocular health and allow detection and treatment of any of the previously described ocular surface diseases. Since ophthalmic surgeries may exacerbate pre-existing ocular surface diseases, induce their de novo occurrence, or cause other ocular surface damage, preoperative management of any existent conditions to optimize the ocular surface is vital [5, 32, 33]. The American Society of Cataract and Refractive Surgery (ASCRS) Cornea Clinical Committee recently developed the ASCRS Preoperative Ocular Surface Disease Algorithm, a clinical diagnostic tool to help surgeons diagnose and treat ocular surface disease before refractive surgery [34]. Potential presurgical assessments are summarized in Table 2.

Common ophthalmic surgeries affecting the ocular surface include cataract surgeries, refractive surgeries, glaucoma surgeries, and corneal transplants. These surgeries employ different procedures and treat distinct conditions but often impact the ocular surface in similar ways. For example, multiple ophthalmic surgeries involve transection of corneal afferent nerves, possibly employing vertical nasal or temporal incisions that disrupt the corneal nerve plexus. This interrupts the sensory feedback mechanism vital to tear secretion; impaired corneal sensation secondary to corneal afferent nerve damage then causes infrequent blinking, decreased lacrimation, and increased tear
| Table 1  | Summary of medical management options |
|----------|----------------------------------------|
| **Topical products** |  |
| **Ocular lubricants** | Use(s): Allergic conjunctivitis, DED, epithelial basement membrane dystrophy, SND | Function: Improvement of tear quantity and/or quality | Mechanism of action: Ocular lubrication [31] Supplementation or substitution of tear film components [31] |
| | Contraindications or drawbacks: Often inadequate for long-term management [31] |
| **Antihistamines** | Use(s): Allergic conjunctivitis | Function: Ocular allergy symptom relief | Mechanism of action: H1 receptor antagonists |
| | Contraindications: None |
| **Antibiotics** | Use(s): Blepharitis | Function: Treatment of infections | Mechanism of action: Bactericidal or bacteriostatic action |
| | Contraindications: Risk of resistance with chronic or repeated use [58] |
| **Azithromycin** | Use(s): Blepharitis, MGD | Function: Reduction of inflammation | Mechanism of action: Bacteriostatic action [59] Inhibition of multiple inflammatory mediators [31] Bacterial lipase inhibition [59] |
| **Anti-inflammatories** |  |
| **Non-steroidal anti-inflammatories** | Use(s): Allergic conjunctivitis | Function: Reduction of inflammation | Mechanism of action: Cyclooxygenase inhibition |
| | Contraindications: Risk of corneal toxicity with epithelial compromise [60] |
| **Corticosteroids** | Use(s): Allergic conjunctivitis, blepharitis, DED, MGD | Function: Reduction of inflammation | Mechanism of action: Inhibition of multiple inflammatory mediators |
| | Contraindications: Risk of cataracts or elevated IOP with long-term use [13] |
| **Immunomodulators** |  |
| **Cyclosporine A** | Use(s): Allergic conjunctivitis, blepharitis, DED, MGD | Function: Reduction of inflammation | Mechanism of action: Calcineurin inhibition [61] |
| | Contraindications: Long-term therapy required [31] |
| **Cyclosporine ophthalmic solution (Cequa™, Sun Pharmaceutical Industries, Inc.)** | Use(s): DED | Function: Increase in tear production | Mechanism of action: |

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| Use(s) | Function | Mechanism of action | Contraindications or drawbacks |
|--------|----------|---------------------|-------------------------------|
| Cyclosporine ophthalmic emulsion (Restasis®, Allergan, Inc.) | DED | Increase in tear production [62] | |
| Lifitegrast 5% ophthalmic solution (Xiidra®, Novartis Pharmaceuticals Corporation) | DED | Treatment of DED signs and symptoms [63] | None |
| Systemic products | | | |
| Omega-3 fatty acids | Blepharitis, DED, MGD | Reduction of inflammation [64] | Inhibition of proinflammatory mediators [64] | Debated efficacy [65] |
| Oral antibiotics | | | |
| Macrolides | Blepharitis, MGD | Treatment of infection | Bacteriostatic and/or bactericidal action | Long-term use may cause resistance [31] |
| | | Reduction of inflammation [13] | Inhibition of multiple inflammatory mediators [31] | Oral azithromycin may cause abnormal cardiac electrical activity [66] |
| Tetracyclines | Blepharitis, DED, MGD | Treatment of infection | Bacteriostatic action | Long-term use may cause resistance [31] |
| | | Reduction of inflammation [13] | Inhibition of multiple inflammatory mediators [31] | Contraindicated during pregnancy and in young children [13] |
| | | Tear film stabilization [67] | Bacterial lipase inhibition [31] | Risk of photosensitization, GI upset, vaginitis [13] |
| Use(s) | Function | Mechanism of action | Contraindications or drawbacks |
|--------|----------|---------------------|-------------------------------|
| **Procedural therapies** | | | |
| Meibomian gland thermal pulsation and expression | Blepharitis, MGD | Meibomian gland expression [13] | None |
| | | Improvement of meibomian gland function [13] | |
| | | Relief of MGD symptoms [13] | |
| Punctal occlusion | DED | Tear film stabilization | Obstruction of punctal orifices for reduction of tear drainage [31] |
| | | | Controversial with active ocular surface inflammation [31] |
| | | | Risks include epiphora, local irritation, infection [31] |
| Intense pulsed light | Blepharitis, DED, MGD | Reduction of eyelid inflammation and telangiectasia [13] | Mechanism of action unclear; use of light source with photothermal effect [13] |
| MBE | Blepharitis, Demodex infestation, MGD | Removal of eyelid margin debris [13] | Mechanical debridement and exfoliation of eyelid margin [13] |
| | | Resolution of meibomian gland obstruction [13] | |
| **Other** | | | |
| **Lifestyle changes** | | | |
| Regular screen breaks | DED | Tear film stabilization | Promotion of normal blinking to prevent abnormal tear film evaporation [68] |
| | | | None |
osmolarity, leading to tear film instability [5, 35]. The following sections review common ophthalmic surgeries and their peri- and post-operative effects on the ocular surface.

Cataract Surgery

Cataract surgery is the most common ophthalmic surgery performed in the Western world, with increasing annual incidence...
Ocular surface damage during cataract surgery may be caused by exposure to topical sterilizing solutions, operating microscope light phototoxicity, lid speculum trauma, transection of corneal epithelium and nerves, extensive corneal irrigation, or use of preservative-containing eyedrops [5]. Many patients experience some postoperative ocular inflammation and temporarily decreased conjunctival goblet cell density—changes that may lead to ocular symptoms including foreign body sensation, eye fatigue, and ocular redness [5, 38].

Refractive Surgeries

Laser-Assisted In Situ Keratomileusis
Laser-assisted in situ keratomileusis (LASIK) corrects myopia, hyperopia, presbyopia, and/or astigmatism through laser modification of corneal architecture. LASIK damages the ocular surface by transecting the corneal epithelium and afferent nerves during corneal flap creation and through extensive intraoperative irrigation and suction ring use, which may both damage conjunctival goblet cells [35, 39, 40]. Additionally, LASIK alters corneal curvature, which may affect lubrication of the ocular surface during blinking [35]. Patients undergoing LASIK may have short-term, long-term, or permanent

| Preoperative assessment | Concept being evaluated |
|------------------------|-------------------------|
| Patient history        | Ocular disease signs or symptoms [16] |
| Clinical examination   | Ocular morphology [16] |
|                        | Ocular disease signs [16] |
| Slit-lamp biomicroscopy| Ocular morphology [16] |
|                        | Meibomian gland expressibility: meibum quality and volume [16] |
|                        | Tear film lipid layer, thickness, spread time and rate [16] |
| Tear evaluation        |                                      |
| Osmolarity test        | Tear osmolarity [16] |
| Tear breakup time      | Tear film stability: time between blink and break in tear film [16] |
| Meniscus height        | Tear volume [16] |
| Schirmer test          | Tear secretion over 5 min [16] |
| Ocular surface staining| Integrity of conjunctival and corneal epithelium [13] |
| Tonometry              | Intraocular pressure [13] |
| Fundoscopic exam       | Ocular fundus (retina, macula, optic nerve) morphology [72] |
| Visual acuity test     | Accuracy of distance vision [73] |
| Visual field test      | Extent of peripheral vision [74] |
| Biometry               | Corneal refractive power [75] |
|                        | Eye length [75] |
| Corneal topography/keratometry | Anterior corneal surface curvature [75] |
| Corneal tomography     | Corneal thickness and shape [76] |
postoperative tear film instability and decreased tear secretion [40, 41].

**Limbal Relaxing Incisions**

Limbal relaxing incisions are non-perforating corneal incisions made during or after cataract surgery to reduce pre-existing astigmatism. Placement at the limbus minimizes central corneal irregularity and patient discomfort [42]. However, transection of the corneal epithelium and nerves dependent on incision placement and variability due to the axis of astigmatism or surgeon dependence may cause postoperative complications [42].

**Femtosecond Laser-Assisted Astigmatic Keratotomy**

Femtosecond laser-assisted astigmatic keratotomy (FSLAK) corrects astigmatism by making relaxing corneal stromal incision(s) at the site of greatest astigmatism, leaving the epithelium intact. Laser use has increased accuracy and safety compared to manual blades, reducing incisional variability [43]. However, FSLAK may still cause postoperative ocular surface inflammation due to coupling difficulties, and/or corneal scarring [44].

**Conductive Keratoplasty**

Conductive keratoplasty corrects mild-to-moderate hyperopia via delivery of radiofrequency current through a tip inserted into multiple sites in the peripheral cornea, heating and contracting stromal collagen, and increasing central corneal curvature [45]. Surgical complications are uncommon [45]. However, because regression of procedural correction invariably occurs over time, this procedure is infrequently performed in the modern day [46].

**Radial Keratotomy**

During radial keratotomy, multiple radial incisions are made in the anterior cornea to correct myopia [47]. Though commonly performed in the late 20th century, due to concern for poor long-term refractive stability and overcorrection and with the introduction of more dependable laser surgeries, the popularity of this procedure has decreased [48].

**Trabeculectomy**

Trabeculectomy, a common surgical glaucoma treatment, lowers intraocular pressure through creation of a trans-scleral fistula [49]. Trabeculectomy promotes ocular surface inflammation with conjunctival bleb formation at the limbus and increases tear film osmolarity and dry eye symptoms [49, 50]. Inflammation overlying the fistula, whether surgically induced or secondary to preservative toxicity from topical hypotensive medications, may cause conjunctival thickening and scarring, potentially leading to surgical failure [49, 50].

**Corneal Transplantation**

Corneal transplantation, or penetrating keratoplasty, is a main method of sight restoration for corneal blindness [51]. Keratoplasty may lead to postoperative corneal epithelial defects, impaired meibomian gland function, tear film instability secondary to sutures or wound edge irregularity, and ocular surface inflammation, potentially leading to allograft rejection [32].

**Pterygium Surgery**

A pterygium is a fibroblastic growth continuous with the conjunctiva that extends onto the cornea [52]. It may obstruct vision and/or induce corneal deformity and astigmatism, restrict ocular movement, and cause patient discomfort [53]. Surgical removal may successfully reverse corneal topographic changes [52]. Newer surgical techniques have decreased the pterygium recurrence rate to an estimated 1.22%, but patients may still experience ocular irritation following surgery [53, 54].

**POSTOPERATIVE COMPLICATIONS AND MAINTENANCE**

The risk and incidence of postoperative ophthalmic surgery complications vary by procedure, but surgeries affecting the ocular surface in similar ways are often associated with comparable adverse effects. For example, despite
utilizing distinct surgical techniques, cataract surgery and LASIK both disrupt corneal nerve function as previously described and thus are both frequently associated with postoperative DED [5, 33]. Failure of preoperative ocular surface optimization makes postoperative complications increasingly likely [55]. Common complications of ophthalmic surgeries and their risk factors are listed in Table 3.

In addition to presurgical evaluation and management, postsurgical ocular surface maintenance is also vital for positive outcomes. Medical management should be adjusted as appropriate to address any postoperative ocular surface changes. Artificial tears solutions should be administered to protect the ocular surface and assist in regeneration of normal tear film following surgery, and any appropriate pharmacological interventions should also be utilized [40]. Eyelid cleansing should be implemented to help manage blepharitis, reduce Demodex populations, and minimize allergic conjunctivitis [13]. Ocular sun protection should be used to reduce UV light exposure, which can promote cataract formation and ocular neoplasms [56, 57]. Eye protection also decreases airborne irritant exposure [18]. Patients should regularly visit their ophthalmologist or optometrist, especially for symptom exacerbation [13].

### CONCLUSIONS

Ophthalmic surgeries may compromise the ocular surface in multiple similar ways, and resulting postsurgical complications can affect patients’ visual function and ocular health and comfort. Preoperatively optimizing the ocular surface through appropriate medical management of any pre-existing ocular surface diseases minimizes the incidence of postoperative complications and improves surgical outcomes.

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REFERENCES

1. Craig JP, Nichols KK, Akpek EK, et al. TFOS DEWS II definition and classification report. Ocul Surf. 2017;15(3):276–83.
2. Bron AJ, de Paiva CS, Chauhan SK, et al. TFOS DEWS II pathophysiology report. Ocul Surf. 2017;15(3):438–510.
3. 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(9):1090–6.
4. Donthineni PR, Das AV, Shanbhag SS, Basu S. Cataract surgery in dry eye disease: visual outcomes and complications. Front Med (Lausanne). 2020;7:575834.
5. Kasetsuwan N, Satipitakul V, Changul T, Jarlyakosol S. Incidence and pattern of dry eye after cataract surgery. PLoS ONE. 2013;8(11):e78657.
6. Gibbons A, Ali TK, Waren DP, Donaldson KE. Causes and correction of dissatisfaction after implantation of presbyopia-correcting intraocular lenses. Clin Ophthalmol. 2016;10:1965–70.
7. Karincaoglu Y, Esrefoglu Seyhan M, Bayram N, Aycan O, Taskapan H. Incidence of Demodex folliculorum in patients with end stage chronic renal failure. Ren Fail. 2005;27(5):495–9.
8. Wesolowska M, Knysz B, Reich A, et al. Prevalence of Demodex spp. in eyelash follicles in different populations. Arch Med Sci. 2014;10(2):319–24.
9. English FP, Nutting WB. Demodicosis of ophthalmic concern. Am J Ophthalmol. 1981;91(3):362–72.
10. Koo H, Kim TH, Kim KW, Wee SW, Chun YS, Kim JC. Ocular surface discomfort and Demodex: effect of tea tree oil eyelid scrub in Demodex blepharitis. J Korean Med Sci. 2012;27(12):1574–9.

11. Gao YY, Di Pasquale MA, Li W, et al. High prevalence of Demodex in eyelashes with cylindrical dandruff. Invest Ophthalmol Vis Sci. 2005;46(9):3089–94.

12. Nowomiejska K, Lukasik P, Brzozowska A, et al. Prevalence of ocular demodicosis and ocular surface conditions in patients selected for cataract surgery. J Clin Med. 2020;9(10):3069.

13. Amescua G, Akpek EK, Farid M, et al. Blepharitis preferred practice Pattern(R). Ophthalmology. 2019;126(1):P56–93.

14. Lemp MA, Nichols KK. Blepharitis in the United States 2009: a survey-based perspective on prevalence and treatment. Ocul Surf. 2009;7(2):S1–14.

15. Lalwani GA, Flynn HW Jr, Scott IU, et al. Acute-onset endophthalmitis after clear corneal cataract surgery (1996–2005). Clinical features, causative organisms, and visual acuity outcomes. Ophthalmology. 2008;115(3):473–6.

16. Nichols KK, Foulks GN, Bron AJ, et al. The international workshop on meibomian gland dysfunction: executive summary. Invest Ophthalmol Vis Sci. 2011;52(4):1922–8.

17. Cochener B, Cassan A, Omiel L. Prevalence of meibomian gland dysfunction at the time of cataract surgery. J Cataract Refract Surg. 2018;44(2):144–8.

18. Varu DM, Rhee MK, Akpek EK, et al. Conjunctivitis preferred practice Pattern(R). Ophthalmology. 2019;126(1):P94–169.

19. Singh K, Axelrod S, Bielory L. The epidemiology of ocular and nasal allergy in the United States, 1988–1994. J Allergy Clin Immunol. 2010;126(4):778–783.e776.

20. Boorstein SM, Henk HJ, Elner VM. Atopy: a patient-specific risk factor for diffuse lamellar keratitis. Ophthalmology. 2003;110(1):131–7.

21. Yang H-Y, Fujishima H, Toda I, et al. Allergic conjunctivitis as a risk factor for regression and haze after photorefractive keratotomy. Am J Ophthalmol. 1998;125(1):54–8.

22. Craig JP, Nelson JD, Azar DT, et al. TFOS DEWS II report executive summary. Ocul Surf. 2017;15(4):802–12.

23. Epidemiology Subcommittee of the International Dry Eye Workshop. The epidemiology of dry eye disease: report of the Epidemiology Subcommittee of the International Dry Eye Workshop (2007). Ocul Surf. 2007;5(2):93–107.

24. Weiss JS, Moller HU, Lisch W, et al. The IC3D classification of the corneal dystrophies. Cornea. 2008;27(Suppl 2):S1–83.

25. Waring GO, Rodrigues MM, Laibson PR. Corneal dystrophies. I. Dystrophies of the epithelium, Bowman’s layer and stroma. Surv Ophthalmol. 1978;23(2):71–122.

26. Lee WS, Lam CK, Manche EE. Phototherapeutic keratectomy for epithelial basement membrane dystrophy. Clin Ophthalmol. 2017;11:15–22.

27. Dastghieb KA, Clinch TE, Manche EE, Hersh P, Ramsey J. Sloughing of corneal epithelium and wound healing complications associated with laser in situ keratomileusis in patients with epithelial basement membrane dystrophy. Am J Ophthalmol. 2000;130(3):297–303.

28. 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(8):1119–23.

29. Farjo AA, Halperin GI, Syed N, Sutphin JE, Wagoner MD. Salzmann’s nodular corneal degeneration: clinical characteristics and surgical outcomes. Cornea. 2006;25:11–5.

30. Stone DU, Astley RA, Shaver RP, Chodosh J. Histopathology of Salzmann nodular corneal degeneration. Cornea. 2008;27:148–51.

31. Jones L, Downie LE, Korb D, et al. TFOS DEWS II management and therapy report. Ocul Surf. 2017;15(3):575–628.

32. Kim KY, Chung B, Kim EK, Seo KY, Jun I, Kim TI. Changes in ocular surface and meibomian gland after penetrating keratoplasty. BMC Ophthalmol. 2021;21(1):85.

33. De Paiva CS, Chen Z, Koch DD, et al. The incidence and risk factors for developing dry eye after myopic LASIK. Am J Ophthalmol. 2006;141(3):438–45.

34. Starr CE, Gupta PK, Farid M, et al. An algorithm for the preoperative diagnosis and treatment of ocular surface disorders. J Cataract Refract Surg. 2019;45(5):669–84.

35. Donnenfeld ED, Solomon K, Perry HD, et al. The effect of hinge position on corneal sensation and
36. Gollogly HE, Hodge DO, St Sauver JL, Erie JC. Increasing incidence of cataract surgery: population-based study. J Cataract Refract Surg. 2013;39(9):1383–9.

37. Chan E, Mahroo OA, Spalton Dj. Complications of cataract surgery. Clin Exp Optom. 2010;93(6):379–89.

38. Li X, Hu L, Hu J, Wang W. Investigation of dry eye disease and analysis of the pathogenic factors in patients after cataract surgery. Cornea. 2007;26:516–20.

39. Murakami Y, Manche EE. Prospective, randomized comparison of self-reported postoperative dry eye and visual fluctuation in LASIK and photorefractive keratectomy. Ophthalmology. 2012;119(11):2220–4.

40. Albietz JM, McLennan SG, Lenton LM. Ocular surface management of photorefractive keratectomy and laser in situ keratomileusis. J Refract Surg. 2003;19:636–44.

41. Nejima R, Miyata K, Tanabe T, et al. Corneal barrier function, tear film stability, and corneal sensation after photorefractive keratectomy and laser in situ keratomileusis. Am J Ophthalmol. 2005;139(1):64–71.

42. Monaco G, Scialdone A. Long-term outcomes of limbal relaxing incisions during cataract surgery: aberrometric analysis. Clin Ophthalmol. 2015;9:1581–7.

43. Hoffart L, Proust H, Matonti F, Conrath J, Ridings B. Correction of postkeratoplasty astigmatism by femtosecond laser compared with mechanized astigmatic keratotomy. Am J Ophthalmol. 2009;147(5):779–807 e771.

44. Ruckl T, Drexl AK, Bachernegg A, et al. Femtosecond laser-assisted intrastromal arcuate keratotomy to reduce corneal astigmatism. J Cataract Refract Surg. 2013;39(4):528–38.

45. Asbell PA, Maloney RK, Davidorf J, et al. Conductive keratoplasty for the correction of hyperopia. Trans Am Ophthalmol Soc. 2001;99:79–87.

46. Ehrlich JS, Manche EE. Regression of effect over long-term follow-up of conductive keratoplasty to correct mild to moderate hyperopia. J Cataract Refract Surg. 2009;35(9):1591–6.

47. Waring GO, Moffitt SD, Gelender H, et al. Rationale for and design of the National Eye Institute Prospective Evaluation of Radial Keratotomy (PERK) study. Ophthalmology. 1983;90(1):40–58.

48. Waring GO, Lynn MJ, Nizam A, et al. Results of the Prospective Evaluation of Radial Keratotomy (PERK) study five years after surgery. Ophthalmology. 1991;98(8):1164–76.

49. Lee SY, Wong TT, Chua J, Boo C, Soh YF, Tong L. Effect of chronic anti-glaucoma medications and trabeculectomy on tear osmolarity. Eye. 2013;27(10):1142–50.

50. Cvenkel B, Kopitar AN, Ihan A. Inflammatory molecules in aqueous humour and on ocular surface and glaucoma surgery outcome. Mediators Inflamm. 2010;2010:939602.

51. Frigo AC, Fasolo A, Capuzzo C, et al. Corneal transplantation activity over 7 years: changing trends for indications, patient demographics and surgical techniques from the Corneal Transplant Epidemiological Study (CORTES). Transplant Proc. 2015;47(2):528–35.

52. Errais K, Bouden J, Mill-Bousset I, Anane R, Beltaif O, Meddeh-Ouertani A. Effect of pterygium surgery on corneal topography. Eur J Ophthalmol. 2008;18(2):177–81.

53. Alpay A, Uğurbas SH, Erdoğan B. Comparing techniques for pterygium surgery. Clin Ophthalmol. 2009;3:69–74.

54. Shusko A, Schechter BA, Hovanesian JA. Pterygium surgery utilizing limbal conjunctival autograft and subconjunctival amniotic membrane graft in high-risk populations. Clin Ophthalmol. 2020;14:2087–90.

55. Sangwan VS, Burman S. Cataract surgery in Stevens–Johnson syndrome. J Cataract Refract Surg. 2005;31(4):860–2.

56. Neale RE, Purdie JL, Hirst LW, Green AC. Sun exposure as a risk factor for nuclear cataract. Epidemiology. 2003;14(6):707–12.

57. Gichuhi S, Macharia E, Kabiru J, et al. Risk factors for ocular surface squamous neoplasia in Kenya: a case–control study. Trop Med Int Health. 2016;21(12):1522–30.

58. Dave SB, Toma HS, Kim SJ. Ophthalmic antibiotic use and multidrug-resistant staphylococcus epidermidis: a controlled, longitudinal study. Ophthalmology. 2011;118(10):2035–40.

59. Foulks GN, Borchman D, Yappert M, Kim SH, McKay JW. Topical azithromycin therapy for meibomian gland dysfunction: clinical response and lipid alterations. Cornea. 2010;29(7):781–8.
60. Khalifa YM, Mifflin MD. Keratitis and corneal melt with ketorolac tromethamine after conductive keratoplasty. Cornea. 2011;30(4):477–8.

61. Cequa (cyclosporine ophthalmic solution) full prescribing information. Sun Pharmaceutical Industries, Inc., Princeton, NJ. 2019.

62. Restasis (cyclosporine ophthalmic emulsion) full prescribing information. Allergan, Madison, NJ. 2012.

63. Xiidra (lifitegrast ophthalmic solution) full prescribing information. Novartis Pharmaceuticals Corporation, East Hanover, NJ. 2020.

64. Macsai MS. The role of omega-3 dietary supplementation in blepharitis and meibomian gland dysfunction (an AOS thesis). Trans Am Ophthalmol Soc. 2008;106:336–56.

65. The Dry Eye Assessment and Management Study Research Group. n-3 fatty acid supplementation for the treatment of dry eye disease. N Engl J Med. 2018;378(18):1681–90.

66. United States Food and Drug Administration. Azithromycin (Zithromax or Zmax) and the risk of potentially fatal heart rhythms. 2013.

67. Yoo S, Lee D, Chang M. The effect of low-dose doxycycline therapy in chronic meibomian gland dysfunction. Korean J Ophthalmol. 2005;19(4):258–63.

68. Akkaya S, Atakan T, Acikalin B, Aksoy S, Ozkurt Y. Effects of long-term computer use on eye dryness. North Clin Istab. 2018;5(4):319–22.

69. Gao YY, Di Pascuale MA, Li W, et al. In vitro and in vivo killing of ocular Demodex by tea tree oil. Br J Ophthalmol. 2005;89(11):1468–73.

70. Walton SF, McKinnon M, Pizzutto S, Dougall A, Williams E, Currie BJ. Acaricidal activity of Melaleuca alternifolia (tea tree) oil: in vitro sensitivity of Sarcoptes scabiei var hominis to terpinen-4-ol. Arch Dermatol. 2004;140:563–6.

71. Stroman DW, Mintun K, Epstein AB, et al. Reduction in bacterial load using hypochlorous acid hygiene solution on ocular skin. Clin Ophthalmol. 2017;11:707–14.

72. Schneiderman H. The funduscopic examination. In: Walker HK, Hall WD, Hurst JW, editors. Clinical methods: the history, physical, and laboratory examinations. 3rd ed. Boston: Butterworth Publishers; 1990. p. 573–80.

73. Marsden J, Stevens S, Ebri A. How to measure distance visual acuity. Community Eye Health. 2014;27(85):16.

74. Damato BE. Oculokinetic perimetry: a simple visual field test for use in the community. Br J Ophthalmol. 1985;69:927–31.

75. National Institute for Health and Care Excellence Guideline (UK). Cataracts in adults: management. London; 2017.

76. Ambrosio R Jr, Caiado AL, Guerra FP, et al. Novel pachymetric parameters based on corneal tomography for diagnosing keratoconus. J Refract Surg. 2011;27(10):753–8.

77. Savini G, Huang J, Lombardo M, et al. Objective monitoring of corneal backward light scattering after femtosecond laser-assisted LASIK. J Refract Surg. 2016;32(1):20–5.

78. Igarashi A, Kamiya K, Kobashi H, Shimizu K. Effect of rebamipide ophthalmic suspension on intraocular light scattering for dry eye after corneal refractive surgery. Cornea. 2015;34:895–900.

79. Roszkowska AM, Urso M, Signorino GA, Spadea L, Aragona P. Photorefractive keratectomy after cataract surgery in uncommon cases: long-term results. Int J Ophthalmol. 2018;11(4):612–5.

80. Savini G, Barboni P, Ducoli P, Borrelli E, Hoffer KJ. Influence of intraocular lens haptic design on refractive error. J Cataract Refract Surg. 2014;40(9):1473–8.

81. Borasio E, Stevens J, Smith GT. Estimation of true corneal power after keratorefractive surgery in eyes requiring cataract surgery: BESSI formula. J Cataract Refract Surg. 2006;32(12):2004–14.

82. Albietz JM, Lenton LM, McLennan SG. Chronic dry eye and regression after laser in situ keratomileusis for myopia. J Cataract Refract Surg. 2004;30(3):675–84.