



# Unseen Consequences: The Expanding Burden of Iatrogenic Dry Eye Disease in Surgical and Cosmetic Practice

Arzu Taşkıran Çömez

University of Health Sciences Türkiye, İstanbul Prof. Dr. Cemil Taşcıoğlu City Hospital, Clinic of Ophthalmology, İstanbul, Türkiye

## Abstract

Iatrogenic dry eye disease (DED) has emerged as a significant but overlooked complication in both ophthalmic surgery and aesthetic medicine. Characterized by disruption of tear film homeostasis, ocular surface inflammation, and neurosensory dysfunction, this condition may arise acutely or chronically following medical interventions. The Tear Film and Ocular Surface Society Dry Eye Workshop II and III reports have highlighted iatrogenic DED as a distinct clinical entity, underscoring its multifactorial etiology and the need for tailored risk mitigation strategies. Refractive procedures such as laser in situ keratomileusis and photorefractive keratectomy are frequently implicated due to corneal nerve transection and subsequent hypoesthesia, which impair blink dynamics and lacrimal gland feedback. Cataract surgery, though largely safe, contributes via surface desiccation, intraoperative light exposure, and topical medication toxicity. Oculoplastic interventions, including blepharoplasty, introduce mechanical alterations that disrupt eyelid function and tear film distribution. Moreover, the growing demand for periocular cosmetic procedures such as botulinum toxin injections and permanent eyeliner tattooing has introduced additional risks, particularly in individuals with preexisting meibomian gland dysfunction or marginal tear function. Emerging evidence suggests that the cumulative effect of repeated exposures and inadequate preoperative screening has

led to a rise in persistent ocular surface morbidity. Prevention therefore necessitates a multidisciplinary approach encompassing ophthalmologists, dermatologists, plastic surgeons, and primary care providers. Systematic pretreatment ocular surface assessment, patient counseling, and adoption of minimally disruptive techniques are essential. By integrating evidence-based risk stratification into routine care, clinicians can significantly reduce the burden of iatrogenic DED and preserve long-term visual quality and patient satisfaction in the face of expanding therapeutic and cosmetic practices.

**Keywords:** Iatrogenic dry eye, ocular surface disease, refractive surgery, cataract surgery, blepharoplasty, botulinum toxin, cosmetic procedures, eyelid surgery

## Introduction

Dry eye disease (DED) is a multifactorial and complex condition involving disruption of ocular surface homeostasis, characterized by tear film instability, hyperosmolarity, inflammation, and neurosensory dysfunction. As defined in the Tear Film and Ocular Surface Society Dry Eye Workshop (TFOS DEWS) II report by Craig et al.,<sup>1</sup> DED results from the loss of tear film homeostasis, presenting clinically as ocular discomfort, fluctuating vision, and signs of inflammation. Among its subtypes, iatrogenic DED remains underappreciated and refers specifically to tear film dysfunction and symptoms induced by medical or surgical interventions.

The DEWS III update expands upon this concept by incorporating recent data on epidemiology, diagnostic tools, and pathophysiologic mechanisms, emphasizing the increasing burden of iatrogenic triggers in routine practice.<sup>2</sup> Common contributors include anterior segment procedures (e.g., phacoemulsification, corneal refractive surgeries such as laser in situ keratomileusis [LASIK] and photorefractive keratectomy [PRK]), eyelid and periocular

**Cite this article as:** Taşkıran Çömez A. Unseen Consequences: The Expanding Burden of Iatrogenic Dry Eye Disease in Surgical and Cosmetic Practice. *Turk J Ophthalmol.* 2026;56:187-197

**Address for Correspondence:** Arzu Taşkıran Çömez, University of Health Sciences Türkiye, İstanbul Prof. Dr. Cemil Taşcıoğlu City Hospital, Clinic of Ophthalmology, İstanbul, Türkiye

**E-mail:** arzucomez@yahoo.com

**ORCID-ID:** orcid.org/0000-0001-8048-8146

**Received:** 04.08.2025

**Revision Requested:** 08.10.2025

**Last Revision Received:** 26.11.2025

**Accepted:** 06.12.2025

**Publication Date:** 24.06.2026

**DOI:** 10.4274/tjo.galenos.2025.69812



aesthetic surgeries (e.g., blepharoplasty), botulinum toxin (BoNT) injections, chronic use of preserved eye drops, and contact lens wear. Pharmacologic agents such as systemic retinoids, hormonal therapies, and antidepressants have also been linked to meibomian gland dysfunction (MGD) and aqueous deficiency.<sup>3,4</sup>

Importantly, iatrogenic DED is now understood not only as an acute postoperative complication but also as a chronic, often subclinical condition resulting from repeated or prolonged therapeutic exposure. Both intraoperative factors (e.g., corneal nerve disruption) and postoperative mechanisms (e.g., inflammation, tear film destabilization) contribute to its development.<sup>5</sup>

The DEWS III consensus strongly encourages the integration of iatrogenic risk profiling into clinical workflows, especially for surgical patients and those undergoing aesthetic procedures.<sup>2</sup> Risk is notably elevated in individuals with preexisting ocular surface instability, autoimmune diseases, and in older adults.<sup>6</sup> Among all causes, surgical interventions remain leading contributors, particularly anterior segment and oculoplastic procedures, due to their mechanical, neurotrophic, and anatomical impact on the ocular surface. This review aims to synthesize current understanding of iatrogenic DED by integrating DEWS frameworks with updated clinical evidence, outlining mechanisms, risk factors, and preventive strategies.

## Methods

This narrative review synthesized peer-reviewed evidence on iatrogenic DED across surgical, cosmetic, pharmacologic, and device-related contexts. The author searched PubMed/MEDLINE, Embase, Scopus, and the Cochrane Library from January 1, 2000 to August 31, 2025, with no filter applied for study design at this stage. Core search strings combined terms and keywords related to dry eye and iatrogenic triggers, for example: (*dry eye* OR *ocular surface* OR *meibomian gland dysfunction*) AND (*refractive* OR *LASIK* OR *PRK* OR *SMILE* OR *cataract* OR *femtosecond* OR *blepharoplasty* OR *oculoplastic* OR *botulinum* OR *eyeliner tattoo* OR *contact lens* OR *preservative* OR *benzalkonium* OR *glaucoma drops* OR *isotretinoin* OR *intravitreal*). Searches were limited to English and Turkish literature.

Inclusion criteria: original clinical studies (randomized/non-randomized), cohort/case-control studies, large case series, systematic reviews, meta-analyses, consensus statements/guidelines (e.g., TFOS DEWS reports), and mechanistic or imaging studies with direct relevance to iatrogenic DED.

Exclusion criteria: non-peer-reviewed content, single-patient case reports unless clinically instructive for

mechanisms, conference abstracts without full text, non-ocular surface endpoints, and duplicate publications.

The author screened titles/abstracts and reviewed full texts, as well as performed backward citation tracking of included articles and key consensus documents to identify additional eligible studies. Given the narrative scope, a qualitative appraisal focusing on study design, sample size, outcome definitions (e.g., Ocular Surface Disease Index [OSDI], tear break-up time [TBUT], Schirmer test, staining), and generalizability was conducted rather than a formal risk-of-bias meta-analysis.

## Surgical Contributors to Iatrogenic Dry Eye Disease

### Refractive Surgery

Refractive procedures, particularly LASIK and PRK, are well-established contributors to iatrogenic DED through their direct impact on corneal innervation. These surgeries often involve transection of subbasal nerve fibers, resulting in reduced corneal sensitivity and impaired reflex tear production. As demonstrated by Wilson and Ambrósio,<sup>7</sup> this post-LASIK corneal hypoesthesia can persist for as long as six months and contributes to both evaporative and aqueous-deficient subtypes of DED.

During the early postoperative period, transient dry eye symptoms are nearly universal. According to D'Souza et al.,<sup>8</sup> this occurs primarily due to corneal nerve transection, which leads to decreased stimulation of the lacrimal gland and reduced blink reflex. While PRK tends to damage the subbasal nerve plexus, LASIK often affects deeper stromal nerves, with both contributing to altered ocular surface homeostasis.<sup>9</sup>

The TFOS DEWS II Iatrogenic Report provides additional insight into these mechanisms. Gomes et al.<sup>10</sup> introduced the concept of “neurosensory block” to describe how corneal nerve injury impairs afferent signaling pathways essential for tear secretion and blink regulation. Preserved topical medications, particularly those containing benzalkonium chloride (BAK), can further destabilize the tear film by damaging epithelial microvilli and depleting goblet cells.<sup>10</sup>

*In vivo* confocal microscopy studies confirm that refractive surgery results in corneal nerve fiber transection, and follow-up imaging shows that nerve regeneration can take between 3 to 6 months, and even up to a year, to return to baseline status.<sup>11,12</sup> Identified surgical risk factors include flap configuration (e.g., hinge position, diameter, and thickness), size of the ablation zone, female sex, smoking, existing ocular surface disorders, and environmental exposures.<sup>13</sup>

Turu et al.<sup>14</sup> highlighted that LASIK-induced dry eye stems from both neurogenic and inflammatory mechanisms. Neurogenic elements involve corneal denervation, reduced sensory feedback, decreased tear secretion, and increased evaporation, while inflammatory factors involve cytokine-mediated epithelial injury. Confocal imaging has shown that stromal nerve density may decrease by up to 90% after LASIK, with only partial recovery at 1-year follow-up.<sup>14</sup>

D'Souza et al.<sup>8</sup> proposed a structured diagnostic and therapeutic algorithm for managing dry eye following refractive surgery. They emphasized that early identification of high-risk patients is key to reducing postoperative complications. Preoperative assessments should include objective measures such as Schirmer test, TBUT, and evaluation of ocular surface integrity and meibomian gland function. Subjective screening tools such as the OSDI, Dry Eye Questionnaire-5, and Impact of Dry Eye on Everyday Living are also valuable in detecting underlying dry eye before surgery.<sup>15</sup> In cases with subclinical signs of dry eye or lid margin abnormalities, postponing surgery and initiating ocular surface optimization therapies may improve long-term outcomes.

### Small Incision Lenticule Extraction

Small Incision Lenticule Extraction (SMILE) is considered a significant technological progression in the field of corneal refractive surgery, offering high refractive accuracy while inducing less damage to corneal nerves when compared with traditional LASIK procedures.<sup>16</sup> In contrast to LASIK, which requires the creation of a corneal flap, SMILE involves the use of a femtosecond laser to sculpt an intrastromal lenticule that is extracted through a small peripheral incision. This technique leaves the anterior corneal architecture—including Bowman's layer and the subbasal nerve plexus—largely undisturbed, thereby helping preserve corneal sensitivity and tear film function and reducing the incidence of postoperative dry eye symptoms.<sup>17</sup>

Comparative studies analyzing the outcomes of SMILE and femtosecond LASIK have consistently shown similar levels of refractive correction. However, SMILE has demonstrated superior outcomes in terms of tear film stability and neurosensory recovery. Patients undergoing SMILE tend to experience faster improvement in dry eye symptoms, which often return to preoperative baseline by six months. In contrast, LASIK-treated patients frequently exhibit longer-lasting symptoms beyond this recovery window, likely due to greater corneal nerve disruption.<sup>18,19</sup>

Nonetheless, SMILE is not entirely exempt from iatrogenic effects. Some degree of subbasal nerve interruption occurs during lenticule dissection, which

can transiently impair ocular surface function. Dry eye symptoms, although less frequent and severe than in LASIK, may still develop postoperatively, especially in patients with preexisting tear film instability or MGD.<sup>20,21</sup> Despite this, the relatively nerve-sparing nature of SMILE makes it a preferred option for patients considered at elevated risk for postoperative DED.

Evidence from D'Souza et al.,<sup>8</sup> Gomes et al.,<sup>10</sup> Turu et al.,<sup>14</sup> and studies specific to SMILE have led to a more nuanced understanding of post-refractive surgery dry eye that recognizes the multifactorial and evolving nature of the condition.<sup>15,16,18</sup>

### Cataract Surgery

Although cataract surgery is generally regarded as a highly safe and effective procedure, it remains a prominent cause of both transient and persistent postoperative DED. In a broad narrative review by Mencucci et al.<sup>22</sup> representing the PICASSO Board, it was reported that up to 34% of patients without a prior history of ocular surface disease developed dry eye symptoms within the first 1 to 3 months following phacoemulsification surgery. Contributing factors include prolonged exposure to the intense illumination of the surgical microscope, ocular surface toxicity from preserved topical medications, and intraoperative desiccation of the cornea. Supporting these findings, Han et al.<sup>23</sup> and other authors demonstrated significant postoperative reductions in TBUT and Schirmer test scores, particularly in patients with marginal preoperative ocular surface parameters.<sup>22,24</sup>

Complementing this, Ishrat et al.<sup>25</sup> found that dry eye was more common after small-incision cataract surgery compared to standard phacoemulsification. They attributed this to increased tear film instability, reporting statistically significant differences in TBUT at 1 week, 1 month, and 3 months postoperatively.

Mencucci et al.<sup>22</sup> also detailed several intraoperative factors that exacerbate dry eye risk. These include the use of lid speculums that distort lid-globe congruity, the direct neuroepithelial trauma caused by corneal incisions, and the phototoxicity of microscope illumination, which reduces goblet cell density and promotes pro-inflammatory cytokine release. Larger and grooved incisions were particularly implicated in delayed epithelial healing and impaired nerve regeneration. Moreover, commonly used topical anesthetics and mydriatics, especially those preserved with BAK, have been shown to induce epithelial cell apoptosis, disrupt mucin production, and create a pro-inflammatory microenvironment even before the surgical procedure begins.<sup>26,27,28,29</sup>

Further evidence provided by Mencucci et al.<sup>22</sup> suggests that femtosecond laser-assisted cataract surgery (FLACS) may be associated with an even higher risk of postoperative

dry eye than conventional phacoemulsification. The longer duration of the procedure, elevated laser energy exposure, and the use of suction rings may collectively contribute to inflammatory edema, goblet cell dysfunction, and further destabilization of the tear film.<sup>22,30</sup>

One of the key conceptual contributions of the PICASSO Board was the proposal of the “3+2” pathophysiological model, which outlines three central mechanisms—tear film instability, epithelial damage, and ocular surface inflammation—alongside two aggravating factors: lid margin dysfunction and neurosensory impairment. This model serves as a framework for comprehensive diagnostic and therapeutic strategies that span preoperative, intraoperative, and postoperative phases, with the ultimate aim of preserving or restoring ocular surface homeostasis during cataract surgery.<sup>22</sup>

Wolffsohn et al.<sup>4</sup> also emphasized that thorough preoperative screening is essential for reducing the risk of postoperative complications. Recommended assessments include TBUT, OSDI scores, fluorescein staining, meibography, and point-of-care tests such as tear osmolarity and matrix metalloproteinase-9 measurements.<sup>31</sup>

These recommendations apply to both preoperative optimization and postoperative management. Preservative-free artificial tears, eyelid hygiene, warm compresses, and omega-3 fatty acid supplementation are appropriate in both periods to stabilize the tear film and address MGD. Topical anti-inflammatories should be used postoperatively (short courses of corticosteroids). When feasible, anti-inflammatory treatment should also be initiated preoperatively with immunomodulators (cyclosporine or lifitegrast) to reduce baseline inflammation and continued after surgery.<sup>32</sup> In refractory cases, punctal occlusion is considered after ocular surface inflammation is controlled, and pulsed light therapy is generally reserved for chronic/refractory MGD outside the immediate perioperative window.<sup>4,20,30,32</sup>

### Oculoplastic Surgeries

Oculoplastic procedures, most notably blepharoplasty, can disrupt lid-globe congruity, impair blink dynamics, and adversely affect meibomian gland performance. Normal tear homeostasis requires coordinated aqueous production, blink-mediated distribution, and unobstructed drainage. Therefore, surgical alterations that weaken orbicularis function, modify eyelid position, or disturb lacrimal outflow can destabilize the tear film and precipitate ocular surface symptoms.<sup>33,34,35,36,37,38,39</sup> Blepharoplasty may alter eyelid positioning and compromise blink strength, while resection of the orbicularis oculi can induce scarring and neural injury, leading to incomplete blinking, reduced

blink frequency, and lagophthalmos. These changes impair meibomian lipid secretion and further destabilize the tear film.<sup>10,35,40,41</sup> Zhang et al.<sup>42</sup> showed that even subtle trauma to the orbicularis during cosmetic blepharoplasty can inhibit blink-induced meibum expression, reduce lipid layer thickness, and destabilize the ocular surface.

Prospective data demonstrate transient but measurable postoperative tear film changes. Sanad et al.<sup>33</sup> reported significant declines in tear meniscus height and TBUT that typically returned to baseline by six months. In a large retrospective cohort (n=892), Prischmann et al.<sup>34</sup> noted dry eye complaints in ~26.5%, with higher risk when upper and lower lids were operated together versus single-lid procedures. As patient factors (age, sex, baseline tear status) and technique modulate symptom severity and duration, Hamawy et al.<sup>35</sup> recommended careful preoperative risk stratification and conservative skin excision. In patients with known DED, Saadat and Dresner<sup>37</sup> observed postoperative worsening in 8% and no change in 83% when anatomic structures were preserved, suggesting blepharoplasty can be performed with minimal risk in properly selected cases.

Early postoperative perturbations are typically self-limited. Shao et al.<sup>38</sup> noted that OSDI and tear meniscus height were increased and Schirmer scores were reduced at week 1 after lower blepharoplasty and normalized by month 3. Using anterior-segment optical coherence tomography, they also documented a decreased cornea-lower lid angle and increased lower lid margin reflex distance at week 1. Both of these findings approached baseline by month 3, paralleling symptom resolution (dryness, epiphora, chemosis).<sup>38</sup>

Comparative studies contextualize the risk of postoperative DED. Aksu Ceylan and Yeniad<sup>36</sup> found greater decline in Schirmer test scores after blepharoplasty than after levator resection, attributing differences to orbicularis weakening with downstream effects on blink reflex and corneal sensitivity. Other eyelid surgeries such as ptosis repair and ectropion correction can also alter blink kinematics and tear physiology. In a series by Zhang et al.,<sup>43</sup> nearly 25% of patients exhibited postoperative blink pattern changes, most often those with preexisting lagophthalmos or facial nerve dysfunction, underscoring the role of eyelid closure in tear distribution. Full-thickness eyelid reconstruction data show that despite Schirmer and TBUT values comparable to controls, patients frequently report ocular discomfort, highlighting the importance of structural stability for tear film homeostasis.<sup>44</sup> In a cohort of 63 upper blepharoplasties, Mian et al.<sup>45</sup> observed reductions in Standard Patient Evaluation of Eye Dryness scores at 1 month and 1 year in both orbicularis-sparing and orbicularis-excising techniques, with no significant difference between approaches.

Lacrimal drainage pathology can also amplify the DED symptom burden. In patients with nasolacrimal duct obstruction and dry eye symptoms, silicone stent implantation achieved a 76.7% surgical success rate with significant improvements in Glasgow Benefit Inventory, particularly in general and social well-being.<sup>46</sup> These findings suggest that treating epiphora can enhance subjective comfort even without large changes in physical health scores. When lower lid retraction or malposition is anticipated, canthopexy or lateral canthal suspension provides needed support and reduces scleral show. Long-term series report lower malposition rates and improved ocular surface outcomes when using this approach.<sup>47</sup> During upper blepharoplasty, vigilance for lacrimal gland prolapse is essential, as unrecognized or traumatized gland tissue may depress aqueous production and worsen postoperative dryness, particularly in older patients or those with prior eyelid surgery.<sup>48</sup> Preventive measures include preoperative identification of high-risk patients, meticulous tissue-sparing technique to preserve eyelid mechanics and orbicularis integrity, and postoperative anti-inflammatory therapy with frequent lubrication. Adjunct lid margin therapies are advisable when MGD coexists.<sup>10,35,40,41</sup>

### Cosmetic and Aesthetic Interventions

The growing popularity of periocular cosmetic procedures has introduced additional risk factors for iatrogenic DED. Treatments such as BoNT-A injections, permanent eyeliner tattooing, and eyelash extensions (EEs) are increasingly associated with adverse effects on tear film integrity, blink dynamics, and meibomian gland function.

Kocabeyoglu et al.<sup>49</sup> evaluated ocular surface changes in patients with blepharospasm receiving periocular BoNT-A injections. They reported that ocular surface test results improved 2 weeks after injection, started to deteriorate after 3 months, and almost returned to baseline after 6 months. In parallel with improvements in objective test results, subjective complaints also decreased within 2 weeks and increased between 3 and 6 months postinjection. The authors concluded that BoNT-A may have a temporary beneficial effect on ocular surface parameters in patients with blepharospasm, but the effect appears transient and may not apply universally.<sup>49</sup>

Despite some therapeutic benefit, the TFOS DEWS III report emphasized the potential iatrogenic consequences of cosmetic BoNT-A injections, particularly when administered near the lateral canthal area.<sup>2</sup> The toxin may interfere with blink force and amplitude due to its chemodenervation of the orbicularis oculi muscle, leading to lagophthalmos, incomplete blinking, or lower lid laxity, factors that can increase evaporative stress and destabilize the tear film.<sup>6,50</sup>

EEs represent another aesthetic modality with documented ocular surface implications. In a prospective study, Grupcheva et al.<sup>51</sup> demonstrated that the removal of EEs resulted in statistically significant improvements in both subjective symptoms and objective findings. Following EE removal, OSDI scores decreased from 33.4 to 26.7, TBUT increased from 11.25 to 13.96 seconds, and both blink frequency and corneal staining improved. These findings suggest that EEs may contribute to ocular surface instability by interfering with lid hygiene, reducing blink efficiency, and impairing meibomian gland expression. Importantly, discontinuing EEs led to measurable recovery in tear film stability and ocular comfort.

Consistent with these observations, Masud et al.<sup>52</sup> found that long-term use of EEs correlated with higher rates of meibomian gland dropout, lid margin irregularities, and diminished tear meniscus height when compared to control subjects. They attributed these effects to the combined mechanical and chemical irritation from repeated application, particularly due to cyanoacrylate-based adhesives. These changes were particularly detrimental in individuals with preexisting MGD, amplifying tear film instability and ocular surface inflammation.

Lee et al.<sup>53</sup> provided a detailed review of ocular complications arising from cosmetic eyelid tattooing, with a specific focus on permanent eyeliner tattoos. They reported a range of short- and long-term complications, including allergic reactions, eyelid edema, conjunctivitis, pigment migration, granuloma formation, and structural disruption of the meibomian gland orifices. The authors highlighted the need for stricter procedural guidelines and practitioner training, noting that tattoo placement too close to the mucocutaneous junction can result in permanent meibomian gland damage and chronic ocular surface disease.

These concerns were reflected in the TFOS DEWS III report by Stapleton et al.,<sup>2</sup> who included aesthetic procedures among the recognized contributors to iatrogenic DED. They advocated for pre-procedural ocular surface screening and routine postoperative follow-up for patients undergoing cosmetic interventions. Moreover, the report recommended interdisciplinary collaboration between dermatologists, plastic surgeons, and ophthalmologists to minimize ocular surface complications and ensure informed consent.

Taken together, these findings underscore that cosmetic procedures, despite being minimally invasive, can produce lasting disruptions in ocular surface homeostasis. Proper patient selection, clinician awareness, and preventive strategies are necessary to mitigate these risks.

### Pharmacologic and Device-Related Triggers

Pharmacologic agents, particularly those administered topically in ophthalmology, represent a well-established category of contributors to iatrogenic DED. Among these, preservatives (especially BAK) have been studied extensively for their cytotoxic effects on the ocular surface.<sup>3</sup>

Beyond BAK, other excipients such as thiomersal and ethylenediaminetetraacetic acid may also contribute to ocular surface toxicity, especially in individuals with underlying dry eye conditions or known allergic sensitivities. The chronic administration of topical antiglaucoma medications poses particular concern in this regard. According to the findings of Mocan et al.,<sup>54</sup> patients receiving long-term treatment with prostaglandin analogs and beta-adrenergic blockers often present with signs of conjunctival hyperemia, punctate epithelial erosions, and MGD, features that collectively promote tear film instability and ocular surface disease.<sup>55,56</sup>

Systemic medications also contribute significantly to iatrogenic DED. Isotretinoin, a systemic retinoid commonly prescribed in dermatology, has been shown to induce meibomian gland atrophy, resulting in a marked reduction in lipid layer secretion. In parallel, systemic agents such as antihistamines, tricyclic antidepressants, selective serotonin reuptake inhibitors, and beta-blockers have been implicated in reduced aqueous tear production, further exacerbating tear film instability and evaporative stress in predisposed individuals.<sup>4,57,58</sup>

Furthermore, chemotherapeutic agents, particularly those targeting epidermal growth factor receptors, have been associated with direct injury to the ocular surface. These agents can deplete goblet cells, increase ocular surface inflammation, and impair epithelial regeneration, contributing to a cascade of tear film dysfunction and chronic ocular surface compromise.<sup>59</sup>

Contact lens use represents another prevalent and significant contributor to DED.<sup>10,60</sup> Estimates indicate that approximately half of soft contact lens users report symptoms consistent with contact lens-related dryness.<sup>61</sup> In a study conducted in Japan, Koh et al.<sup>62</sup> found that over 70% of individuals using soft contact lenses experienced ocular dryness.

Clinical awareness of contact lens discomfort has increased substantially since the publication of the TFOS DEWS II Contact Lens Discomfort Report.<sup>63,64</sup> The primary pathophysiologic mechanisms implicated in contact lens discomfort are inadequate tear distribution and increased mechanical interaction between the lens and ocular surface structures, leading to frictional damage.<sup>65</sup>

Prolonged contact lens wear (especially of rigid gas-permeable and soft contact lenses) has been associated with microtrauma to the ocular surface and disruption of tear film dynamics, including reduced tear exchange and compromised lipid spreading.<sup>66,67</sup> Tear film changes associated with soft contact lens usage include accelerated tear evaporation, reduced tear volume, instability of the pre-lens lipid layer, and compositional shifts in tear film content.<sup>68</sup>

Two reviews by Efron<sup>69,70</sup> highlighted both overt and subclinical inflammation associated with contact lens wear. They documented evidence of inflammatory responses even in users of modern lens materials, such as hydrogel and silicone hydrogel lenses, under routine conditions. Contact lens discomfort is a major reason for discontinuation of contact lens wear.<sup>71</sup>

Another often-overlooked source of iatrogenic DED is repeated intravitreal injection therapy. In a prospective study by Srinagesh et al.,<sup>72</sup> involving 12 patients undergoing multiple intravitreal anti-vascular endothelial growth factor injections, cumulative procedural exposure was associated with worsening ocular surface parameters, presumably due to repeated mechanical trauma and the inflammatory effects of antiseptics used perioperatively. Additionally, frequent use of topical antibiotics during the peri-injection period may exacerbate epithelial damage through direct cytotoxicity.<sup>73,74</sup>

Special consideration must also be given to patients receiving keratoprosthesis implants. In their retrospective study of patients with Boston Type I keratoprostheses, Zhang et al.<sup>75</sup> reported that aggressive management of the ocular surface (including intensive lubrication, punctal occlusion, and ongoing anti-inflammatory treatment) was essential to ensure prosthesis retention and prevent sterile keratolysis.

### Management and Preventative Strategies

The effective prevention and long-term management of iatrogenic DED should follow an etiology-directed, stepwise algorithm that spans preoperative optimization, intraoperative protection, and severity-tiered postoperative care, along with continuous patient education. Stapleton et al.<sup>2</sup> emphasized within the TFOS DEWS III framework that routine ocular surface assessment (symptoms, TBUT, staining, meibography, osmolarity, validated questionnaires) should be integrated into preoperative planning for cataract, refractive, and oculoplastic candidates to identify subclinical instability early. In parallel, the recent TFOS DEWS III: Management and Therapy report synthesized first-line measures (replenish, conserve, stimulate the tear film), targeted meibomian gland interventions, anti-

inflammatory strategies, and advanced therapies within an evidence-based prescribing algorithm.<sup>76</sup> The interventions involved at each stage of this algorithm are outlined below.

**Preoperative Optimization (Risk Modification):**

Address modifiable drivers, especially MGD, via lid hygiene education, regular warm compresses, and lid margin care. When feasible, modify systemic or topical regimens known to cause ocular surface toxicity. Lipid-enriched artificial tears and nutritional support (e.g., omega-3/omega-6) can improve lipid layer quality and symptom control.<sup>77</sup> For eyes with clinically significant inflammation or epithelial compromise, start targeted anti-inflammatory therapy to improve surgical readiness and recovery. Cyclosporine A or lifitegrast may be used to reduce T-cell-mediated inflammation and restore homeostasis.<sup>6,78</sup>

**Intraoperative Protection (Iatrogenic Stress Minimization):** Limit microscope light exposure and surface desiccation. Use preservative-free solutions when possible, and provide frequent non-preserved lubrication (or a moisture chamber) during longer cases. Technique choices that preserve corneal innervation and eyelid-globe congruity (e.g., thoughtful incision architecture, flap/ablation parameters, and preservation of orbicularis/eyelid position in oculoplastic procedures) reduce postoperative evaporative burden.

**Postoperative Care (Severity-Tiered Management):**

Initiate preservative-free artificial tears promptly. Consider short, judicious corticosteroid courses for inflammatory phenotypes. Escalate when needed to punctal occlusion or autologous serum/platelet-rich plasma in refractory disease. Continue sustained lid-margin therapy for MGD-predominant phenotypes and consider in-office MGD procedures (thermal pulsation, meibum expression) where appropriate. Depending on etiology and severity, the TFOS DEWS III algorithm also recognizes the benefit of integrating adjunctive methods such as intense pulsed light, low-level light therapy, neuromodulation (nasal neurostimulation), scleral lenses for severe exposure/evaporative cases, and amniotic membrane in advanced epithelial disease.<sup>76</sup>

**Patient Education (Cross-Cutting):** Reinforce blink training, screen ergonomics, ambient humidity control, proper drop instillation, and adherence, which are the core tenets highlighted by TFOS DEWS III to sustain outcomes and reduce chronicity.<sup>76</sup>

**Refractive Surgery (LASIK/PRK/SMILE):** Screen preoperatively for borderline tear function and lid margin disease. Defer elective procedures when the ocular surface is unstable and optimize first. Technique selection that

preserves innervation and prudent flap/ablation parameters support faster neurosensory recovery. Early postoperative lubrication is universal. Manage persistent neuropathic-inflammatory phenotypes with anti-inflammatory therapy and surface rehabilitation.

**Cataract Surgery (Including FLACS):** Conduct preoperative optimization of the ocular surface and avoid preserved perioperative drops when feasible (or switch to preservative-free/fixed combinations). Ensure intraoperative lubrication and minimize light exposure. Postoperatively, preservative-free tears plus short anti-inflammatory courses are first-line. Escalate in moderate-severe cases (e.g., punctal occlusion) while continuing MGD care.

**Oculoplastic Procedures (e.g., Blepharoplasty, Ptosis Repair, Eyelid Reconstruction):** Pre-select patients with fragile tear film or preexisting DED. Utilize surgical techniques that preserve orbicularis continuity, blink amplitude, and lid position. Provide intra-/postoperative edema control and frequent lubrication. Where lower lid malposition or retraction is anticipated, supportive canthal measures reduce scleral show and exposure. Vigilance for lacrimal gland prolapse is essential; inadvertent trauma risks aqueous deficiency.

**Cosmetic Interventions (BoNT-A, EEs, Permanent Eyeliner):** Ensure prior ocular surface screening and informed consent about DED risk. For BoNT-A, make dose/location choices that avoid excessive blink weakening. Monitor for transient changes and support with lubrication and lid margin care. For EEs, counsel on lid hygiene and consider discontinuation if they exacerbate symptoms. Eyelid tattooing should avoid the mucocutaneous junction to protect the meibomian gland orifices. Manage resultant lid margin inflammation promptly.

**Chronic Topical Therapy (Especially Glaucoma):** Prefer preservative-free or fixed-combination formulations to reduce cumulative BAK exposure. Monitor for signs of meibomian dysfunction and epithelial toxicity, and co-manage with surface-stabilizing therapy.

**Contact Lens Wear and Procedure-Heavy Care Pathways (e.g., Repeated Intravitreal Injections, Keratoprosthesis):** Recognize frictional/mechanical and inflammatory mechanisms. Optimize lens fit/wearing schedule and treat contact lens-related DED per established guidance. Around injection pathways, minimize epithelial insult from antiseptics/antibiotics and provide surface support. After keratoprosthesis, intensive lubrication, punctal occlusion, and sustained anti-inflammatory therapy may be necessary for device retention and surface integrity.

## Discussion

Iatrogenic DED has become increasingly recognized as a substantial contributor to ocular surface dysfunction in the context of modern surgical techniques and pharmacological treatments. The multifaceted nature of its pathophysiology illustrates the inherent challenges in both prevention and therapeutic management. As substantiated by numerous prospective and retrospective investigations, the ocular surface demonstrates increased vulnerability to interventions that disturb anatomical integrity, impair neurosensory pathways, or trigger inflammatory responses.<sup>7,22,25,77</sup>

Epitropoulos<sup>78</sup> emphasized that refractive surgeries frequently precipitate the transition of patients from a subclinical status to overtly symptomatic DED following the procedure, especially among individuals with preexisting borderline tear function or unrecognized MGD.

Although cataract surgery is generally regarded as a safe and effective intervention, it also constitutes a common iatrogenic cause of DED. Importantly, there are studies reporting a significant association between MGD and cataract surgery, with asymptomatic MGD found to be twice as prevalent as symptomatic cases.<sup>21,79</sup>

Oculoplastic surgeries introduce a distinct set of challenges due to their direct alteration of eyelid architecture and blink dynamics.<sup>80,81</sup> Even minor modifications to eyelid position, blink amplitude, or orbicularis oculi muscle function can severely disrupt the eyelid's role in tear film distribution and ocular surface protection. The essential interdependence of eyelid mechanics, lacrimal gland secretion, and blinking function is prominently underscored in the TFOS DEWS III report and further corroborated by recent surgical outcome analyses. These studies consistently reveal that inadequate preservation of eyelid contour or orbicularis muscle continuity is closely linked to increased severity of dry eye symptoms in the postoperative period.<sup>2,33,42</sup>

This complex relationship is further exacerbated by the rising popularity of cosmetic procedures in the periocular region. As patient demand for such interventions continues to increase, the literature documenting their frequently underestimated negative consequences on the ocular surface environment is also increasing.<sup>52,82,83</sup>

Among these procedures, BoNT injections can impair blink completeness and frequency, thereby accelerating tear evaporation and compromising uniform tear distribution. This mechanism is particularly pertinent in elderly populations, in whom baseline tear production may already be marginal.<sup>50</sup> Similarly, cosmetic practices such as

permanent eyeliner tattooing can provoke inflammation of the lid margin and worsen preexisting MGD, especially when performed without prior evaluation of ocular surface health.<sup>53</sup> Masud et al.<sup>52</sup> emphasized that healthcare providers must be aware of the potential adverse outcomes associated with these cosmetic enhancements, not only due to their capacity to damage the ocular surface but also because of their broader implications for visual function and postoperative satisfaction.

Given the expanding and evolving understanding of iatrogenic DED, it is evident that reducing its prevalence and severity demands more than only recognition. It requires the proactive implementation of comprehensive prevention strategies.

## Conclusion

Iatrogenic DED represents a growing and underappreciated clinical challenge across ophthalmology and aesthetic medicine. Its etiology is inherently multifactorial, originating from surgical injury, pharmacologic toxicity, and cosmetic manipulation, and its effect on patient quality of life can be profound.

Fortunately, with the structured guidance provided by the TFOS DEWS II and III consensus frameworks, clinicians are now better equipped to recognize, prevent, and manage iatrogenic DED. Risk stratification, proactive screening, and tailored perioperative strategies are the cornerstones of effective care. Multidisciplinary collaboration among ophthalmologists, oculoplastic surgeons, dermatologists, plastic surgeons, and other care providers will be essential to alleviating long-term complications. By integrating evidence-based preventive strategies into standard practice, we can significantly reduce the incidence and impact of iatrogenic DED.

## Declarations

**Conflict of Interest:** No conflict of interest was declared by the author.

**Financial Disclosure:** The author declared that this study received no financial support.

## References

1. Craig JP, Nichols KK, Akpek EK, Caffery B, Dua HS, Joo CK, Liu Z, Nelson JD, Nichols JJ, Tsubota K, Stapleton F. TFOS DEWS II definition and classification report. *Ocul Surf.* 2017;15:276-283.
2. Stapleton F, Argüeso P, Asbell P, Azar D, Bosworth C, Chen W, Ciolino JB, Craig JP, Gallar J, Galor A, Gomes JAP, Jalbert I, Jie Y, Jones L, Konomi K, Liu Y, Merayo-Llodes J, Oliveira FR, Perez VL, Rocha EM, Sullivan BD, Sullivan DA, Vehof J, Vitale S, Willcox M, Wolffsohn JS, Dogru M. TFOS DEWS III: Digest. *Am J Ophthalmol.* 2025;279:451-553.

3. Baudouin C, Labbé A, Liang H, Pauly A, Brignole-Baudouin F. Preservatives in eyedrops: the good, the bad and the ugly. *Prog Retin Eye Res.* 2010;29:312-334.
4. Wolffsohn JS, Arita R, Chalmers R, Djalilian A, Dogru M, Dumbleton K, Gupta PK, Karpecki P, Lazreg S, Pult H, Sullivan BD, Tomlinson A, Tong L, Villani E, Yoon KC, Jones L, Craig JP. TFOS DEWS II diagnostic methodology report. *Ocul Surf.* 2017;15:539-574.
5. Kobashi H, Kamiya K, Shimizu K. Dry eye after Small Incision Lenticule Extraction and femtosecond laser-assisted LASIK: meta-analysis. *Cornea.* 2017;36:85-91.
6. Stapleton F, Alves M, Bunya VY, Jalbert I, Lekhanont K, Malet F, Na KS, Schaumberg D, Uchino M, Vehof J, Viso E, Vitale S, Jones L. TFOS DEWS II epidemiology report. *Ocul Surf.* 2017;15:334-365.
7. Wilson SE, Ambrósio R. Laser in situ keratomileusis-induced neurotrophic epitheliopathy. *Am J Ophthalmol.* 2001;132:405-406.
8. D'Souza S, James E, Swarup R, Mahuvakar S, Pradhan A, Gupta K. Algorithmic approach to diagnosis and management of post-refractive surgery dry eye disease. *Indian J Ophthalmol.* 2020;68:2888-2894.
9. Bower KS, Sia RK, Ryan DS, Mines MJ, Dartt DA. Chronic dry eye in photorefractive keratectomy and laser in situ keratomileusis: manifestations, incidence, and predictive factors. *J Cataract Refract Surg.* 2015;41:2624-2634.
10. Gomes JAP, Azar DT, Baudouin C, Efron N, Hirayama M, Horwath-Winter J, Kim T, Mehta JS, Messmer EM, Pepose JS, Sangwan VS, Weiner AL, Wilson SE, Wolffsohn JS. TFOS DEWS II iatrogenic report. *Ocul Surf.* 2017;15:511-538.
11. Lee BH, McLaren JW, Erie JC, Hodge DO, Bourne WM. Reinnervation in the cornea after LASIK. *Invest Ophthalmol Vis Sci.* 2002;43:3660-3664.
12. Battat L, Macri A, Dursun D, Pflugfelder SC. Effects of laser in situ keratomileusis on tear production, clearance, and the ocular surface. *Ophthalmology.* 2001;108:1230-1235.
13. Feng YE, Yu JG, Wang DD, Li JH, Huang JH, Shi JL, Ye T, Wang QM, Zhao YE. The effect of hinge location on corneal sensation and dry eye after LASIK: a systematic review and meta-analysis. *Graefes Arch Clin Exp Ophthalmol.* 2013;251:357-366.
14. Turu L, Alexandrescu C, Stana D, Tudosescu R. Dry eye disease after LASIK. *J Med Life.* 2012;5:82-84.
15. Barber L, Khodai O, Croley T, Lievens C, Montaquila S, Ziemanski J, McCart M, Lunacek O, Burk C, Patel V. Dry eye symptoms and impact on vision-related function across International Task Force guidelines severity levels in the United States. *BMC Ophthalmol.* 2018;18:260.
16. Cai WT, Liu QY, Ren CD, Wei QQ, Liu JL, Wang QY, Du YR, He MM, Yu J. Dry eye and corneal sensitivity after Small Incision Lenticule Extraction extraction and femtosecond laser-assisted in situ keratomileusis: a meta-analysis. *Int J Ophthalmol.* 2017;10:632-638.
17. Li M, Niu L, Qin B, Zhou Z, Ni K, Le Q, Xiang J, Wei A, Ma W, Zhou X. Confocal comparison of corneal reinnervation after Small Incision Lenticule Extraction (SMILE) and femtosecond laser in situ keratomileusis (FS-LASIK). *PLoS One.* 2013;8:e81435.
18. Reinstein DZ, Carp GI, Archer TJ, Gobbe M. Outcomes of Small Incision Lenticule Extraction (SMILE) in low myopia. *J Refract Surg.* 2014;30:812-818. Erratum in: *J Refract Surg.* 2015;31:60.
19. Dong Z, Zhou X, Wu J, Zhang Z, Li T, Zhou Z, Zhang S, Li G. Small Incision Lenticule Extraction (SMILE) and femtosecond laser LASIK: comparison of corneal wound healing and inflammation. *Br J Ophthalmol.* 2014;98:263-269.
20. Can İ. Dry eye after cataract and corneal laser refractive surgeries. *MN Oftalmoloji.* 2016;23(Suppl 1):64-71.
21. Öncel B, Kılıç İ, Akova YA. Anterior segment surgeries and dry eye: diagnosis and therapeutic approach. *Turk J Ophthalmol.* 2012;42:227-234.
22. Mencucci R, Vignapiano R, Rubino P, Favuzza E, Cantera E, Aragona P, Rolando M. Iatrogenic dry eye disease: dealing with the conundrum of post-cataract discomfort. A P.I.C.A.S.S.O. Board Narrative Review. *Ophthalmol Ther.* 2021;10:211-223.
23. Han KE, Yoon SC, Ahn JM, Nam SM, Stulting RD, Kim EK, Seo KY. Evaluation of dry eye and meibomian gland dysfunction after cataract surgery. *Am J Ophthalmol.* 2014;157:1144-1150.e1.
24. Hwang HB, Kim HS. Phototoxic effects of an operating microscope on the ocular surface and tear film. *Cornea.* 2014;33:82-90.
25. Ishrat S, Nema N, Chandravanshi SCL. Incidence and pattern of dry eye after cataract surgery. *Saudi J Ophthalmol.* 2019;33:34-40.
26. Fraunfelder FT, Sciubba JJ, Mathers WD. The role of medications in causing dry eye. *J Ophthalmol.* 2012;2012:285851.
27. Patel M, Fraunfelder FW. Toxicity of topical ophthalmic anesthetics. *Expert Opin Drug Metab Toxicol.* 2013;9:983-988.
28. Sarkar J, Chaudhary S, Namavari A, Ozturk O, Chang JH, Yeo L, Sonawane S, Khanolkar V, Hallak J, Jain S. Corneal neurotoxicity due to topical benzalkonium chloride. *Invest Ophthalmol Vis Sci.* 2012;53:1792-1802.
29. Cha SH, Lee JS, Oum BS, Kim CD. Corneal epithelial cellular dysfunction from benzalkonium chloride (BAC) in vitro. *Clin Exp Ophthalmol.* 2004;32:180-184.
30. Shao D, Zhu X, Sun W, Cheng P, Chen W, Wang H. Effects of femtosecond laser-assisted cataract surgery on dry eye. *Exp Ther Med.* 2018;16:5073-5078.
31. Starr CE, Gupta PK, Farid M, Beckman KA, Chan CC, Yeu E, Gomes JAP, Ayers BD, Berdahl JP, Holland EJ, Kim T, Mah FS; ASCRS Cornea Clinical Committee. An algorithm for the preoperative diagnosis and treatment of ocular surface disorders. *J Cataract Refract Surg.* 2019;45:669-684.
32. Jones L, Downie LE, Korb D, Benitez-Del-Castillo JM, Dana R, Deng SX, Dong PN, Geerling G, Hida RY, Liu Y, Seo KY, Tauber J, Wakamatsu TH, Xu J, Wolffsohn JS, Craig JP. TFOS DEWS II management and therapy report. *Ocul Surf.* 2017;15:575-628.
33. Sanad AA, Shabana RR, Awara AM, Shalaby OE. Ocular surface changes before and after upper eyelid blepharoplasty. *Int J Med Ophthalmol.* 2023;5:7-11.
34. Prischmann J, Sufyan A, Ting JY, Ruffin C, Perkins SW. Dry eye symptoms and chemosis following blepharoplasty: a 10-year retrospective review of 892 cases in a single-surgeon series. *JAMA Facial Plast Surg.* 2013;15:39-46.
35. Hamawy AH, Farkas JB, Fagien S, Rohrich RJ. Preventing and managing dry eyes after periorbital surgery: a retrospective review. *Plast Reconstr Surg.* 2009;123:353-359.
36. Aksu Ceylan N, Yeniad B. Effects of upper eyelid surgery on the ocular surface and corneal topography. *Turk J Ophthalmol.* 2022;52:50-56.
37. Saadat D, Dresner SC. Safety of blepharoplasty in patients with preoperative dry eyes. *Arch Facial Plast Surg.* 2004;6:101-104.
38. Shao C, Fu Y, Lu L, Chen J, Shen Q, Zhu H, Fan X. Dynamic changes of tear fluid after cosmetic transcutaneous lower blepharoplasty

- measured by optical coherence tomography. *Am J Ophthalmol*. 2014;158:55-63.e1.
39. Mack W. Blepharoplasty complications. *Facial Plast Surg*. 2012;28:273-287.
  40. Su Y, Liang Q, Su G, Wang N, Baudouin C, Labbé A. Spontaneous eye blink patterns in dry eye: clinical correlations. *Invest Ophthalmol Vis Sci*. 2018;59:5149-5156.
  41. Kiang L, Deptula P, Mazhar M, Murariu D, Parsa FD. Muscle-sparing blepharoplasty: a prospective left-right comparative study. *Arch Plast Surg*. 2014;41:576-583.
  42. Zhang SY, Yan Y, Fu Y. Cosmetic blepharoplasty and dry eye disease: a review of the incidence, clinical manifestations, mechanisms and prevention. *Int J Ophthalmol*. 2020;13:488-492.
  43. Zhang S, Yan Y, Lu Y, Zhou Y, Fu Y. Effect of transcutaneous upper eyelid blepharoplasty on blink parameters and lipid layer thickness. *Front Med (Lausanne)*. 2021;8:732041.
  44. Gonnermann J, Klein JP, Klamann MK, Maier AK, Pleyer U, Jousseaume AM, Bertelmann E. Dry eye symptoms in patients after eyelid reconstruction with full-thickness eyelid defects: using the Tomey TG-1000 thermographer. *Ophthalmic Res*. 2012;48:192-198.
  45. Mian OT, Lippe CM, Khan A, Bugg VA, Bryant JC, Riaz KM, Dvorak JD, Ding K, Moreau A. Dry eye in the upper blepharoplasty patient: a study comparing orbicularis-sparing versus orbicularis-excising techniques. *Graefes Arch Clin Exp Ophthalmol*. 2023;261:3625-3634.
  46. Oh JR, Chang JH, Yoon JS, Jang SY. Change in quality of life of patients undergoing silicone stent intubation for nasolacrimal duct stenosis combined with dry eye syndrome. *Br J Ophthalmol*. 2015;99:1519-1522.
  47. Codner MA, Wolfli JN, Anzarut A. Primary transcutaneous lower blepharoplasty with routine lateral canthal support: a comprehensive 10-year review. *Plast Reconstr Surg*. 2008;121:241-250.
  48. Massry GG. Prevalence of lacrimal gland prolapse in the functional blepharoplasty population. *Ophthalmic Plast Reconstr Surg*. 2011;27:410-413.
  49. Kocabeyoglu S, Sekeroglu HT, Mocan MC, Muz E, Irkeç M, Sanac AS. Ocular surface alterations in blepharospasm patients treated with botulinum toxin A injection. *Eur J Ophthalmol*. 2014;24:830-834.
  50. Dutton JJ, Fowler AM. Botulinum toxin in ophthalmology. *Surv Ophthalmol*. 2007;52:13-31.
  51. Grupcheva CN, Grupchev DI, Usheva N, Grupcheva LO. Beauty versus health-how eyelash extensions may affect dry eye disease? *J Clin Med*. 2024;13:3101.
  52. Masud M, Moshirfar M, Shah TJ, Gomez AT, Avila MR, Ronquillo YC. Eyelid cosmetic enhancements and their associated ocular adverse effects. *Med Hypothesis Discov Innov Ophthalmol*. 2019;8:96-103.
  53. Lee KWA, Chan LKW, Lee AWK, Lee CH, Wan J, Yi KH. Ocular and Periocular tattoo adverse effects: a review. *Diagnostics (Basel)*. 2024;14:2150.
  54. Mocan MC, Uzunozmanoglu E, Kocabeyoglu S, Karakaya J, Irkeç M. The association of chronic topical prostaglandin analog use with meibomian gland dysfunction. *J Glaucoma*. 2016;25:770-774.
  55. Zhou X, Zhang X, Zhou D, Zhao Y, Duan X. A narrative review of ocular surface disease related to anti-glaucomatous medications. *Ophthalmol Ther*. 2022;11:1681-1704.
  56. Shen W, Huang B, Yang J. Ocular surface changes in prostaglandin analogue-treated patients. *J Ophthalmol*. 2019;2019:9798272.
  57. Tong L, Chaurasia SS, Mehta JS, Beuerman RW. Screening for meibomian gland disease: its relation to dry eye subtypes and symptoms in a tertiary referral clinic in singapore. *Invest Ophthalmol Vis Sci*. 2010;51:3449-3454.
  58. The epidemiology of dry eye disease: report of the Epidemiology Subcommittee of the International Dry Eye Workshop (2007). *Ocul Surf*. 2007;5:93-107.
  59. Borkar DS, Lacouture ME, Basti S. Spectrum of ocular toxicities from epidermal growth factor receptor inhibitors and their intermediate-term follow-up: a five-year review. *Support Care Cancer*. 2013;21:1167-1174.
  60. Tsubota K, Yokoi N, Shimazaki J, Watanabe H, Dogru M, Yamada M, Kinoshita S, Kim HM, Tchah HW, Hyon JY, Yoon KC, Seo KY, Sun X, Chen W, Liang L, Li M, Liu Z; Asia Dry Eye Society. New perspectives on dry eye definition and diagnosis: a consensus report by the Asia Dry Eye Society. *Ocul Surf*. 2017;15:65-76.
  61. Dumbleton K, Caffery B, Dogru M, Hickson-Curran S, Kern J, Kojima T, Morgan PB, Purslow C, Robertson DM, Nelson JD; members of the TFOS International Workshop on Contact Lens Discomfort. The TFOS International Workshop on Contact Lens Discomfort: report of the subcommittee on epidemiology. *Invest Ophthalmol Vis Sci*. 2013;54:TFOS20-TFOS36.
  62. Koh S, Chalmers R, Kabata D, Shintani A, Nishida K. Translation and validation of the 8-item Contact Lens Dry Eye Questionnaire (CLDEQ-8) among Japanese soft contact lens wearers: The J-CLDEQ-8. *Cont Lens Anterior Eye*. 2019;42:533-539.
  63. Koh S. Contact lens wear and dry eye: beyond the known. *Asia Pac J Ophthalmol (Phila)*. 2020;9:498-504.
  64. Nichols KK, Redfern RL, Jacob JT, Nelson JD, Fonn D, Forstot SL, Huang JF, Holden BA, Nichols JJ; members of the TFOS International Workshop on Contact Lens Discomfort. The TFOS International Workshop on Contact Lens Discomfort: report of the definition and classification subcommittee. *Invest Ophthalmol Vis Sci*. 2013;54:TFOS14-TFOS19.
  65. Duong K, McGwin G Jr, Franklin QX, Cox J, Pucker AD. Treating uncomfortable contact lens wear with orthokeratology. *Eye Contact Lens*. 2021;47:74-80.
  66. Şengör T, Alkibay S, Ermeç Sertoğlu A, Aydın Kurna S. Survey to determine perceptions and practices in contact lens use and identify key features of safe use education. *Turk J Ophthalmol*. 2018;48:288-294.
  67. Alipour F, Khaheishi S, Soleimanzadeh M, Heidarzadeh S, Heydarzadeh S. Contact lens-related complications: a review. *J Ophthalmic Vis Res*. 2017;12:193-204.
  68. Nichols JJ, Sinnott LT. Tear film, contact lens, and patient-related factors associated with contact lens-related dry eye. *Invest Ophthalmol Vis Sci*. 2006;47:1319-1328.
  69. Efron N. Is contact lens wear inflammatory? *Br J Ophthalmol*. 2012;96:1447-1448.
  70. Efron N. Contact lens wear is intrinsically inflammatory. *Clin Exp Optom*. 2017;100:3-19.
  71. Papas EB, Ciolino JB, Jacobs D, Miller WL, Pult H, Sahin A, Srinivasan S, Tauber J, Wolffsohn JS, Nelson JD; members of the TFOS International Workshop on Contact Lens Discomfort. The TFOS International Workshop on Contact Lens Discomfort: report of the management and therapy subcommittee. *Invest Ophthalmol Vis Sci*. 2013;54:TFOS183-TFOS203.
  72. Srinagesh V, Ellenberg D, Scharper PH, Etter J. Intravitreal Dry Eye Study. *Invest Ophthalmol Vis Sci*. 2014;55:3696-3696.

73. Laude A, Lim JW, Srinagesh V, Tong L. The effect of intravitreal injections on dry eye, and proposed management strategies. *Clin Ophthalmol.* 2017;11:1491-1497.
74. Garg P, Roy A, Sharma S. Endophthalmitis after cataract surgery: epidemiology, risk factors, and evidence on protection. *Curr Opin Ophthalmol.* 2017;28:67-72.
75. Zhang Y, Ou Z, Zhou J, Zhai J, Gu J, Chen J. Treatment of oculoplastic and ocular surface disease in eyes implanted with a type I Boston Keratoprosthesis in Southern China: a retrospective study. *Adv Ther.* 2020;37:3206-3222.
76. Jones L, Craig JP, Markoulli M, Karpecki P, Akpek EK, Basu S, Bitton E, Chen W, Dhaliwal DK, Dogru M, Gomes JAP, Koehler M, Mehta JS, Perez VL, Stapleton F, Sullivan DA, Tauber J, Tong L, Travé-Huarte S, Wolffsohn JS; TFOS Collaborator Group. TFOS DEWS III: management and therapy. *Am J Ophthalmol.* 2025;279:289-386.
77. Şimşek C, Doğru M, Kojima T, Tsubota K. Current management and treatment of dry eye disease. *Turk J Ophthalmol.* 2018;48:309-313.
78. Epitropoulos A. Refractive surgery for patients with severe dry eye. *Cataract & Refractive Surgery Today.* March 2015. Available from: <https://crstoday.com/articles/2015-mar/refractive-surgery-for-patients-with-severe-dry-eye>
79. Bron AJ, de Paiva CS, Chauhan SK, Bonini S, Gabison EE, Jain S, Knop E, Markoulli M, Ogawa Y, Perez V, Uchino Y, Yokoi N, Zoukhri D, Sullivan DA. Corrigendum to "TFOS DEWS II pathophysiology report" [*Ocul Surf.* 15(3)(2017)]. *Ocul Surf.* 2019;17:842.
80. Aygun O, Arat YO, Dikmetas O, Karakaya J, Baytaroglu A, Irkeç M. Effect of upper eyelid blepharoplasty on the ocular surface, tear film, and corneal microstructure. *Arq Bras Oftalmol.* 2024;87:e20220220.
81. Ciftci MD, Selver OB. A review of oculoplastic surgery and ocular surface disorders. *Med Hypothesis Discov Innov Optom.* 2022;3:5-11.
82. Nagendran ST, Ali MJ, Dogru M, Malhotra R. Complications and adverse effects of periocular aesthetic treatments. *Surv Ophthalmol.* 2022;67:741-757.
83. Han J, Xie Z, Zhu X, Ruan W, Lin M, Xu Z, Miao L, Zhong J, Lu F, Hu L. The effects of eyelash extensions on the ocular surface. *Cont Lens Anterior Eye.* 2024;47:102109.