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REVIEW

Advances in the management and therapy of dry eye disease: Insights from TFOS DEWS III

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Abstract

Dry eye disease (DED) is a multifactorial ocular surface disorder driven by tear film instability, hyperosmolar stress, and immune-mediated inflammation, ultimately leading to visual disturbance and reduced quality of life. This review integrates current evidence-based recommendations consistent with the Tear Film and Ocular Surface Society Dry Eye Workshop III (TFOS DEWS III) Diagnostic Methodology and aligns therapeutic interventions with prevalent DED phenotypes to facilitate precision-based management. The proposed stepwise algorithm advances from environmental and behavioral modification, tear supplementation, and ocular surface lubrication to targeted eyelid therapies, anti-inflammatory and immunomodulatory treatments, device-assisted modalities, ocular surface rehabilitation, nutritional supplementation, and perioperative prophylaxis. Given the heterogeneity of disease mechanisms and the variable strength of supporting evidence, DED management must be tailored to individual phenotypic expression and disease severity, with systematic re-evaluation of symptoms, objective clinical parameters, and therapeutic safety.

Keywords: Dry eye disease; ocular surface; DEWS III.

The Tear Film and Ocular Surface Society Dry Eye Workshop III (TFOS DEWS III) Management and Therapy report updates the evidence presented in TFOS DEWS II and integrates it with current diagnostic recommendations, acknowledging the heterogeneity of dry eye disease (DED) and the overlap among therapeutic mechanisms (anti-inflammatory, antimicrobial, and eyelid-directed). Its aim is to summarize available treatments, critically appraise the supporting data, and align therapeutic interventions with the TFOS DEWS III subclassifications to enable precise, individualized care.^[1] The TFOS DEWS III Management and Therapy Report updates and expands

the treatment framework established by TFOS DEWS II by shifting from a stepwise approach to a more personalized, phenotype-based model of dry eye care. Whereas DEWS II focused on defining the disease and outlining diagnostic standards, DEWS III integrates advances in imaging, biomarkers, and new therapeutic technologies to guide mechanism-specific management. This updated report places greater emphasis on meibomian gland dysfunction, neurosensory abnormalities, lifestyle factors, and modern device-based and pharmacologic treatments, providing a more clinically actionable roadmap for contemporary dry eye therapy.



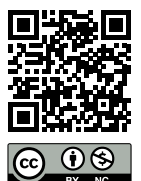
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Lifestyle Advice

Lifestyle is increasingly recognized as a key determinant of both the onset and long-term control of DED. Prolonged screen use—particularly with computers, tablets, and smartphones—is associated with a reduced blink rate, a higher frequency of incomplete blinks, and consequent tear film breakup, thereby promoting evaporative loss and exposing the ocular surface epithelium to mechanical and osmotic stress.^[2] In addition, several other modifiable factors—including sleep quality, the application and removal practices of periocular cosmetics, systemic and topical medications, and overall nutritional status—may exacerbate or mitigate symptoms by influencing tear film composition, meibomian and lacrimal gland function, ocular surface inflammation, and neurosensory processing.^[2-4] Psychological comorbidities, particularly anxiety and depression, are common in individuals with DED and are thought to amplify symptom perception, impair adherence to therapy, and further reduce health-related quality of life.^[5,6] Environmental stressors such as low ambient humidity, wind exposure, air pollution, and chronic ultraviolet irradiation can additionally destabilize the tear film and induce or perpetuate ocular surface damage and inflammation.^[7] Consequently, lifestyle and environmental optimization constitute a fundamental component of DED management. Recommended measures include implementing scheduled screen breaks with conscious, complete blinking, minimizing exposure to excessively dry or windy environments, improving indoor humidity with humidifiers, and using moisture-retaining eyewear or wrap-around glasses in high-risk settings.^[8,9]

Tear Insufficiency

Tear Replenishment — Supplements and Stabilisers

Tear supplements are considered first-line therapy across virtually all DED etiologies, and their clinical performance is determined by the overall formulation rather than a single “active” ingredient.^[10] Key determinants include the polymer base, the presence and type of preservatives, and the delivery system (single-dose vs. multi-dose, viscosity, drop vs. gel). Aqueous-oriented polymers such as carboxymethylcellulose (CMC), carbomer, hydroxypropyl (HP) guar, hydroxypropyl methylcellulose (HPMC), polyvinyl alcohol, polyvinylpyrrolidone, and polyethylene glycol (PVA/PVP/PEG) primarily augment the aqueous layer of the tear film, improve lubrication, and prolong residence time, making them particularly suitable for aqueous-deficient phenotypes.^[10-14] Beyond simple lubrication, many contemporary formulations incorporate excipients that

target specific components of the DED vicious cycle. Hypo-osmotic preparations aim to normalize elevated tear osmolarity, thereby reducing osmotic stress and epithelial injury.^[15, 16] Osmoprotectants such as L-carnitine, erythritol, trehalose, betaine, sorbitol, and glycerin enhance cellular resilience, enabling ocular surface epithelial cells to better tolerate desiccating and inflammatory stress.^[17-19] Buffers and salts maintain physiologic pH and tonicity, which are critical for epithelial integrity, patient comfort, and tolerance of concomitant topical medications.^[10-13]

Given the cumulative toxicity associated with benzalkonium chloride (BAK) on the corneal and conjunctival epithelium, modern strategies favor preservative-free unit-dose or advanced multi-dose systems; alternatively, milder oxidizing preservatives or very low concentrations of polyquaternium-1 (PQ-1) may be used, particularly in patients requiring chronic or high-frequency instillation.^[18-20] Within this landscape, several formulations have supportive clinical evidence. HP guar-containing gels have been associated with reduced tear osmolarity and, in some studies, increased goblet cell density when used with PEG/borate systems.^[20] High-molecular-weight HA, as well as fixed combinations of CMC and HA, has been linked to improved tear film stability, better corneal and conjunctival staining scores, and longer tear breakup time (TBUT).^[21, 22] Xanthan gum, with its shear-thinning rheological properties, enhances drop residency, TBUT, and conjunctival hyperemia.^[23] Combination formulations including HP guar, HA, and xanthan gum aim to maximize surface hydration and epithelial recovery, and have shown benefit in postoperative cataract patients and individuals with heavy digital device use.^[24] Lipid nanoemulsions extend TBUT and are particularly useful in evaporative DED associated with meibomian gland dysfunction (MGD).^[25, 26] Perfluorohexyloctane not only forms a highly stable anti-evaporative monolayer but may also activate TRPM8 channels, potentially modulating ocular surface sensation and improving symptoms.^[27] Trehalose, frequently combined with HA, stabilizes proteins and cell membranes and reduces oxidative stress-induced damage,^[28, 29] while ectoine has been shown to decrease pro-inflammatory cytokine expression, enhance mucin production, and prolong TBUT.^[30] Antioxidant-enriched formulations and drops containing vitamins A and B12 may support epithelial repair, corneal nerve health, and symptom improvement; however, substantial heterogeneity in formulations, dosing regimens, and outcome measures—as well as a limited number of standardized head-to-head comparative trials—currently constrains robust conclusions across products.^[31-33]

Tear Conservation Devices

Therapeutic/scleral lenses maintain a fluid reservoir for severe, refractory DED; monitor for hypoxia, keratitis, and fit issues.^[34]

Moisture-retaining spectacles raise periocular humidity to reduce evaporation.^[35, 36] Punctal occlusion, temporary or semi-permanent, reduces drainage; thermal cauterization offers permanence. Complications include foreign body sensation, extrusion, and canaliculitis; careful selection and follow-up are essential.^[37]

Newer designs, such as cross-linked HA canalicular fillers and biodegradable plugs, enable titratable retention with favorable profiles.^[38]

Tear Restoration/Stimulation

Lipid Layer (MGD Directed): Home-based measures such as the application of warm compresses at 40–45 °C, adequate dietary intake of omega-3 fatty acids, topical azithromycin, and selenium disulfide-containing preparations primarily target meibomian gland obstruction, soften inspissated secretions, and thereby improve tear film stability. Office-based interventions, including thermal pulsation systems, intense pulsed light (IPL), low-level light therapy (LLLT), radiofrequency devices, quantum molecular resonance (QMR) technologies, and autologous plasma-based approaches, further enhance gland patency, promote more physiological meibum expression, and can lead to sustained prolongation of TBUT. In more advanced or refractory cases, meibomian gland probing and lid margin debridement are employed to mechanically disrupt fibrotic tissue and biofilm at the gland orifices, restoring outflow and improving ocular surface homeostasis.^[39-45]

Aqueous: Diquafosol is a P2Y₂ receptor agonist that stimulates both aqueous and mucin secretion, thereby enhancing tear film volume and improving ocular surface wetting. Oral secretagogues such as pilocarpine and cevimeline similarly increase exocrine gland output and can augment lacrimal flow; however, their use is limited by systemic cholinergic adverse effects and, therefore, requires careful patient selection and regular systemic monitoring.^[46-48]

Mucin: Diquafosol boosts membrane/gel-forming mucins; rebamipide increases goblet cells and reduces inflammation in markets where available.^[46, 47, 49]

Neuromodulation: Intranasal or transcutaneous neurostimulation devices (e.g., iTear100®) increase reflex tearing by activating sensory pathways within the nasal mucosa and associated trigeminal circuits, thereby

augmenting basal and reflex lacrimation. In parallel, pharmacological agents such as varenicline nasal spray, acoltremon, and Cryosim 3 act on nasolacrimal and ophthalmic neural pathways to enhance tear production, offering a neuromodulatory approach to managing tear deficiency.^[50-52]

Treatments for Eyelid Abnormalities

Blink and Lid Closure

Incomplete blinking, which is particularly common during prolonged digital device use, impairs the normal distribution of meibum across the ocular surface and thereby accelerates tear film evaporation and destabilization. Digital prompts, scheduled breaks, and structured blink-training protocols can help patients re-establish a more complete and regular blink pattern, improving lipid layer spread and tear film stability.^[53] Nocturnal lagophthalmos can further exacerbate DED by increasing overnight ocular surface exposure, worsening both clinical signs and sleep quality. First-line management typically includes lubricating ointments at bedtime, eyelid taping, or the use of moisture-chamber goggles, whereas more invasive procedures—such as partial tarsorrhaphy—are reserved for persistent or severe cases that do not respond to conservative measures.^[54, 55] Partial blinking itself has been identified as an independent risk factor for DED, conferring approximately a 2.2-fold increased risk and being associated with higher OSDI scores, more pronounced MGD and gland dropout, reduced lipid layer thickness (LLT), and shorter non-invasive tear breakup time (NIBUT).^[56] Targeted blink-rehabilitation strategies have been shown to improve LLT and NIBUT and to alleviate subjective symptoms, underscoring the importance of blink quality—beyond simply blink rate—as a modifiable behavioral parameter in DED management.^[56-58]

Reducing Eyelid Microbial Load

Routine lid hygiene is a cornerstone of eyelid disease management and has been shown to mitigate both anterior and posterior blepharitis, reduce microbial and biofilm burden, and attenuate secondary inflammation affecting the lid margin and ocular surface. Regular application of warm compresses followed by gentle mechanical cleansing of the lid margin helps remove crusts, debris, and inspissated secretions, thereby improving meibomian gland function and supporting tear film stability.^[59, 60]

Anti-Demodex: Targeted anti-Demodex therapy is often required when Demodex infestation contributes to

blepharitis. Tea tree oil (TTO)–based lid wipes are generally considered first-line therapy, typically applied over a 4–6-week course, and have been shown to significantly reduce mite density and associated inflammation. For patients who are intolerant of TTO or in more refractory cases, ivermectin or metronidazole gels offer effective alternatives with documented anti-Demodex and anti-inflammatory activity.^[61-64]

Hypochlorous Acid 0.01%: Hypochlorous acid (HOCl) 0.01% has demonstrated >90% reduction in lid bacterial load while preserving commensal flora, offering an attractive antimicrobial approach with a favorable tolerability profile.^[65-67]

Lid Hygiene Products: Overall, dedicated lid hygiene preparations—including wipes, gels, foams, and sprays—tend to outperform traditional baby shampoo in terms of efficacy and patient comfort and are associated with fewer adverse effects such as irritation or dermatitis.^[68,69]

Low-Level Blue Light: Adjunctive modalities are emerging for biofilm and microbial control. Low-level blue light therapy has been proposed as a means to disrupt lid margin biofilm and reduce microbial colonization.^[70]

Manuka Honey: Manuka honey–based formulations exhibit both antimicrobial and anti-inflammatory activity, with reported improvements in symptoms and lipid layer quality; in clinical use, mild and transient stinging is relatively common but generally self-limited.^[71,72]

Topical Antibiotics: Topical antibiotics—including erythromycin, azithromycin, vancomycin, and hypochlorous acid (HOCl)–based preparations—are used to reduce lid bacterial burden, particularly in cases with significant *Staphylococcus* colonization or rosacea-associated blepharitis, and are often incorporated into a broader regimen of lid hygiene and anti-inflammatory therapy.^[69]

Anti-Inflammatory Pharmacological Therapies

Topical Corticosteroids: Topical corticosteroids provide rapid and often substantial relief during inflammatory flares of DED by suppressing ocular surface inflammation and improving patient comfort; however, their use should generally be limited to short-term courses or intermittent pulsed regimens because of well-recognized risks, including intraocular pressure elevation and cataract formation with prolonged or uncontrolled exposure. Meta-analytic data indicate that, although corticosteroids can produce modest improvements in symptoms and corneal/conjunctival staining, these benefits must be weighed against their safety profile.^[73] Loteprednol etabonate 0.25% (Eysuvis®),

an ester-based “soft” steroid, is FDA-approved for up to 2 weeks of use for “dry eye flares” and has demonstrated improvements in both symptoms and clinical signs with relatively low rates of adverse events in appropriately selected patients.^[73]

Medicated Tear Supplements: Formulations that combine a lubricating base with very low-dose corticosteroid—for example, 0.2% hyaluronic acid (HA) plus 0.001% hydrocortisone—have demonstrated superiority over HA–trehalose comparators in terms of TBUT and lipid layer thickness (LLT), although intraocular pressure should still be monitored when such formulations are used beyond the short term.^[74]

Topical Immunomodulators are central to longer-term control of inflammatory DED. Cyclosporine A improves lacrimal gland function, enhances goblet cell density, and helps restore ocular surface homeostasis, making it a key maintenance therapy in chronic disease.^[75] Lifitegrast 5% has been shown to reduce symptoms of dryness and improve corneal staining with a generally favorable tolerability profile, providing an alternative or adjunct to cyclosporine in suitable patients.^[76] Tacrolimus is typically reserved for severe or refractory cases, including those with significant ocular surface inflammation or associated immune-mediated disease.^[77]

Emerging compounds—including carbonized nanogels, fenofibrate, ferulic acid, lacritin and its analogue lacripep™, naringenin, repository corticotropin injection, reproxalap, porphyrin-based agents, and the mitochondria-targeted antioxidant Visomitin (SkQ1)—are under investigation at various stages of preclinical and clinical development, with the aim of more precisely modulating oxidative stress, lipid metabolism, neurosensory pathways, or tear film biology.^[74,78,79]

Oral Antimicrobial Agents (Tetracyclines, Macrolides)

Systemic antibiotics are used primarily in MGD- and rosacea-associated DED, where they exert anti-inflammatory, anti-angiogenic, and lipid-stabilizing effects rather than functioning solely as antimicrobial agents.^[80] Tetracyclines downregulate matrix metalloproteinases (MMPs) and pro-inflammatory cytokines, leading to improvements in TBUT and corneal and conjunctival staining; symptom relief is often modest but clinically meaningful in appropriately selected patients. However, these agents are contraindicated during pregnancy and in young children due to their effects on teeth and bone, and gastrointestinal

intolerance and photosensitivity are among the most common adverse events, necessitating careful counseling and monitoring.^[80,81] Macrolides, particularly azithromycin, also modulate lid margin inflammation and have been shown to enhance meibomian gland secretion and activity. Short 5-day courses of oral or topical azithromycin can achieve improvements in signs and symptoms comparable to those obtained with 4-week doxycycline regimens, but with fewer gastrointestinal side effects, making them an attractive option for many patients. Erythromycin is often preferred in pediatric populations due to its favorable safety profile and long-standing clinical use.^[82, 83] Overall, these systemic agents should be viewed as short-term adjunctive therapies aimed at reducing inflammation and improving gland function, rather than as chronic stand-alone treatments for DED.^[82]

Ocular Surface Promoters / Regenerators

Autologous or allogeneic blood-derived eye drops, including serum, platelet-rich plasma (PRP), plasma rich in growth factors (PRGF), and umbilical cord serum, provide a complex mixture of trophic and immunomodulatory factors that more closely resemble natural tears than conventional artificial lubricants. These preparations supply epidermal, nerve, transforming, and insulin-like growth factors (EGF, NGF, TGF, IGF), as well as vitamin A, albumin, and a range of anti-inflammatory mediators, thereby supporting epithelial proliferation, migration, nerve health, and ocular surface homeostasis. Randomized controlled trials and meta-analyses have demonstrated that, compared with artificial tears, such biological drops yield greater improvements in symptoms, corneal, and conjunctival staining.^[84-88]

Umbilical cord serum may offer higher concentrations of trophic factors and anti-inflammatory mediators than adult serum, potentially translating into stronger regenerative effects in selected patients.^[89] Whole blood autologous eye drops, which are prepared without fractionation, have also been reported to improve signs and symptoms; however, their success is highly dependent on patient adherence to storage, handling, and dosing requirements.^[90]

Lubricin (recombinant human proteoglycan 4, rhPRG4), a boundary-lubricating glycoprotein, outperformed 0.18% hyaluronic acid in a small randomized controlled trial and has been shown to bind and inhibit matrix metalloproteinase-9 (MMP-9), suggesting both mechanical and biochemical protective effects on the ocular surface.^[91]

Amniotic membrane-derived extracts and amniotic membrane eye drops have been used in refractory epithelial defects and severe ocular surface disease, providing anti-inflammatory, anti-fibrotic, and pro-healing signals when conventional therapy is insufficient.^[92]

Cenegermin, a recombinant human NGF, is approved for the treatment of neurotrophic keratitis and has shown preliminary benefit in DED, particularly in patients with significant neurosensory compromise.^[93]

Additional emerging biologic or peptide-based therapies, including RGN-259 (a thymosin β 4 analogue), silk-derived protein (SDP-4) formulations, and topical insulin, have generated early supportive data for improving epithelial integrity and symptoms, but remain at an investigational stage and require further robust, controlled studies before routine incorporation into DED management algorithms.^[94, 95]

Treatments for Anatomical Surface Abnormalities

Conjunctivochalasis, Lid-Parallel Conjunctival Folds (LIPCOF), pinguecula, and pterygium can interfere with smooth tear spreading and destabilize the tear film, thereby exacerbating DED symptoms; correction of these structural abnormalities is associated with symptomatic improvement.^[96]

In conjunctivochalasis, initial management typically consists of lubricants and topical anti-inflammatory agents. In refractory cases, surgical approaches such as conjunctival excision, cauterization, scleral fixation, or laser/plasma conjunctivoplasty may be employed, with reported improvements in OSDI scores, TBUT, and ocular surface staining.^[96-98]

Pinguecula and pterygium are largely related to ultraviolet exposure and environmental irritants. Medical therapy focuses on lubricants and anti-inflammatory drops for symptom control, while surgical excision is reserved for symptomatic lesions or those threatening the visual axis, helping to restore tear film stability and patient comfort.^[99, 100]

Nutritional Modifications and Alternative Therapies

Nutritional and complementary interventions may modulate ocular surface inflammation and tear film stability, although the supporting evidence is heterogeneous.^[101] Polyunsaturated fatty acids (PUFAs), particularly omega-3 fatty acids such as eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), may reduce inflammation,

support epithelial repair, and enhance tear secretion; neuroprotectin D1, a DHA-derived lipid mediator, appears to protect corneal nerves.^[102, 103] Despite the null findings of the DREAM trial, multiple randomized controlled trials and meta-analyses have reported improvements in symptoms, Schirmer scores, TBUT, and tear osmolarity with omega-3 supplementation. Higher dietary omega-3 and lower omega-6 intake have been associated with reduced DED risk; flaxseed oil has shown benefit over approximately 6 months, whereas omega-6-rich oils and trans fats may exacerbate inflammation.^[104-107]

Regarding vitamins and minerals, topical vitamin A supports epithelial integrity, while systemic supplementation is mainly indicated in the setting of deficiency. Vitamin B12, often combined with hyaluronic acid, has been associated with improvements in OSDI, TBUT, and Schirmer scores, including in patients with Sjögren's syndrome. Vitamin D deficiency correlates with DED, and supplementation has been reported to improve tear function parameters and osmolarity. Trace elements such as selenium, zinc, and copper contribute to antioxidant defense and may therefore be relevant in ocular surface protection.^[107, 108]

Among alternative interventions, acupuncture appears to provide modest symptomatic relief and is generally safe when used alongside artificial tears. Various botanical agents—including goji berry, curcumin, cassiae semen, and ferulic acid—demonstrate anti-inflammatory activity in preclinical models, but clinical data remain limited. Adequate systemic hydration supports overall ocular surface homeostasis. Lactoferrin levels are reduced in DED, and oral supplementation may help preserve tear secretion and reduce oxidative stress. Manuka honey-based topical preparations have been reported to improve OSDI, Schirmer scores, and staining, with meta-analytic data providing supportive evidence. Oral royal jelly has yielded modest benefits in some studies, and a multi-nutrient formulation (Blink™ NutriTears®) has been shown to improve tear film stability and reduce MMP-9 levels.^[109-111]

Prevention and Treatment of Surgical Iatrogenic DED

Cataract and keratorefractive procedures (LASIK, PRK, SMILE) can precipitate or exacerbate DED; preoperative surface optimization is essential for accurate biometry and outcomes.^[112, 113] Perioperative control of MGD and blepharitis reduces postoperative flares and improves visual

quality (938). After refractive surgery, transient dryness is common (6–12 months), yet a subset develops persistent evaporative DED refractory to standard tears.^[114-116] For refractory cases, targeted MGD-directed therapies, intense pulsed light (\pm heat mask), and vectored thermal pulsation improve tear stability, lipid quality, OSDI, and TBUT (up to ~8 months).^[116, 117]

Surgical Management

In DED, surgical interventions are reserved for severe or refractory cases that fail to respond to medical or device-based therapies, with the goals of conserving tears, restoring anatomic integrity, and protecting the ocular surface. Permanent punctal occlusion increases tear volume, while tarsorrhaphy reduces exposure and protects the cornea. For eyelid abnormalities, botulinum toxin can be beneficial in managing blepharospasm and spastic entropion; blepharoplasty for dermatochalasis may improve comfort but can alter tear dynamics; entropion and ectropion repair help restore tear-film stability; and lagophthalmos may be treated with gold or platinum weights, cartilage grafts, or tarsorrhaphy—each demonstrating success rates exceeding 80%.^[118]

For ocular surface irregularities, symptomatic conjunctivochalasis may be managed with excision, cautery, or fixation techniques, all of which have high rates of symptomatic improvement. Pterygium and pinguecula are best treated with limbal-conjunctival autografting, with amniotic membrane transplantation as an alternative, and adjuvant therapies such as cyclosporine A, mitomycin-C, 5-fluorouracil, β -irradiation, or anti-VEGF agents may be employed to reduce recurrence. In severe alacrima, salivary gland transplantation can provide continuous lubrication: submandibular gland transfer offers stronger secretory capacity but carries higher complication rates, whereas minor salivary gland grafting is simpler and generally better tolerated.^[118]

Although still experimental, lacrimal gland reinnervation for neurogenic DED has shown promising results, with reported sustained improvements in Schirmer scores, TBUT, and corneal staining.^[20]

Conclusion

The TFOS DEWS III Management and Therapy report supports a pathophysiology-based, evidence-informed framework for personalized DED care. Within this paradigm, first-line interventions—including lifestyle and environmental modification, optimization of digital

behavior and sleep, tear supplementation, and ocular surface protection—aim primarily to stabilize the tear film, restore homeostasis, and interrupt early components of the DED vicious cycle. MGD, as a major driver of evaporative disease, is addressed with heat-based, gland-expressing, and lid hygiene-focused therapies, supplemented when appropriate by anti-inflammatory and antimicrobial lid treatments to normalize lipid secretion and improve tear film stability. Predominantly inflammatory or immune-mediated phenotypes are managed with topical immunomodulators and, in selected cases, biologic or blood-derived tears that support epithelial, goblet cell, and neurosensory recovery.

In more advanced or refractory disease—particularly in the presence of persistent epithelial defects, cicatrizing changes, or significant structural abnormalities—surgical strategies such as amniotic membrane transplantation, limbal or conjunctival grafting, and corrective eyelid or conjunctival procedures may be required to re-establish ocular surface integrity and function. Overall, TFOS DEWS III advocates a flexible, phenotype- and pathophysiology-aligned treatment algorithm that integrates lifestyle modification, meibomian gland optimization, neurosensory and structural considerations, and targeted pharmacologic and biologic therapies. Such an individualized, multimodal approach not only improves clinical signs and symptoms but also aims to restore tear film homeostasis and enhance long-term visual function and quality of life for patients living with DED.

While the current report introduces the armamentarium of available treatment options for dry eye disease, it does not provide severity-based recommendations or specify which combination of treatments delivers sufficient results for dry eyes. The Delphi approach in with TFOS society will tackle these issues to recognize the preferred approach to management in different regions of the world.

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