THE GLOBAL PULSE OF HEALTHCARE · OPHTHALMOLOGY INSIGHT REPORT

Dry Eye Disease Management: When Symptoms Persist Beyond First-Line Treatment

Ophthalmology perspectives on DED phenotype, diagnostic weighting, first-line treatment, persistent symptoms, adherence, ocular-surface inflammation, meibomian gland dysfunction, and the move toward more personalized care.

PHENOTYPE → DIAGNOSIS → FIRST-LINE THERAPY → ESCALATION → LONG-TERM CONTROL

61 Survey records	**6** Countries represented USA · UK · Canada · Italy · France · Germany	**83.6%** Completion rate	**9008491** SGID

WHY THIS REPORT

Dry eye treatment becomes difficult when persistent symptoms expose more than one active disease driver.

Dry eye disease often looks simple at first presentation: burning, fluctuating vision, foreign-body sensation, dryness, or discomfort. The management challenge begins when the patient does not improve as expected, when signs and symptoms diverge, or when meibomian, inflammatory, autoimmune, medication-related, or neurosensory factors overlap.

Clinicians then have to decide whether the first-line plan was insufficient, whether adherence or affordability limited exposure to treatment, whether the original phenotype was incomplete, or whether the patient belongs to a subgroup that needs a different pathway altogether.

The MDForLives survey explores how ophthalmology HCPs make those decisions: which phenotype most often drives management, which findings carry diagnostic weight, what they start first, why treatment fails, how they escalate persistent cases, which subgroups change strategy, and what they expect to transform DED care over the next several years.

> **Survey frame:** 61 survey records with an 83.6% completion rate. Country metadata represents USA · UK · Canada · Italy · France · Germany. Substantive DED questions were answered by 53 respondents. Internal operational, required-text, after-question, and honeypot fields were excluded from analysis.

CLINICAL CONTEXT

TFOS DEWS III reinforces a driver-based model of diagnosis and management rather than a single dry-eye pathway.

TFOS DEWS III, published in 2025, updates dry eye disease as a multifactorial, symptomatic disease involving loss of tear-film and/or ocular-surface homeostasis, with tear-film instability, hyperosmolarity, inflammation and damage, and neurosensory abnormalities among the etiologic factors.

Its diagnostic methodology combines symptoms with objective evidence of lost homeostasis and then uses subclassification to identify etiologic drivers. The framework separates tear-film deficiencies, eyelid and blink abnormalities, ocular-surface abnormalities, and neural dysfunction so that management can be directed toward what is actually driving disease in the individual patient.

The management report similarly emphasizes tear-film support, meibomian gland management, anti-inflammatory therapy, biologic tear substitutes, contact-lens options, and other interventions according to presentation and evidence. This makes “failure” of a first-line treatment a reason to reassess mechanism, implementation, and disease burden rather than simply move to a stronger version of the same therapy.

The MDForLives findings are survey responses from this respondent group and should be interpreted separately from external guidance. They show how clinicians prioritize those decisions in real-world practice.

EXECUTIVE SUMMARY

Six findings that show where dry eye management shifts from first-line support to phenotype-driven escalation.

01

58.5%

Mixed mechanisms dominate

Mixed-mechanism DED is the phenotype most often selected as driving management.

02

47.2%

Staining carries diagnostic weight

Ocular-surface staining is the single finding most often selected as influencing DED diagnosis.

03

60.4%

Preservative-free tears start care

Preservative-free artificial tears remain the most common initial treatment.

04

49.1%

Adherence explains early failure

Poor treatment adherence narrowly leads persistent ocular-surface inflammation as the explanation for first-line failure.

05

67.9%

Symptoms drive continuation

Sustained symptom improvement is the outcome most often used to support long-term treatment.

06

45.3%

Personalized algorithms lead the future

Personalized DED treatment algorithms are the development most often expected to transform care.

01

PHENOTYPE

Mixed-mechanism disease is the dominant management reality

58.5% say mixed-mechanism dry eye most often drives management.

CONTEXT

A dry-eye diagnosis does not automatically identify the treatment target. Mixed presentations can combine tear-film instability, meibomian dysfunction, aqueous deficiency, inflammation, ocular-surface damage, medication effects, and neurosensory abnormalities.

WHAT THE DATA SHOWS

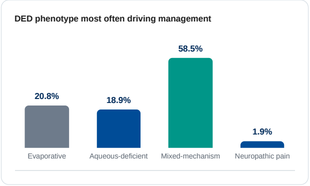
The survey distribution puts this overlap at the center of routine care rather than at its margins. The result also helps explain why treatment can appear to fail even when one mechanism has improved.

THE INSIGHT

Phenotyping has to remain dynamic. A patient who begins as “evaporative” may still need reassessment for inflammation, autoimmune disease, neuropathic pain, or treatment-related ocular-surface injury when the response is incomplete.

WHY IT MATTERS

A mixed phenotype makes sequencing and combination more important than escalation by intensity alone.



02

DIAGNOSTIC WEIGHT

Ocular-surface staining is the finding clinicians most often use to anchor the diagnosis

47.2% select ocular-surface staining as the most influential diagnostic finding.

CONTEXT

DED is diagnosed from a combination of symptoms and objective evidence rather than a single sign. Staining is valuable because it makes epithelial damage visible, but its meaning depends on the rest of the tear-film and ocular-surface examination.

WHAT THE DATA SHOWS

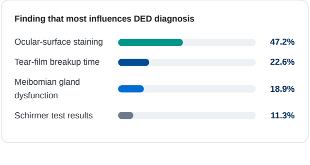
The MDForLives distribution suggests that visible surface damage is carrying the greatest single diagnostic weight in this group, while tear stability and gland findings help explain mechanism and treatment direction.

THE INSIGHT

TFOS DEWS III recommends a standardized diagnostic approach and subclassification of etiologic drivers. That broader framework is especially relevant when symptoms and staining do not move together.

WHY IT MATTERS

A diagnostic test is most useful when it changes the next treatment decision, not simply when it confirms that the ocular surface is abnormal.



03

FIRST-LINE TREATMENT

Preservative-free artificial tears remain the usual starting point, even in a mixed-mechanism population

60.4% initiate preservative-free artificial tears first.

CONTEXT

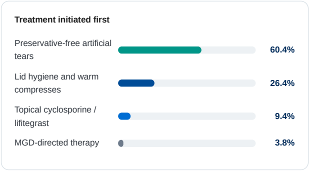
Tear supplementation is low burden, accessible, and broadly applicable, which makes it a practical first step across several DED presentations. But a first step is not the same as a full treatment strategy.

WHAT THE DATA SHOWS

The tension in the survey is clear: most respondents describe mixed disease, yet most still start with a broad tear-support intervention. That is clinically reasonable as long as there is an explicit plan for reassessment and phenotype-specific escalation.

THE INSIGHT

Patients with substantial MGD, inflammation, autoimmune disease, or neurosensory pain may need additional or different therapy earlier depending on severity, contraindications, and clinical findings.



WHY IT MATTERS

First-line treatment works best when it is paired with a predefined question: what finding, symptom, or time point will tell us that the current plan is not enough?

04

WHY TREATMENT FAILS

Poor adherence and persistent inflammation account for most perceived first-line failure

49.1% identify poor treatment adherence as the most common explanation for initial treatment failure.

CONTEXT

DED treatment can be demanding: multiple daily doses, delayed onset of benefit, discomfort on instillation, lid care, device-based procedures, follow-up, and cost all compete with routine life. Incomplete exposure to therapy can therefore look like biological nonresponse.

WHAT THE DATA SHOWS

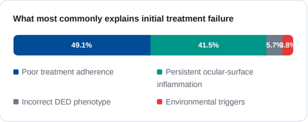
At the same time, the near-equal prominence of persistent inflammation shows that adherence is not the whole story. A well-used lubricant will not fully control an inflammatory driver, and a gland-directed plan may not address autoimmune or neurosensory contributors.

THE INSIGHT

The open comments add real examples of insurance restrictions, long-standing multimodal treatment, and patients whose symptoms remain despite good compliance.

WHY IT MATTERS

Before changing therapies, confirm the treatment was actually usable, affordable, tolerated, and matched to the active driver.


What most commonly explains initial treatment failure

05

PERSISTENT SYMPTOMS

When symptoms persist, clinicians split between suppressing inflammation and intensifying MGD treatment

32.1% escalate anti-inflammatory therapy and 32.1% intensify MGD-directed treatment.

CONTEXT

Persistent symptoms are a fork in the pathway. One route treats inflammation more aggressively; another targets eyelid and gland dysfunction; a third questions the original diagnosis or exposure pattern; a fourth moves to advanced surface therapy.

WHAT THE DATA SHOWS

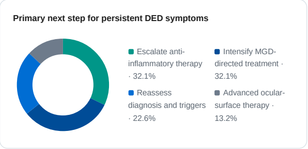
The tie between anti-inflammatory and MGD escalation reflects the mixed-mechanism reality seen earlier in the survey. It also highlights why persistent DED is not a single escalation ladder.

THE INSIGHT

Reassessment becomes particularly important when symptoms are disproportionate to signs, when neuropathic pain is possible, when medication toxicity is present, or when autoimmune disease changes the ocular-surface context.

WHY IT MATTERS

The next step should follow the suspected driver, not merely the fact that symptoms are still present.


Primary next step for persistent DED symptoms

06

REFRACTORY SUBGROUPS

Refractory MGD and autoimmune-associated dry eye most often force a different treatment strategy

37.7% select refractory meibomian gland dysfunction as the subgroup that most changes treatment strategy.

CONTEXT

These subgroups change more than the product choice. They can alter visit frequency, diagnostic depth, coordination with systemic disease care, procedural options, expectations for response, and how clinicians define success.

WHAT THE DATA SHOWS

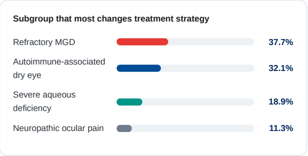
The open-ended responses include autoimmune disease, GVHD, Graves-related disease, persistent meibomitis, toxic or medication-related surface disease, Demodex, and post-refractive neuropathic symptoms.

THE INSIGHT

Some cases improved only after the suspected driver changed - for example, shifting toward immunomodulation, MGD therapy, Demodex treatment, punctal or surface-support strategies, scleral lenses, or multimodal care.

WHY IT MATTERS

Refractory DED is often a diagnostic problem, a comorbidity problem, and an access problem at the same time.


Subgroup that most changes treatment strategy

07

LONG-TERM CONTROL

Sustained symptom improvement matters more than an isolated surface metric when deciding whether to continue treatment

67.9% select sustained symptom improvement as the outcome that most supports long-term therapy.

CONTEXT

DED is a symptomatic disease, and patients judge treatment through comfort, visual fluctuation, functional burden, and day-to-day relief. Objective findings remain important, but they do not always correlate tightly with symptoms.

WHAT THE DATA SHOWS

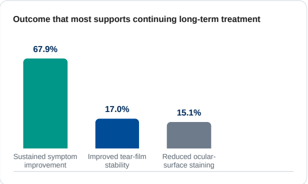
The survey result reflects that reality. A treatment that improves staining without improving the patient's experience may not feel successful, while symptom improvement without full normalization of signs may still support continuation if the ocular surface is stable and risk is acceptable.

THE INSIGHT

Open-ended cases describe both directions of mismatch: patients whose surface improved but symptoms persisted, and patients whose comfort improved despite residual staining or complex disease.

WHY IT MATTERS

Long-term monitoring needs parallel tracks for symptoms, function, ocular-surface signs, safety, and the specific mechanism being treated.


Outcome that most supports continuing long-term treatment

08

ADVANCED-CARE BARRIERS

Adherence and affordability remain the practical ceiling on advanced DED management

49.1% select poor patient adherence as the biggest barrier to advanced DED management.

CONTEXT

Advanced DED care can involve multiple prescriptions, procedures, repeated visits, lid therapy, biologic products, contact lenses, or long-term maintenance. The treatment burden grows at the same time that the disease burden is already high.

WHAT THE DATA SHOWS

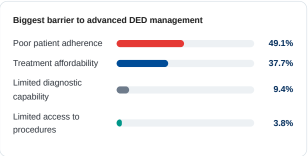
The survey shows that implementation barriers outweigh technology barriers. Even when clinicians can identify a mechanism and know what they would like to do next, adherence and affordability can determine whether the plan is realistic.

THE INSIGHT

The open comments include direct examples of insurance not covering advanced therapies or preservative-free medications, forcing alternative plans despite clinical rationale.

WHY IT MATTERS

Access and adherence are not administrative footnotes; they are variables that can alter the clinical outcome and should be addressed as part of treatment selection.


Biggest barrier to advanced DED management

09

WHAT COMES NEXT

Personalized treatment algorithms lead the future vision for dry eye care

45.3% select personalized DED treatment algorithms as the development most likely to transform care.

CONTEXT

The preference for personalization fits the rest of the survey. Mixed mechanisms dominate, persistent symptoms have several plausible next steps, and refractory subgroups need different strategies. A more useful future tool would therefore help match phenotype, severity, comorbidity, signs, symptoms, prior response, and patient constraints to the next intervention.

WHAT THE DATA SHOWS

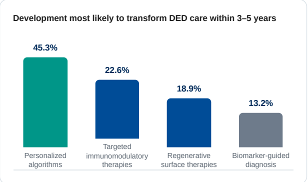
TFOS DEWS III also points toward more etiologic subclassification and notes future opportunities including artificial intelligence and biomarker testing. The evidence base will still need to determine when such tools improve decisions rather than simply add data.

THE INSIGHT

The survey does not suggest that algorithms should replace clinical judgment. It suggests a need to make complex, multimodal DED decisions more consistent and individualized.

WHY IT MATTERS

The future value of personalization will be measured by whether it reduces trial-and-error while keeping treatment practical for the patient.


Development most likely to transform DED care within 3–5 years

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OPEN RESPONSE LENS

Real cases show that persistent dry eye often becomes a search for the driver that the first treatment did not address

Respondents were asked to describe challenging DED cases with persistent symptoms, what changed their management approach, and what remained unresolved. The comments were not formally coded, so the themes below are qualitative rather than prevalence estimates.

Meibomian disease remained difficult even after treatment

Several comments describe persistent meibomitis or meibomian gland dysfunction despite standard lid care, prescription therapy, or IPL, with clinicians changing the plan toward more targeted gland-directed management.

Autoimmune and systemic disease changed the ocular-surface context

Responses mention autoimmune disease, GVHD, Graves-related disease, checkpoint-inhibitor exposure, and other systemic contexts in which ocular-surface inflammation or tissue damage required broader thinking than routine lubrication.

Symptoms and signs did not always improve together

Some clinicians described improved staining or surface appearance with persistent symptoms, while others reported partial comfort gains despite ongoing objective abnormalities. That mismatch complicated decisions about whether to continue, escalate, or reassess.

Neuropathic symptoms required a different pathway

A post-refractive case with neuropathic keratitis illustrates how severe symptoms can persist despite efforts to improve tear production, shifting management toward neurosensory pain strategies and scleral-lens support.

Access and insurance restrictions changed treatment

Multiple comments describe coverage problems for newer or preservative-free therapies. In those cases, the clinically preferred plan was modified because the patient could not obtain or sustain it.

Reassessment sometimes revealed a different driver

Comments include Demodex/blepharitis, medication-related ocular-surface disease, environmental exposure, and toxic injury. Improvement followed when management shifted toward the newly recognized cause rather than simply adding more dry-eye therapy.

PEOPLE ALSO ASK · TOPIC FAQ

Frequently asked questions about dry eye disease diagnosis and management.

What is dry eye disease and how is it diagnosed?

TFOS DEWS III defines dry eye as a multifactorial, symptomatic disease involving loss of tear-film and/or ocular-surface homeostasis. Diagnosis combines symptoms with objective evidence such as tear-film instability, hyperosmolarity, or ocular-surface staining, while excluding mimicking conditions.

What is mixed-mechanism dry eye?

Mixed-mechanism dry eye describes patients in whom more than one etiologic driver contributes to disease, such as tear-film deficiency together with meibomian gland dysfunction, ocular-surface inflammation, eyelid abnormalities, or neurosensory factors. Management is therefore usually phenotype- and driver-directed rather than based on one treatment alone.

How is meibomian gland dysfunction related to dry eye disease?

Meibomian gland dysfunction can destabilize the tear film by reducing or altering its lipid layer, increasing evaporation and ocular-surface stress. Lid hygiene, warming, gland-directed procedures, and other therapies may be considered according to the individual presentation.

When is anti-inflammatory treatment considered in dry eye disease?

Anti-inflammatory therapy may be considered when ocular-surface inflammation is an important disease driver or when symptoms persist despite basic tear-support measures. Choice of treatment depends on the clinical phenotype, severity, contraindications, access, and response to prior therapy.

Why can dry eye symptoms persist despite treatment?

Persistent symptoms can reflect incomplete adherence, ongoing inflammation, untreated meibomian gland dysfunction, environmental or medication-related triggers, autoimmune disease, neurosensory abnormalities, or a mismatch between the presumed mechanism and the actual disease drivers.

What is neuropathic ocular pain in dry eye disease?

Neuropathic ocular pain occurs when abnormal sensory processing contributes to ocular discomfort that may be disproportionate to visible surface findings. It requires careful differential assessment because standard tear-focused therapy alone may not fully address the symptoms.

OPHTHALMOLOGY PRACTICE LENS

What this means at four decision points in the dry-eye consultation.

The survey suggests that DED management becomes more efficient when each follow-up visit clarifies whether the problem is phenotype, inflammation, meibomian function, adherence/access, or a mismatch between symptoms and signs.

1. Reconfirm the active drivers

Phenotype before escalation: review tear-film deficiency, MGD, ocular-surface damage, inflammation, medications, systemic disease, and neurosensory features rather than assuming the original mechanism is still dominant.

2. Separate nonresponse from nonuse

Check adherence, tolerability, technique, duration, cost, and access before concluding that a therapy has biologically failed.

3. Track symptoms and signs in parallel

Do not rely on one metric. Compare patient-reported improvement, function, staining, tear-film stability, and the specific mechanism being treated.

4. Recognize refractory subgroups early

Autoimmune disease, severe aqueous deficiency, refractory MGD, and neuropathic ocular pain may need earlier multimodal or specialist-level strategies rather than repeated first-line cycling.

CLOSING PERSPECTIVE

The hardest dry-eye cases are rarely asking for “more treatment”; they are asking for a better match between mechanism, burden, and what the patient can sustain.

The survey shows a coherent management story. Mixed mechanisms dominate. Ocular-surface staining anchors diagnosis for many clinicians. Preservative-free tears commonly start care. But once symptoms persist, the pathway branches quickly into inflammation, meibomian gland treatment, reassessment, and advanced ocular-surface strategies.

The strongest friction is not purely biological. Adherence is the leading explanation for initial failure and the leading barrier to advanced care, while affordability is close behind. These are clinical realities because a therapy that cannot be obtained, tolerated, or used consistently cannot deliver its intended effect.

The data also highlight the importance of refractory subgroups and symptom-sign mismatch. Autoimmune disease, severe aqueous deficiency, MGD, neuropathic pain, medication toxicity, and access barriers can all change what “success” means and how long a patient may need multimodal care.

That helps explain why personalized treatment algorithms lead the future-development question. The value is not automation for its own sake. It is the possibility of reducing trial-and-error by connecting phenotype, objective findings, symptom burden, comorbidity, prior response, and real-world feasibility to a clearer next decision.

Phenotype again when the response is incomplete

Persistent DED can reveal a second driver that was not dominant at the first visit.

Treat adherence and access as clinical variables

Confirm that the patient can obtain, tolerate, and sustain the recommended plan.

Use symptoms and signs together

Improvement in one domain does not guarantee that the patient’s burden has resolved.

Escalate according to the driver, not the visit count

Inflammation, MGD, autoimmune disease, aqueous deficiency, and neuropathic pain do not require the same next step.

Help future ocular-surface insights reflect the complexity of real-world dry eye care.

Join MDForLives to share clinical perspectives on DED diagnosis, phenotype, treatment response, meibomian gland dysfunction, adherence, and personalized eye care.

Join the Panel

REFERENCES AND DATA SOURCE

1. **MDForLives internal survey report.** All Markets Report for SGID 9008491, Project: Engagement Campaign | Ophth Insight Survey_Oct. 61 survey records, 83.6% completion rate. Country metadata: USA · UK · Canada · Italy · France · Germany. Substantive DED questions were answered by 53 respondents.
2. **TFOS DEWS III: Diagnostic Methodology.** American Journal of Ophthalmology, 2025. Source
3. **TFOS DEWS III: Management and Therapy.** American Journal of Ophthalmology, 2025. Source
4. **TFOS DEWS III: Executive Summary.** American Journal of Ophthalmology, 2025. Source

External sources provide clinical context. Survey percentages represent MDForLives respondent selections and are reported separately from external evidence.

MDForLives

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The Global Pulse of Healthcare

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