Research Article

Dry Eye Disease: Current Concepts in Diagnosis and Management—A Narrative Review

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Abstract

Dry eye disease (DED) is a common, multifactorial ocular surface disorder characterized by loss of tear film homeostasis, resulting in ocular discomfort, visual disturbances, and impaired quality of life. This review provides an overview of the classification, epidemiology, risk factors, pathophysiology, clinical manifestations, diagnostic evaluation, and management of DED. The disease is broadly classified into aqueous-deficient and evaporative dry eye, although both mechanisms frequently coexist. Tear film instability, hyperosmolarity, ocular surface inflammation, epithelial damage, and neurosensory abnormalities play important roles in disease development and progression. Several factors, including ageing, hormonal changes, systemic diseases, prolonged digital screen use, environmental conditions, medications, and contact lens wear, contribute to its occurrence. Clinical diagnosis requires a comprehensive evaluation of symptoms and ocular surface findings using tests such as Schirmer's test, tear breakup time, ocular surface staining, and tear osmolarity assessment. Management involves patient education, lifestyle modifications, artificial tears, anti-inflammatory treatment, and targeted therapies according to disease severity and underlying causes. Emerging technologies, including artificial intelligence, advanced ocular imaging, and tear biomarker analysis, may improve diagnostic accuracy and support individualized treatment. A better understanding of the underlying disease mechanisms and the development of effective therapeutic strategies are essential for improving long-term outcomes in patients with DED.

Keywords: Dry eye disease, tear film instability, ocular surface inflammation, tear hyperosmolarity, Schirmer test, tear breakup time, diagnosis, management.

Introduction

Dry eye disease (DED) is a common ocular surface disorder that is multifactorial and is associated with disruption of normal tear film and ocular surface homeostasis. It causes symptoms including dryness, burning sensation, foreign body sensation, fluctuating vision, photophobia and ocular discomfort. The dry eye disease develops due to complex interactions of the tear film, cornea, conjunctiva, lacrimal gland, meibomian gland, ocular surface immune system and sensory nerves. [1]

The way that DED is currently understood has advanced beyond seeing it simply as a disorder characterised by insufficient tear production and now involves the recognition that it is a self-sustaining inflammatory and neurosensory disorder. Instability of the tear film and excessive evaporation can cause the osmolarity of the tears to rise, thereby damaging the epithelium of the ocular surface and triggering inflammatory pathways; meanwhile, the inflammation and damage to the epithelium themselves undermine the stability of the tear film, thus setting up a vicious cycle of the disease. [2]

Definition

According to the TFOS DEWS II definition, dry eye disease is described as a disease of the ocular surface that is multifactorial and characterized by the disruption of tear film homeostasis as well as ocular symptoms, with causes of the condition including tear film instability, hyperosmolarity, ocular surface inflammation, epithelial damage and

neurosensory abnormalities.

The definition stresses the fact that DED is multifactorial and identifies loss of tear-film homeostasis as the key concept. [1]

Major components of DED

- Loss of tear film homeostasis
- Tear film instability
- Tear hyperosmolarity
- Ocular surface inflammation and damage
- Neurosensory abnormalities
- Ocular symptoms such as discomfort and visual disturbance. [1]

Classification

The major pathophysiological classification is:

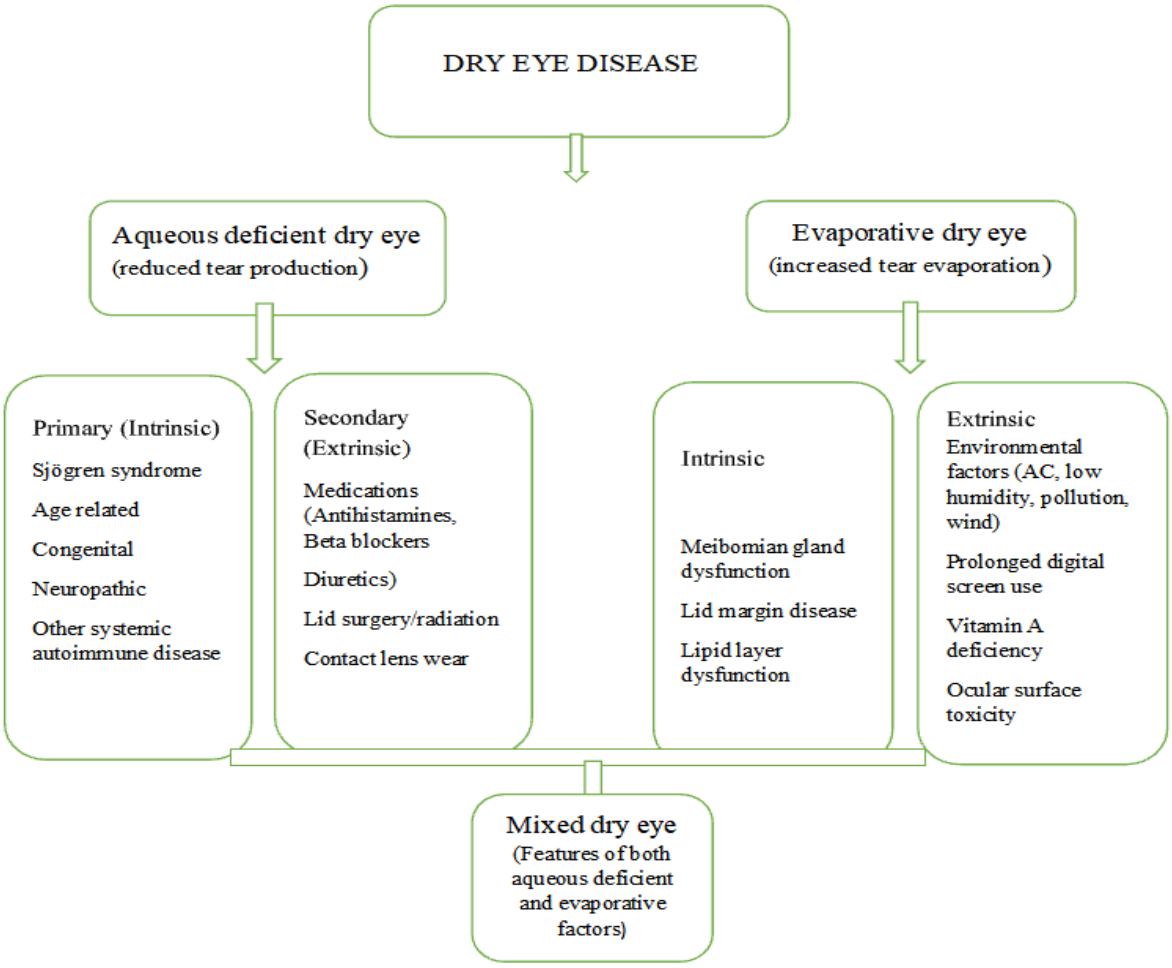


Fig1: Flowchart of dry eye disease classification

A. Aqueous deficient dry eye

It happens in cases where the secretion of aqueous tear is inadequate.

Examples:

- Lacrimal gland dysfunction
- Sjögren syndrome-associated dry eye
- Non-Sjögren aqueous-deficient dry eye

B. Evaporative dry eye

It happens when there is excessive evaporation of tears even though the aqueous secretion is relatively sufficient.

Common causes include:

- Meibomian gland dysfunction
- Incomplete blinking
- Lid margin disease
- Reduce blinking
- Environmental factors (AC, low humidity, pollution, wind, prolonged use of digital screen)
- Vitamin A deficiency

C. Mixed dry eye:

Since many patients suffer from both aqueous deficiency and increased evaporation, ADDE and EDE should be viewed as part of a continuous spectrum rather than as entirely distinct diseases. [1]

Epidemiology and Risk Factors

Dry eye disease is one of the most common problems that affect the surface of the eye all over the world. The number of people who have this condition changes a lot depending on how researchers define it. Different studies apply different diagnostic criteria, draw their samples from different populations and use either symptom-based or clinical definitions. A recent review showed that about 10 to 20 % of people aged 40 years or older have moderate to severe symptoms and get treatment, even though the prevalence varies from one population to another. [3]

Some of the Risk factors are as follows

1. Demographic factors
   - Geographic populations
   - Increasing age
   - Females
2. Ocular factors
   - Ocular surface disease
   - Abnormal or incomplete blinking
   - Meibomian gland dysfunction
   - Blepharitis
   - Contact lens wear
   - Previous surgery
3. Systemic disease
   - Diabetes mellitus
   - Migraine
   - Androgen deficiency
   - Some mental health conditions
   - Sjögren syndrome
   - Connective tissue diseases
4. Environmental factors

- Air pollution
- Smoke
- Low humidity
- Air conditioning
- Wind
- Prolonged visual tasks with less blinking

5. Lifestyle factors
   - Poor diet
   - Poor sleep quality
   - Cosmetic eye products
   - Excessive digital screen use
6. Medications
   - Hormonal medications
   - Antihistamines
   - Antidepressants
   - Certain topical ocular medications. [3 4]

Pathophysiology of Dry Eye Disease

Dry eye disease occurs when the normal tear film stability and the ocular surface integrity are disrupted. The process is complicated. It encompasses tear film instability, tear osmolarity, inflammation, epithelial damage, and changes in the function of the nerves of the ocular surface. The tear film usually gives lubrication, sustenance and protection for the cornea and conjunctiva. A reduction in tear production or an increase in tear evaporation causes inconsistency of the tear film, which breaks down more quickly. There are many factors leading to breakdown of the tear film, such as meibomian gland dysfunction, reduced blink rate, exposure to environmental conditions, and aqueous tear deficiency. When the tear film evaporates, the concentration of the electrolytes of the remaining tears rises, and this leads to tear hyperosmolarity. The hyperosmolarity causes stress and damage to the corneal and conjunctival epithelial cells at the same time triggers the release of inflammatory mediators and activating the inflammatory mechanism of the ocular surface in motion.

The ocular surface inflammation caused as a result damages the epithelium. These inflammatory mediators influence goblet cells, epithelial cells and the meibomian glands, leading to a decrease in mucin production and thus aggravating the quality and stability of the tear film. As a result, a self-perpetuating vicious cycle is set up in which instability of the tear film causes hyperosmolarity, which in turn promotes inflammation and epithelial damage, thereby further increasing tear film instability.

Long-standing inflammation and damage to the epithelial damage can also impact the sensory nerves of the cornea. Problems in corneal nerves may lead to abnormal sensation in the eyes, including burning, foreign body sensation, and eye pain. Sometimes neurosensory problems may lead to abnormal symptoms even with no obvious clinical findings. [5, 6]

- **Instability of tear film**

A normal tear film produces a smooth, well-moistened, and transparent ocular surface.

In dry eye disease, there is decreased tear film stability. Rapid tear film breakup occurs between blinks. Increased evaporation can occur. Lipid layer deficiency due to meibomian gland dysfunction increases evaporation. Abnormalities of mucins and goblet cells cause poor wetting of the ocular surface. There is corneal and conjunctival epithelial damage. [5]

- **Hyperosmolarity of Tears**

Hyperosmolarity implies that there is an abnormal elevation in the solute concentration of the tear film.

It occurs primarily due to

- High tear evaporation
- Low aqueous tear secretion
- Low tear quantity
- High concentration of electrolytes and other solutes

Hyperosmolar tears cause osmotic stress in corneal and conjunctival epithelial cells, which stimulate stress pathways and leads to inflammatory cytokine and matrix metalloproteinase (MMP) production. [6,7]
The ramifications include

- Epithelial cell injury
- Heightened inflammatory activation
- Goblet-cell abnormality
- Reduced epithelial barrier function
- Enhanced ocular-surface sensitivity
- Further tear-film instability

- Ocular surface inflammation

Inflammation plays a significant role in the pathophysiology of dry eye disease (DED). Tear film hyperosmolarity and epithelial stress activate inflammatory pathways on the ocular surface, leading to the release of inflammatory mediators and the recruitment and activation of immune cells. Both innate and adaptive immune responses contribute to this inflammatory process.
The major inflammatory responses include:

- Activation of ocular surface epithelial cells.
- Release of pro-inflammatory cytokines and chemokines.
- Activation and maturation of dendritic cells.
- Recruitment and activation of T lymphocytes.
- Increased expression of matrix metalloproteinases (MMPs).
- Disruption of epithelial tight junctions.
- Impairment of conjunctival goblet cell function.

These inflammatory changes contribute to corneal and conjunctival epithelial damage, disruption of the ocular surface barrier, and increased tear film instability, further aggravating the progression of DED . [5, 6, 7]

- Neural and Epithelial Dysfunction

Corneal sensory nerves play an essential role in maintaining ocular surface homeostasis by regulating tear secretion, blinking reflexes, epithelial integrity, and ocular surface sensation. In DED, inflammation, tear hyperosmolarity, and epithelial injury can impair corneal nerve function, resulting in altered sensory responses.
The major manifestations of neural dysfunction include:

- Reduced corneal sensitivity.
- Burning sensations and ocular discomfort.
- Foreign body sensation.
- Altered reflex tear secretion.
- Abnormal blinking responses.
- Neuropathic ocular pain in some patients.

Neurosensory abnormalities may also contribute to a mismatch between clinical signs and patient-reported symptoms, where individuals experience significant discomfort despite minimal visible ocular surface damage.
In addition to neural dysfunction, DED causes structural and functional changes in the corneal and conjunctival i

- Increased epithelial cell apoptosis.
- Disruption of epithelial tight junction integrity.
- Reduced epithelial barrier function.

- Decreased goblet cell density and function.
- Alterations in mucin secretion and the epithelial glycocalyx.
- Increased corneal epithelial staining.
- Development of ocular surface irregularities.

Collectively, neural and epithelial dysfunction compromise ocular surface protection, reduce tear film stability, and contribute to persistent discomfort and disease progression. [7]

Etiology

Dry eye disease (DED) develops from reduced tear production or excessive tear evaporation, leading to tear-film instability and ocular surface damage.

Main mechanisms that are associated with dry eye disease etiology are:

• Decreased tear production - the eye produces insufficient tear production volume.

• Increased tear evaporation - tears evaporate more rapidly from the eye.

• Tear film instability - the improper functionality of the tear film

• Lipid layer abnormality - an abnormal lipid layer allows tears to evaporate more rapidly and leads to dryness and damage to the eye.

• Ocular surface inflammation - inflammation of the ocular surface can decrease tear secretion and produce further damage to the eye surface. [8,9]

Contributing factors

Dry eye disease (DED) is a multifactorial condition influenced by demographic, systemic, environmental, lifestyle, and ocular factors. Several conditions may increase the risk of developing DED or aggravate existing symptoms.

The major contributing factors include:

- **Ageing:** Tear production and tear film function may decline with increasing age, particularly after 50 years.
- **Gender and hormonal changes:** Women, especially during menopause and post menopause, are more susceptible to DED due to hormonal changes that affect lacrimal and meibomian gland function.
- **Autoimmune and systemic diseases:** Conditions such as rheumatoid arthritis, Sjögren's disease, and other systemic disorders are associated with an increased risk of DED.
- **Smoking:** Exposure to cigarette smoke can irritate the ocular surface, increase oxidative stress, and contribute to dry eye symptoms.
- **Prolonged contact lens wear:** Long-term contact lens use may alter tear film stability and corneal sensitivity, contributing to ocular discomfort.
- **Medications:** Certain medications, including antihistamines, antidepressants, and some systemic drugs, may reduce tear secretion.
- **Refractive surgery:** Procedures such as LASIK and PRK may cause temporary or persistent dry eye symptoms due to changes in corneal nerve function.
- **Prolonged screen use and visual tasks:** Extended computer use, television viewing, reading, and driving may reduce blink frequency or blink completeness, increasing tear evaporation.
- **Environmental conditions:** Low humidity, extreme temperatures, air conditioning, and increased airflow can accelerate tear evaporation and worsen ocular surface irritation.
- **Air travel:** Low humidity inside aircraft cabins may increase tear evaporation and aggravate symptoms.
- **Dietary factors:** Low dietary intake of omega-3 fatty acids has been investigated as a possible contributing factor, although the evidence remains inconsistent.
- **Other factors:** Certain systemic illnesses and infections have been investigated for possible associations with DED, although relationships such as that with Helicobacter pylori infection remain uncertain. [8, 9]

Role of Digital Device Use and Environmental Factors

Role of digital device use

- It is becoming increasingly acknowledged that the long-term use of computer, smartphone, tablets and other digital screens is a significant element of lifestyle which leads to dry eye disease (DED). Continuous use of digital screen changes normal blink pattern, specifically by lowering the frequency of blinks and increasing incomplete blink rate. This in turn leads to greater evaporation and instability of the tear film, resulting in dry eyes, a burning sensation, irritation and varying vision. [10]
- The greater the daily use of digital devices, the higher the risk of DED. Reviews have pointed out an association between extended use of visual display terminals and DED, with certain studies finding that symptoms can appear even at relatively low levels of daily exposure.[11]
- A review carried out among young people who use screens found that daily screen use of more than 4 to 5 hours and bad ergonomic conditions were linked to a higher number of symptoms of digital eye strain.
- The viewing conditions in a digital environment are also important; the angle at which you view the screen, the distance at which you're working, the position of the screen, glare and the need for prolonged concentration can all affect blinking and the exposure of the surface of the eye. It follows that improving the working environment, promoting frequent breaks and helping to correct blinking habits may thus aid in reducing symptoms of digital eye strain and dry eyes.[12]

Environmental factor

- Environmental factors may exacerbate DED by increasing tear evaporation and disturbing ocular-surface homeostasis. Low humidity, high temperature, wind or air movement, air conditioning, air pollution and airborne allergens are important factors.[13]
- Air-conditioned environments may be especially relevant for digital device users, since prolonged screen viewing is often accompanied by indoor exposure. One study found that people with digital eye strain spent more time indoors with air conditioning and also had a longer duration of computer use. [14]

Clinical Manifestations

The clinical manifestations of DED include a wide range of symptoms and observable signs. The severity of reported symptoms does not always correspond with the degree of ocular surface damage.

Symptoms

Common symptoms of DED include:

- Ocular dryness.
- Burning or stinging sensation.
- Grittiness or foreign body sensation.
- Eye irritation.
- Sensitivity to light (photophobia).
- Excessive watering or reflex tearing.
- Ocular discomfort or pain.
- Redness.
- Eye fatigue.
- Fluctuating or blurred vision and reduced visual quality.

Signs

Common clinical signs include:

- Tear film instability: Assessed using tear breakup time (TBUT).
- Reduced tear production: Evaluated using the Schirmer test.
- Corneal epithelial staining: Detected using fluorescein and other appropriate vital dyes.
- Conjunctival staining: Evaluated using lissamine green or rose bengal.
- Corneal sensitivity abnormalities: Reduced or altered corneal sensation.

- Corneal nerve abnormalities: Structural or functional changes in corneal nerves.
- Ocular surface inflammation: Characterized by conjunctival redness and inflammatory changes.
- Increased tear film osmolarity: Associated with disruption of tear film homeostasis. [15]

Diagnostic Evaluation

The diagnosis of DED requires a comprehensive assessment of patient symptoms, clinical history, tear film characteristics, and ocular surface integrity. A combination of subjective and objective examinations is used to establish the diagnosis and identify contributing factors.

Patient History and Symptom Assessment

A detailed patient history is an essential first step in evaluating suspected DED. The assessment includes the onset, frequency, duration, and severity of symptoms, along with possible environmental, systemic, and lifestyle-related risk factors.

Common symptoms assessed during history taking include dryness, burning, irritation, photophobia, gritty sensation, eye fatigue, and ocular pain.

Standardized symptom questionnaires, such as the Ocular Surface Disease Index (OSDI), may also be used to assess symptom severity and its impact on daily activities.

Ocular Surface Examination

The ocular surface is primarily examined using a slit-lamp biomicroscope to evaluate structural abnormalities and signs of inflammation.

The examination includes assessment of:

- Corneal surface and epithelial integrity.
- Conjunctival appearance.
- Tear film characteristics.
- Eyelids and lid margins.
- Meibomian gland openings and secretions.
- Signs of epithelial damage.
- Ocular surface inflammation. [15]
- Tear Film Assessment

Tear film assessment is essential for evaluating tear production, stability, and overall ocular surface function. The commonly performed tests include the Schirmer test and tear breakup time.

1.Schirmer Test

The Schirmer test, first described in 1903, is a commonly used clinical method for estimating aqueous tear secretion. It measures the length of wetting of a standardized filter paper strip over a specified period.

i.Schirmer I Test

The Schirmer I test is generally performed without topical anesthesia and measures tear secretion, including basal and reflex components.

Procedure:

- A Schirmer strip is placed in the temporal portion of the lower conjunctival fornix.
- The patient is instructed to close the eyes gently or follow the selected standardized protocol.
- After five minutes, the strip is removed.
- The length of wetting is measured in millimetres.

Wetting greater than 10 mm in five minutes is generally considered adequate, whereas lower values may suggest aqueous tear deficiency. Interpretation depends on the testing protocol.

ii. Schirmer II Test

The Schirmer II test evaluates reflex tear secretion following stimulation of the nasal mucosa.

- Topical ocular anaesthesia is typically administered.
- Reflex tearing is stimulated using a cotton-tipped applicator in the nasal cavity.

- Tear secretion is measured using the Schirmer strip over five minutes.

It is important to distinguish Schirmer II from the anaesthetized Schirmer test performed without nasal stimulation, which is intended to estimate basal tear secretion.

2.Tear Breakup Time (TBUT)

Tear breakup time is commonly used to assess tear film stability. It measures the interval between the last complete blink and the first appearance of disruption in the tear film.

Procedure:

- A small amount of fluorescein dye is instilled into the eye.
- The patient is instructed to blink naturally to distribute the dye.
- The patient is then asked to keep the eyes open without blinking.
- The interval between the last blink and the first appearance of a dark spot in the fluorescein-stained tear film is recorded.

Interpretation

A shorter TBUT indicates reduced tear film stability. Diagnostic thresholds differ by method. Current TFOS DEWS III guidance identifies a non-invasive breakup time below 10 seconds or a fluorescein breakup time below 5 seconds as evidence of tear film instability. [16,17]

Table 1. Clinical Interpretation of Common Tear Film Tests

Test	Generally adequate finding	Finding suggestive of tear deficiency or instability
Schirmer I (without anaesthesia)	Greater than 10 mm/5 min	5–10 mm borderline or reduced; 5 mm or less suggests marked aqueous deficiency
Schirmer II (nasal stimulation)	Protocol-dependent	Reduced reflex secretion based on the standardized protocol
Non-invasive TBUT	10 seconds or longer	Less than 10 seconds
Fluorescein TBUT	Method-dependent	Less than 5 seconds under the current TFOS diagnostic protocol

Ocular Surface Staining

Ocular surface staining is an important diagnostic procedure used to identify corneal and conjunctival epithelial damage associated with DED.

i. Fluorescein staining: Fluorescein is commonly used to detect corneal epithelial defects, superficial punctate epithelial staining, and areas of epithelial disruption.

ii. Rose Bengal staining: Rose Bengal stains ocular surface cells with inadequate mucin protection and can highlight areas of epithelial damage. However, it may cause ocular discomfort and has greater epithelial toxicity than Lissamine green.

iii. Lissamine green staining: Lissamine green is used to identify damaged or inadequately protected ocular surface cells, particularly in the conjunctiva and lid margins. It provides staining patterns similar to rose Bengal but is generally better tolerated. [16]

Tear Osmolarity

Tear osmolarity is a quantitative parameter used to evaluate tear film homeostasis. Increased tear osmolarity is associated with tear film instability, ocular surface inflammation, and epithelial damage.

A tear osmolarity value of 308 mOsm/L or greater in either eye, or an interocular difference greater than 8 mOsm/L, may support a diagnosis of DED when measured using the device for which these thresholds were established.

Some studies have also investigated higher thresholds, including approximately 315 mOsm/L. However, tear osmolarity varies between individuals, between eyes, and across measurements. Therefore, it should be interpreted in conjunction with other clinical tests rather than as an isolated diagnostic measure. [16]

Tear Biomarkers

Tear biomarkers provide information about inflammatory and biochemical changes associated with DED and may assist in disease assessment.

Important inflammatory biomarkers include:

- Matrix metalloproteinase-9 (MMP-9).
- Interleukin-1 receptor antagonist (IL-1Ra).
- Interleukin-6 (IL-6).
- Interleukin-8 (IL-8).
- Interleukin-17 (IL-17).

DED is often associated with increased inflammatory mediator activity, although biomarker concentrations vary depending on disease characteristics and testing methods. Biomarker testing may support clinical evaluation, but routine use is limited by cost, accessibility, and variability in diagnostic performance. [16]

Emerging Diagnostic and Therapeutic Approaches

Advances in diagnostic technologies and treatment strategies aim to improve the early detection, classification, and management of DED.

Emerging diagnostic approaches include artificial intelligence (AI) and machine learning techniques for analyzing clinical data, advanced ocular surface imaging to identify structural changes, and improved tear film analysis to assess tear quality and stability. Tear biomarker analysis may provide additional information about inflammatory activity and ocular surface abnormalities. Genetic profiling and sensor-based contact lens technologies are also being investigated for their potential roles in disease detection and monitoring.

Therapeutic approaches include anti-inflammatory medications, tear secretion stimulants (secretagogues), autologous serum eye drops, punctal plugs, and thermal pulsation therapy for selected patients. More experimental strategies, including gene therapy, stem cell therapy, and microbiome-based treatments, remain under investigation.

Individualized treatment based on disease mechanisms, combined with appropriate lifestyle and environmental modifications, may improve symptom control and long-term ocular surface health. [17]

Differential Diagnosis of Dry Eye disease

Dry eye disease (DED) presents with familiar clinical features that could overlay with other ocular surface diseases; hence, the importance of correctly finding and differentiating it from other presentations with similar symptoms. Various associated factors need to be considered, and the diagnosis is made based on symptoms and examination rather than relying on one specific test. There are several essential differential diagnoses of DED, which include:

i. Blepharitis: characterized by eye irritation, burning sensation, redness, discomfort, and vision fluctuation; thus, it presents similarly to DED. Diagnosis of this condition can be made by examination of the eyelids, lashes and front of the eye. [18]

ii. Meibomian gland dysfunction: this determination regularly exists with evaporative instability of the tear film and often overlays with DED; hence, both should be considered in the differential diagnosis. The evaluation of the meibomian glands, margin, and secretion may be helpful in diagnosing the situation. [18]

iii. Allergic or infectious conjunctivitis: characterized by redness, irritation, burning, and discomfort like those of DED. Hence, inspection of the eye front and identification of any associated conjunctival inflammation are critical. [18]

iv. Exposure related ocular surface disease: This diagnosis is highly related to that of DED due to the exposure-tear film instability association. Thus, evaluating the eyelid function and exposure may help identify the condition. [18]
v. Assessment of tear-film homeostasis: according to recent research, it is essential to considered a combination of factors when it comes to diagnosing DED. For example, checking how stable the tear film is or how salty it is, along, with looking at the eye surface and the symptoms someone has given a diagnosis than just looking at one thing. [19]
vi. Sjogren syndrome: it is important to think about this diagnosis when someone has bad dry eye symptoms that are caused by not enough tears. This is because DED often shows similar signs and symptoms. The signs are dry eye, severe deficiency of the tear film, inflammatory response of the eye surface, punctate epithelial erosions and, in advanced cases, corneal change. [20,21]

Management of Dry Eye Disease

The treatment of dry eye disease (DED) is focused on the relief of symptoms, restoration of the tear film homeostasis, improvement of the ocular-surface health, and management of the underlying causes of the tear-film dysfunction. Due to the multifactorial nature of the disease, the management plan should address the patients' needs according to the severity, clinical manifestations, and inciting factors. DED management includes patient education and lifestyle modification, environmental and behavioural measures, artificial tears, anti-inflammatory treatment, management of meibomian gland dysfunction (MGD), tear conservation, and advanced therapeutic intervention. [21,22]
i.Education and lifestyle modification - Patients need to understand that this condition is long-lasting and will require ongoing care. They should know how important it is to follow the treatment plan and to keep all scheduled appointments. In some situations, patients should be told about the things that happen when they drink enough water, get enough rest and take breaks when they are looking at screens for a long time. Also, patients should be told not to touch their eyes and to take care of their eyelids, especially if they have blepharitis or MGD. When DED is connected to Sjogren syndrome, patients might need help from more than one doctor so that both the eye problems and the body-wide issues can be treated together. [20,21]
ii.Behavioural measures - To decrease tear evaporation and eye irritation patients, patient must should avoid places with air conditioning, fans, smoke or a lot of dust. Other helpful things include keeping the air in the house right and wearing eye protection when its windy. Also, when people use computers or phones for a time they should take breaks and make sure they blink completely. This helps the tears spread out evenly and stops the eyes from getting dry. If the symptoms don't go away these steps should be used along, with the medical care. [21]
iii.Artificial tears and ocular lubricants – Artificial tears (comprising aqueous, lipid contain, get and ointment formulations) together with ocular lubricants may be employed to supplement the tear film, deliver lubrication to the ocular surface, and alleviate symptoms of burning, irritation, and foreign body sensation. Lipid-based formulations may be warranted in evaporative Dry Eye Disease associated with MGD. Owing to the danger of preservative toxicity, preservative-free eye drops may be favoured in patients who administer drops frequently. Ocular lubricant prescribed according to patients' severity of the DED. [22,23]
iv.Other advanced treatments - Patients who have bad or hard-to-treat dry eye disease may need to see a specialist and think about other types of treatments. These might include eye drops made from their blood or other types of tears made from blood. Some patients might also find that special contact lenses or big contact lenses called lenses help them. The right treatment to use depends on how bad the disease's how well the patient has responded to earlier treatments and what the specialist says. Patients who have eye disease that is linked to Sjogren syndrome may need a specialist to check for other problems, with their eyes. The treatment should focus on getting tears and making sure the eyes work better. It should also try to stop damage from happening. If the patient has health problems that are linked to Sjogren syndrome those should be treated too. Specialists should be involved if the other health problems are serious. Specialists should be involved if the other health problems are serious. There is always. Improving ways to treat problems with the surface of the eye and dry eye. New

treatments should only be used in care if they have been shown to work and be safe. [23,24]

Challenges and Future Perspectives

There are challenges and future perspectives for diagnosing and managing dry eye disease (DED). Even though there have been advances in diagnosing and treating people with dry eye syndrome keeping dry eye disease (DED) under control for a long time is still difficult. Dry eye disease (DED) is a disease and how patients respond to treatment and how dry eye disease shows up can differ a lot, which makes management even harder. Also, most clinical signs do not always match the symptoms, which adds another problem for clinicians. [22,24]

A main challenge in managing eye disease (DED) is that treatment must be tailored to each person. Symptomatic treatments, like tears and eye lubricants, usually do not work well enough and only give quick relief. Because of this, the treatment plan should be customized to the part of eye disease (DED) and to the patient's tear-film problems. [21,24]

Another challenge with DED care is making sure people stay on the DED treatment plan for a time. Chronic diseases often need changes in habits and steady treatment, which can feel frustrating and inconvenient for patients. Understanding how high treatment costs, frequent doses and weak response, to treatment can make patients think about stopping therapy. Because of these problems, good counselling and setting patient expectations are key parts of successful DED treatment. [23,24]

Looking to the future, new treatment options that target inflammatory pathways and cellular processes will become increasingly important. Furthermore, the identification of biomarkers for the types of DED will enable clinicians to classify patients more accurately and to determine the most appropriate treatment regime. [24] Future potential, for the diagnosis and treatment of DED is closely linked to the development of diagnostic tests and a better understanding of the mechanisms underlying different forms of the disease. Finally, it should be emphasized that future research needs to be performed to evaluate long-term efficacy and safety of treatments and to conduct cost-effectiveness analyses comparing different treatment options. [21,23,24]

Conclusion

Dry eye disease is a complex and multifactorial ocular surface disorder that affects tear film stability, ocular surface integrity, and visual comfort. Its development involves interactions between reduced tear secretion, excessive tear evaporation, tear hyperosmolarity, inflammation, epithelial damage, and neurosensory abnormalities. Various demographic, systemic, environmental, and lifestyle-related factors contribute to disease onset and progression. [10,14,24] Early identification of these factors and a comprehensive clinical evaluation are important for accurate diagnosis and appropriate management. As no single diagnostic test can reliably identify all forms of DED, a combination of symptom assessment, tear film evaluation, and ocular surface examination is necessary.

The management of DED requires an individualized approach based on the underlying disease mechanisms, clinical severity, and patient-related factors. Conventional treatments, including artificial tears, lifestyle modifications, and anti-inflammatory medications, remain important, while advanced therapies may provide additional benefits in selected patients. Recent developments in ocular surface imaging, tear biomarker analysis, and artificial intelligence offer promising opportunities for improving diagnostic precision and treatment selection. However, further research is required to establish their clinical effectiveness, accessibility, and long-term benefits. Overall, early diagnosis, appropriate treatment, patient education, and regular follow-up are essential for reducing symptoms, maintaining ocular surface health, and improving the quality of life of individuals affected by dry eye disease.

Conflict of Interest

The authors declare that they have no conflict of interest related to this study, its findings and the publication of this manuscript.

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References

1. Craig, J. P., Nichols, K. K., Akpek, E. K., Caffery, B., Dua, H. S., Joo, C. K., Liu, Z., Nelson, J. D., Nichols, J. J., Tsubota, K., & Stapleton, F. (2017). TFOS DEWS II definition and classification report. The Ocular Surface, 15(3), 276–283. https://doi.org/10.1016/j.jtos.2017.05.008
2. Bron, A. J., de Paiva, C. S., Chauhan, S. K., Bonini, S., Gabison, E. E., Jain, S., Knop, E., Markoulli, M., Ogawa, Y., Perez, V., Uchino, Y., Yokoi, N., Zoukhri, D., & Sullivan, D. A. (2017). TFOS DEWS II pathophysiology report. The Ocular Surface, 15(3), 438–510. https://doi.org/10.1016/j.jtos.2017.05.011
3. Britten-Jones, A. C., Wang, M. T. M., Samuels, I., Jennings, C., Stapleton, F., & Craig, J. P. (2024). Epidemiology and risk factors of dry eye disease: Considerations for clinical management. Medicina, 60(9), Article 1458. https://doi.org/10.3390/medicina60091458
4. Qian, L., & Wei, W. (2022). Identified risk factors for dry eye syndrome: A systematic review and meta-analysis. PLOS ONE, 17(8), Article e0271267. https://doi.org/10.1371/journal.pone.0271267
5. Pflugfelder, S. C., & de Paiva, C. S. (2017). The pathophysiology of dry eye disease: What we know and future directions for research. Ophthalmology, 124(11S), S4–S13. https://doi.org/10.1016/j.ophtha.2017.07.010
6. Messmer, E. M. (2022). Pathophysiology of dry eye disease and novel therapeutic targets. Experimental Eye Research, 217, Article 108944. https://doi.org/10.1016/j.exer.2022.108944
7. Mantelli, F., Massaro-Giordano, M., Macchi, I., Lambiase, A., & Bonini, S. (2013). The cellular mechanisms of dry eye: From pathogenesis to treatment. Journal of Cellular Physiology, 228(12), 2253–2256. https://doi.org/10.1002/jcp.24398
8. Gayton, J. L. (2009). Etiology, prevalence, and treatment of dry eye disease. Clinical Ophthalmology, 3, 405–412. https://doi.org/10.2147/OPTH.S5555
9. Huang, R., Su, C., Fang, L., Lu, J., Chen, J., & Ding, Y. (2022). Dry eye syndrome: Comprehensive etiologies and recent clinical trials. International Ophthalmology, 42(10), 3253–3272. https://doi.org/10.1007/s10792-022-02320-7
10. Mehra, D., & Galor, A. (2020). Digital screen use and dry eye: A review. Asia-Pacific Journal of Ophthalmology, 9(6), 491–497. https://doi.org/10.1097/APO.0000000000000328
11. Fjærvoll, H., Fjærvoll, K., Magno, M., Moschowits, E., Vehof, J., Dartt, D. A., & Utheim, T. P. (2022). The association between visual display terminal use and dry eye: A review. Acta Ophthalmologica, 100(4), 357–375. https://doi.org/10.1111/aos.15049
12. Wolffsohn, J. S., Lingham, G., Downie, L. E., Huntjens, B., Inomata, T., Jivraj, S., Kobia-Acquah, E., Muntz, A., Mohamed-Noriega, K., Plainis, S., Read, M., Sayegh, R. R., Singh, S., Utheim, T. P., & Craig, J. P. (2023). TFOS Lifestyle: Impact of the digital environment on the ocular surface. The Ocular Surface, 28, 213–252. https://doi.org/10.1016/j.jtos.2023.04.004
13. Alves, M., Asbell, P., Dogru, M., Giannaccare, G., Grau, A., Gregory, D., Kim, D. H., Marini, M. C., Ngo, W., Nowinska, A., Saldanha, I. J., Villani, E., Wakamatsu, T. H., Yu, M., & Stapleton, F. (2023). TFOS Lifestyle Report: Impact of environmental conditions on the ocular surface. The Ocular Surface, 29, 1–52. https://doi.org/10.1016/j.jtos.2023.04.007
14. Talens-Estarelles, C., García-Marqués, J. V., Cerviño, A., & García-Lázaro, S. (2022). Dry eye-related risk factors for digital eye strain. Eye & Contact Lens, 48(10), 410–415. https://doi.org/10.1097/ICL.0000000000000923
15. Patel, S., Mittal, R., Kumar, N., & Galor, A. (2023). The environment and dry eye—Manifestations,

mechanisms, and more. Frontiers in Toxicology, 5, Article 1173683. https://doi.org/10.3389/ftox.2023.1173683
16. Salomon-Ben Zeev, M., Miller, D. D., & Latkany, R. (2014). Diagnosis of dry eye disease and emerging technologies. Clinical Ophthalmology, 8, 581–590. https://doi.org/10.2147/OPTH.S45444
17. Wolffsohn, J. S., Arita, R., Chalmers, R., Djalilian, A., Dogru, M., Dumbleton, K., Gupta, P. K., Karpecki, P., Lazreg, S., Pult, H., Sullivan, B. D., Tomlinson, A., Tong, L., Villani, E., Yoon, K. C., Jones, L., & Craig, J. P. (2017). TFOS DEWS II diagnostic methodology report. The Ocular Surface, 15(3), 539–574. https://doi.org/10.1016/j.jtos.2017.05.00
18. Narendra, K., Singh, S. K., Deepa, C. K., Meghana, S., Akanth, K. R., Manjushree, M., Rajasubramaniyan, D., Srinivasan, S., Murali, R., & Sowbhagya, H. N. (2025). Exploring new frontiers in dry eye disease: Treatments, mechanisms, and diagnostic innovations—A comprehensive review. Aspects of Molecular Medicine, 5, Article 100090. https://doi.org/10.1016/j.amolm.2025.100090
19. Herwig-Carl, M. C., & Loeffler, K. U. (2018). Differenzialdiagnosen der Benetzungsstörung [Differential diagnoses of dry eye syndrome and blepharitis]. Der Ophthalmologe, 115(2), 107–113. https://doi.org/10.1007/s00347-017-0631-7
20. Wolffsohn, J. S., Travé-Huarte, S., Stapleton, F., Downie, L. E., Schulze, M.-M., Guthrie, S., Stahl, U., Wang, M. T. M., & Craig, J. P. (2025). Relative importance of tear homeostatic signs for the diagnosis of dry eye disease. The Ocular Surface, 36, 151–155. https://doi.org/10.1016/j.jtos.2025.01.010
21. Aragona, P., Giannaccare, G., Mencucci, R., Rubino, P., Cantera, E., & Rolando, M. (2021). Modern approach to the treatment of dry eye, a complex multifactorial disease: A P.I.C.A.S.S.O. board review. British Journal of Ophthalmology, 105(4), 446–453. https://doi.org/10.1136/bjophthalmol-2019-315747
22. Jones, L., Downie, L. E., Korb, D., Benitez-del-Castillo, J. M., Dana, R., Deng, S. X., et al. (2017). TFOS DEWS II management and therapy report. The Ocular Surface, 15(3), 575–628.
23. Shahraki, T., Baradaran-Rafii, A., Ayyala, R., Arabi, A., Jarstad, J., & Memar, F. (2024). New advances in medical management of dry eye: Optimizing treatment strategies for enhanced relief. International Ophthalmology, 44(1), Article 49. https://doi.org/10.1007/s10792-024-02978-1
24. Salvetat, M. L., Pellegrini, F., D'Esposito, F., Musa, M., Tognetto, D., & Giglio, R. (2025). Emerging trends and management for Sjögren syndrome-related dry eye corneal alterations. Applied Sciences, 15(9), Article 5076.