



REVIEW

Dry Eye Disease: From Causes to Patient Care and Clinical Collaboration—A Narrative Review

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ABSTRACT

Dry eye disease (DED) is a common condition of the ocular surface that affects tens of millions

of people worldwide. It is often characterized by decreased tear production or increased evaporation, resulting in a wide range of signs and symptoms. This review provides a comprehensive analysis of the literature related to DED,

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detailing causes, diagnostic tests, and medical management. Several mechanisms contribute to the maintenance of the physiological integrity of the ocular surface, and their dysfunction may result in noticeable symptoms. Accurate diagnosis is therefore essential, even when physiological function is only minimally impaired or no clear pathological signs are present. The review emphasizes the importance of addressing the underlying causes through a combination of treatment options, lifestyle changes, and enhanced communication between patients and healthcare providers to break the cycle of inflammation and tear instability. It aims to raise awareness among patients, healthcare professionals, and researchers regarding the diagnosis and treatment of DED, while also highlighting recent advancements and future challenges in its management.

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Key Summary Points

Effective management of dry eye disease (DED) requires addressing key factors such as inflammation, tear film instability, and environmental or lifestyle contributors, emphasizing the need to target underlying chore mechanisms.

A combination of pharmacological treatments, lifestyle modifications, and personalized education is vital to breaking the cycle of inflammation and tear instability.

Therapeutic advances such as novel anti-inflammatory agents and tear-stabilizing technologies offer promising opportunities for more personalized and effective management of DED.

Healthcare providers play a critical role in educating patients about DED's chronic and fluctuating nature, promoting adherence to long-term care protocols and fostering active patient involvement.

Integrating expertise from ophthalmologists and other healthcare professionals enables tailored strategies that improve patient outcomes and quality of life.

INTRODUCTION

Dry eye disease (DED) is one of the most common ocular conditions worldwide, affecting tens of millions of people [1]. The prevalence of DED is higher in women than in men, although the differences become significant with increasing age, when the prevalence also increases [1]. This widespread condition not only affects visual function, but also significantly reduces the quality of life of those affected. The impact of DED goes beyond individual inconvenience, as it has a significant socio-economic impact, affecting productivity, healthcare costs, and the overall well-being of society [2]. DED is characterized by tear film instability, hyperosmolarity, and ocular surface inflammation resulting from a loss of homeostasis. Corneal nerve injury can cause neural plasticity and

peripheral and central sensitization, resulting in neuropathic ocular pain (NOP) [3]. In the early stages, symptoms may be transient and reversible, often accompanied by subclinical inflammation that can go unnoticed. However, if the ocular surface cannot re-equilibrate, more severe disease can develop, marked by chronic inflammation, persistent epithelial changes, and vision impairment [4]. Therefore, if left untreated, DED can become an irreversible chronic condition driven by reduced tear flow, increased evaporation, and repeated nerve stimulation. This creates a self-perpetuating cycle of inflammation that leads to treatment-resistant disease and permanent ocular surface damage [5]. The heterogeneity of DED presentations and the variability in disease progression pose significant clinical challenges. Treatment strategies must focus on restoring and maintaining ocular surface homeostasis to break the vicious cycle of the disease [6].

This narrative review, conceived by the Italian Dry Eye Consensus Group [7], analyses the importance of addressing the causes of DED through combined treatments and improved communication between patients and healthcare professionals, and aims to raise awareness of the importance of diagnosing and managing DED.

METHODS

A literature search was conducted using the PubMed database for studies and reviews published in English. Search criteria for publications included articles that focused on DED and addressed aetiopathogenesis, risk, chronicity and inflammation, diagnostic approach, pharmacological and non-pharmacological therapy, patient needs, compliance and quality of life, and the doctor–patient relationship. No restrictions were set for the date of publication. Articles considered most relevant and representative of a balanced view of the issue by the Italian Dry Eye Consensus Group were selected and included. This article is based on previously conducted studies and does not contain any studies with human participants or animals performed by any of the authors.

NEW INSIGHTS INTO PATHOGENESIS

DED has traditionally been divided into two categories: evaporative dry eye (EDE), which is typically due to excessive evaporation of the tear film, and aqueous tear-deficient dry eye (ADDE), which is characterized by inefficiency or failure of the lacrimal glands to produce tears. However, these two categories are not mutually exclusive, and DED is often seen clinically as a

combination of these subtypes [8]. Numerous extrinsic (e.g. environmental) and intrinsic (e.g. ageing and autoimmunity) factors contribute to the complexity of DED [9] (Fig. 1). The ocular surface, a highly exposed mucosa, is vulnerable to oxidative stress and environmental insults, which exacerbate tear evaporation and reduce tear clearance. This can lead to the accumulation of harmful agents in the tear film, making it toxic to the ocular surface [10]. The tear film, particularly its lipid layer, serves as a crucial barrier to water evaporation. Disruption of

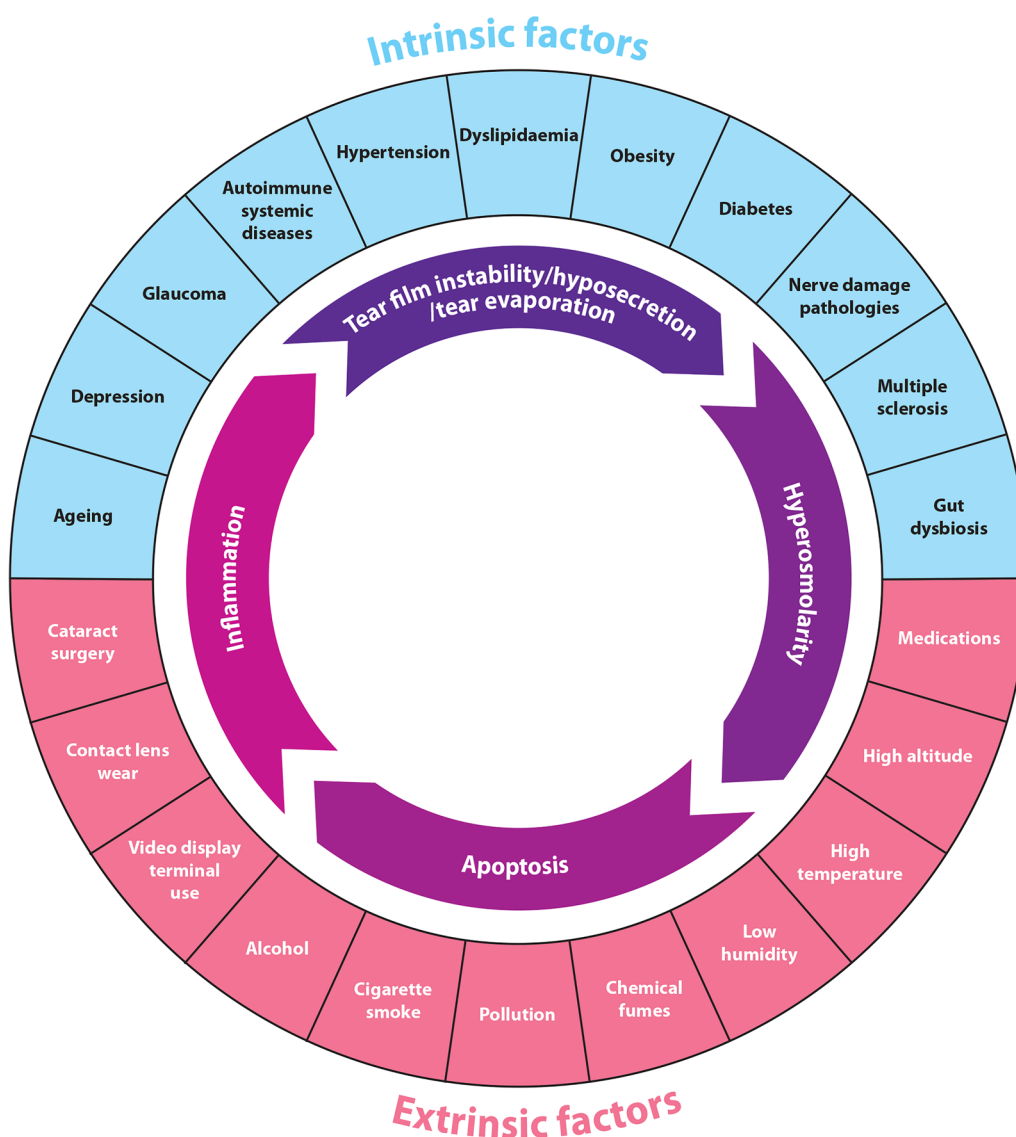


Fig. 1 Extrinsic and intrinsic factors that contribute to the vicious circle of dry eye disease (DED)

this layer exposes the tear film to air, increasing evaporation and leading to tear film thinning, instability, and tear break-up time (TBUT) [10].

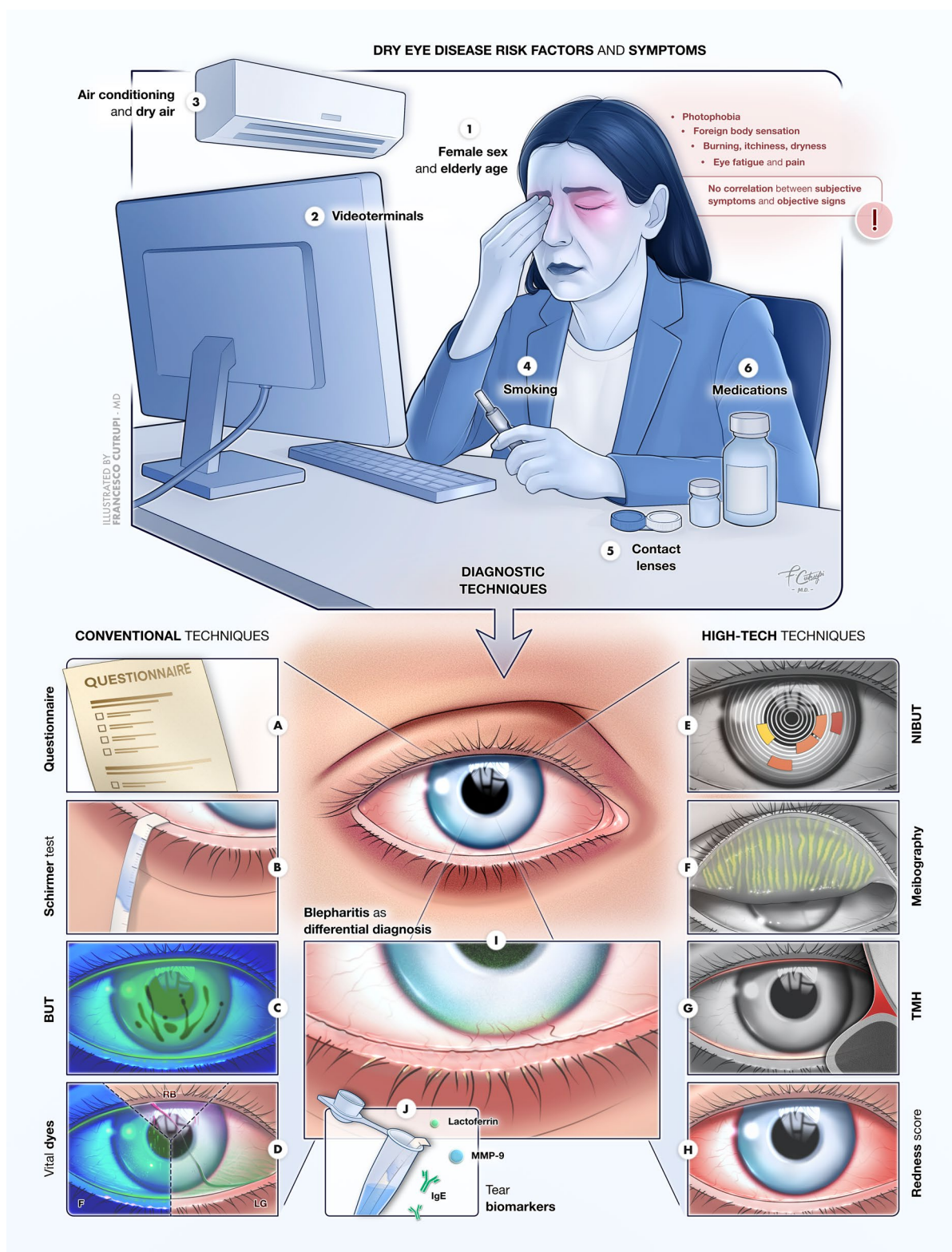
While ADDE is often associated with autoimmune disorders [11], meibomian gland dysfunction (MGD) is the most common cause of EDE [12]. Tear hyperosmolarity also plays a critical role in the development and progression of DED. It occurs in the presence of poor aqueous tear flow, excessive tear evaporation, or a combination of these events, due to changes in tear composition and production [11]. Tear hyperosmolarity can also indirectly cause ocular surface damage by inducing an inflammatory response that leads to apoptosis of corneal and conjunctival epithelial cells [11]. Inflammatory cells, such as neutrophils, macrophages, natural killer cells, dendritic cells, and T cells, play crucial roles in maintaining immune defence at the ocular surface. These cells respond to various triggers and contribute to an inflammatory cascade, activating common signalling pathways like mitogen-activated protein kinase (MAPK), nuclear factor kappa-light-chain-enhancer of activated B cells (NF- κ B), and transforming growth factor-beta (TGF- β) [13]. In addition to immune cell activation, neuronal injury can trigger neurogenic inflammation, perpetuating chronic dry eye inflammation [14]. These factors—chronic immune cell activation, disrupted tear film, and nerve damage—contribute to a self-perpetuating cycle that exacerbates and sustains ocular surface damage [13]. Therefore, osmoprotectants, small organic molecules used in many cell types to restore cell volume and stabilize protein function to allow adaptation to hyperosmolarity, are often contained in tear substitutes to counteract the surface damage caused by a hyperosmolar tear film [15]. There is now a growing body of evidence supporting the efficacy of various osmoprotectants such as trehalose, erythritol, taurine, and L-carnitine [15].

Excessive external stimuli can elicit an adaptive immunological response called para-inflammation, which has intermediate characteristics between the homeostatic and inflammatory states. The physiological role of para-inflammation is to restore tissue homeostasis; however, if tissue malfunction persists, para-inflammation can turn from a beneficial and protective

response to a detrimental and damaging process, inducing a continuous inflammatory state, leading to the chronicity of the disease [10, 16]. Furthermore, topical medications themselves may cause dry eye through the action of active principles and/or preservatives [17]. The risk of iatrogenic DED is also increased by taking five or more systemic drugs (polypharmacy) regardless of the type [18]. Other causes of dry eye may include the use of contact lenses, video display terminals, and eye surgery (e.g. corneal refractive surgery, cataract surgery, lid surgeries) [18]. In particular, more than half of patients presenting for cataract surgery have DED and MGD, even in the absence of frank symptoms. Therefore, as a routine approach to cataract surgery, preoperative assessment for the risk of developing or worsening DED should be encouraged in all patients [19].

DIAGNOSTIC CHALLENGES

The diagnosis of DED usually comprises a combination of subjective complaints assessed with various types of questionnaires and the detection of clinical signs detected with different techniques [20]. Subjective symptoms include photophobia, foreign body sensation, burning, itchiness, dryness, eye fatigue, and pain [21] (Fig. 2). One of the major challenges in the diagnosis of DED is the lack of correlation between the changes and severity of the clinical signs and the symptoms reported by the patient [22, 23]. In fact, some symptomatic patients have minimal ocular surface damage, while others show signs of DED without specific symptoms or no symptoms at all [22]. It is common to see patients with suspected DED and severe corneal neuralgia but no clinical signs. These patients are referred to as ‘pain without stain’ [24]. Therefore, it is essential to identify the general and local risk factors that may increase or predispose to the risk of ocular surface disease [21]. In many cases, detection and assessment of dry eye condition can be obtained quickly and without expensive equipment using a series of slit lamp-assisted tests performed during routine examinations [21]. Some of the conventional diagnostic



◀**Fig. 2** Dry eye disease—risk factors, symptoms, and diagnostic approach. The upper portion of the illustration depicts the main risk factors of DED alongside its common symptoms. The lower portion focuses on diagnostic techniques, including low-tech (left column, boxes A–D) and high-tech methods (right column, boxes E–H). The illustration also highlights the importance of differential diagnosis with blepharitis (box I) and new diagnostic approaches such as tear fluid analysis for biomarkers (box J). *F* fluorescein, *RP* rose bengal, *LG* lissamine green, *BUT* break-up time, *NIBUT* non-invasive break-up time, *TMH* tear meniscus height, *IgE* immunoglobulin E, *MMP-9* matrix metalloproteinase-9

techniques for examination include Schirmer's test, slit lamp examination with TBUT measurement, and ocular surface staining with vital dyes [25, 26] (Fig. 2). In general, these low-tech assessments face challenges with invasiveness and subjectivity, which can affect their repeatability and reproducibility [27]. In recent years, new high-tech instruments have changed clinical practice by providing better assessments of tear film quality, tear volume, and meibomian gland function. These tests, which include non-invasive tear break-up time (NIBUT), tear meniscus height (TMH), infrared meibography, and redness score, have shown better performance in detecting changes over time due to treatment, and results are particularly superior to low-tech assessments when different tests are combined [27] (Fig. 2). In vivo confocal microscopy (IVCM) is an emerging technology that enables in vivo assessment of structural changes in various ocular surface diseases. In dry eye research, IVCM has been utilized to examine the cornea, bulbar and palpebral conjunctiva, meibomian glands, and lacrimal glands [28]. The tear film is a reservoir for potential biomarkers, which have to be rapidly quantifiable. Access to patients' tears allows rapid, objective tear-based diagnostics, and early detection of biomarkers, such as lactoferrin, total immunoglobulin E (IgE), and matrix metalloproteinase 9 (MMP-9), may lead to faster intervention and more targeted disease monitoring [26]. Despite the plethora of tear biomarkers for DED, none of them has a universal clinical value, and the levels of these biomarkers must be considered along with the clinical history and

other ocular surface parameters for an accurate diagnosis [29].

Ideal DED diagnostic tests should be rapid, objective, reproducible, clinically impactful, and easy to use for efficient workflow [26]. In addition, many conditions, such as anterior blepharitis and *Demodex*-related blepharitis, can present with symptoms similar to those of DED; therefore, a differential diagnosis is essential to carefully distinguish DED from other potential causes [30]. Blepharitis is closely associated with DED, sharing various symptoms such as burning, irritation, photophobia, blurred vision, and red eyes. Clinical examination may reveal characteristic features of blepharitis, including scurf, telangiectatic vascular changes at the lid margin, inspissated meibomian glands, conjunctival hyperaemia, punctate keratopathy, and corneal vascularization and ulceration [31].

The classification of DED is based primarily on the severity of both symptoms and clinical signs. However, there is little consensus across Europe regarding precise definitions. In 2015, a group of Italian ocular surface experts proposed a multi-item flowchart to better define the diagnosis and determine appropriate treatments in patients with tear dysfunction [21]. In 2007, the International Dry Eye WorkShop (DEWS) introduced a severity grading scheme for DED, which was later confirmed in DEWS 2017 [8, 32]. In 2014, the ODISSEY Group published a tailored algorithm for severe DED [33]. Currently, it is unclear to what extent such an algorithm is followed in daily practice, as it only applies to severe dry eye. More recently, the work of the Italian Dry Eye Consensus Group has attempted to define practical algorithms for treatment recommendations that may be useful in a clinical setting for general ophthalmologists [34, 35], along with the definition of novel non-invasive screening tools [36] and an overview of eyelid-related issues [31, 37].

Quality of life (QoL) for patients with DED is significantly reduced by symptoms and altered visual function [38]. In diagnosing DED, most questionnaires ask about their impact on QoL, and reliable assessment of these parameters would be of great value in monitoring QoL over time and evaluating the effect of treatment in clinical practice [38]. Questionnaires

are valuable tools in both clinical research and practice for DED, providing consistent information on various aspects of the disease. They help assess objective clinical findings, such as tear film and ocular surface damage, as well as subjective symptoms [38]. The absence or delay of diagnosis is associated with poorer health-related mental health QoL in people with severe DED [39]. Undiagnosed DED may impose a greater burden on sufferers than diagnosed cases, as it leads to uncertainty, limited treatment prospects, and a lack of recognition due to the absence of clear clinical signs. This underscores the value of questionnaires in assessing both objective clinical findings, such as tear film and ocular surface damage, and subjective symptoms [39]. In addition, though effective medications for DED are available, achieving optimal symptom alleviation remains a challenge, as side effects can impact medication adherence [40].

TAILORED MANAGEMENT

The lack of a single clinical assessment method and the wide variety of symptoms make both diagnosis and treatment of DED difficult. For these reasons, it is crucial to educate patients about the chronic nature of DED, emphasizing that treatment is typically lifelong and may take time to show significant improvement [7]. Treatment of DED involves a stepwise approach according to the severity of the disease [41], and adapting lifestyle habits is essential in managing DED (Fig. 3). Patients should be encouraged to avoid aggravating factors such as cigarette smoke, dry heating air, air conditioning, prolonged reading, excessive screen time, and extended contact lens use [41]. Additionally, dietary changes, including increasing oral intake of essential fatty acids, can help alleviate symptoms. Proper lid hygiene is also crucial for maintaining eye health and managing the condition effectively [41]. Eyelid changes leading to lid margin disease, such as blepharitis or MGD, can lead to ocular surface system failure, potentially resulting in severe DED, requiring the establishment of appropriate supportive therapy [31, 37].

As MGD leads to a reduction in the thickness of the lipid layer, it may be beneficial to replace the lipids with lubricating eye drops or sprays containing lipids. A number of studies have shown an improvement in the signs and symptoms of dry eye with the use of lipid-based drops [41].

Artificial tears are the cornerstone of treatment for all stages of DED, aimed at both relieving symptoms and preventing their onset (Fig. 3). They should be applied consistently throughout the day, rather than on an as-needed basis, to prevent symptom exacerbation [6]. Various hydrophilic polymers, such as hyaluronic acid (HA), are commonly used in these formulations to enhance tear film stability. Newer generations combine HA with osmoprotectants and anti-inflammatory agents for a multi-action approach to therapy [6]. However, artificial tears containing preservatives like benzalkonium chloride should be avoided in cases of ocular surface disorders, as they may further irritate the eyes [42].

Effective management of inflammation, which is often present in a subclinical form, is critical for the improvement of DED symptoms and the disruption of the harmful cycle of surface damage and inflammation. Treatment options for controlling this inflammation include topical corticosteroids and cyclosporine A, oral antibiotics (macrolides, such as the immunosuppressant tacrolimus, and tetracyclines), along with essential fatty acids and *N*-acetyl-aspartyl-glutamic acid (NAAGA) [41, 43] (Fig. 3). While secretagogues and biological tear substitutes such as serum are sometimes considered in the management of DED and have been used to treat patients with severe ocular surface lesions unresponsive to conventional treatments [44], their use raises concerns regarding efficacy and safety due to the limited evidence and controlled trials available to support their effectiveness [45]. Furthermore, non-pharmacological therapies also offer valuable options for patients (Fig. 3). Punctal plugs, which use small collagen or silicone plugs to temporarily block the tear ducts, are effective in patients with severe ADDE, blocking tear drainage and increasing tear retention on the ocular surface [20]. However, concomitant anti-inflammatory treatment is indicated during this

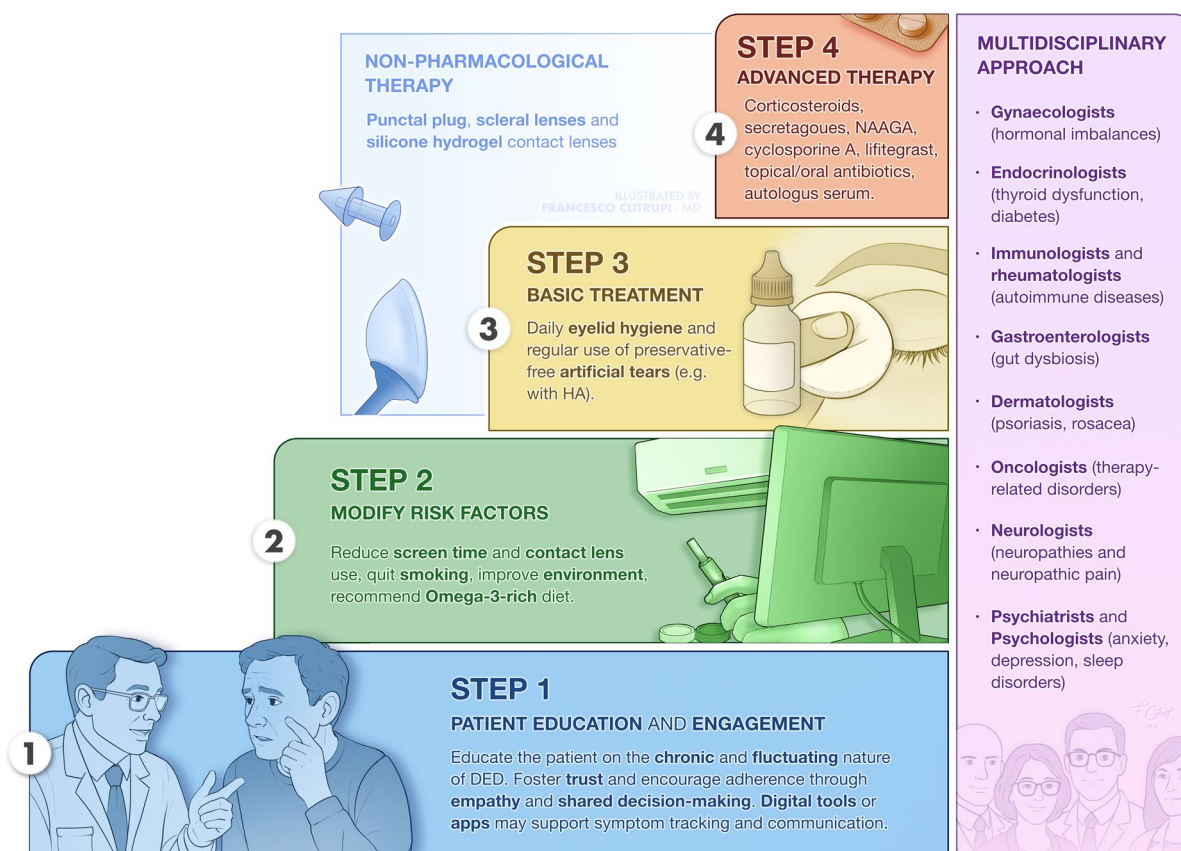


Fig. 3 Stepwise therapeutic approach for dry eye disease. This illustration outlines a stepwise approach to the treatment of DED, beginning with patient education and life-style modification, progressing through basic therapy with eyelid hygiene and artificial tears, and advancing to phar-

macologic and non-pharmacologic interventions according to severity. A multidisciplinary approach is always essential to address systemic and psychological comorbidities and ensure comprehensive care. *HA* hyaluronic acid, *NAAGA* N-acetyl-aspartyl-glutamic acid

procedure, as delayed tear drainage leads to the persistence of toxic and inflammatory factors on the ocular surface [20]. On the other hand, scleral lenses and silicone hydrogel contact lenses provide mechanical support and protection and have been suggested as a possible treatment for DED [6]. Recently, a stepped management algorithm has been developed to provide a stepwise approach to the implementation of the different management and therapeutic options according to the severity of the disease [41]. The subtype of DED, the presence of inflammation, and the presence of external and behavioural factors that may contribute to DED are important features to consider [46]. ED treatment should be personalized based on disease characteristics, patient factors, and environmental influences to

ensure optimal management. The consequences of managing DED include the ability to perform activities of daily living, emotional well-being, and the ability to work or drive, which also affect the productivity of patients at work [47, 48]. In addition, for some patients, the severity of the condition and its chronic nature are also associated with a high level of mood swings, anxiety, and depression [49]. Thus, patient education and counselling to emphasize the need for compliance are key factors in the successful management of DED, as is the development of new treatments aimed at reducing the disabling symptoms of the disease. Consequently, to ensure long-term relief and prevent chronicity of the disease, it is important not only to prescribe the appropriate treatment, giving clear

instructions, but also to monitor its effects over time [38]. Moreover, to improve patient satisfaction, co-operation and adherence, it is important for clinicians to demonstrate disease awareness and empathy, and to understand the patient's condition not only in terms of objective ocular parameters, but also in terms of their personal experience [7]. Experts in the consensus group emphasize the importance of empathy, dialogue, and communication in the management of patients with DED. In addition, they suggest a therapeutic alliance based on three main components: shared goals, agreed interventions, and a therapist–patient bond [50]. For this task, apps could improve doctor–patient communication by asking patients questions about their symptoms, adherence, and disease characteristics over time [7]. In addition, some DED patients may benefit from mental health interventions given the association between DED and depression/anxiety.

MULTIDISCIPLINARY APPROACH IN THE MANAGEMENT OF DED

DED is often linked to various comorbidities, many of which are treated by primary care clinicians or other specialists, such as rheumatologists, gynaecologists, endocrinologists, and gastroenterologists [51]. Given this common overlap, it is essential that practitioners across different specialties develop a clear understanding of DED and its potential connections to the conditions they treat. Therefore, a multidisciplinary approach involving ophthalmologists and other specialists is recommended to ensure effective diagnosis, management, and improved outcomes for patients with DED and its associated conditions (Fig. 3).

Gynaecologists

Some studies have suggested that changes in oestrogen levels alter the ocular surface balance and affect dry eye symptoms in female patients. In female patients with DED, the oestrogen peak is associated with symptoms of impaired tear production [52]. Evidence suggests that longer

duration and greater quantity of hormonal contraception use is significantly associated with a greater chance of DED [53]. Despite these associations, however, the evidence remains inconclusive, as studies have produced conflicting results, making it difficult to establish a definitive connection between hormone therapy and DED [54]. Research has also indicated a possible link between hormone replacement therapy (HRT) and DED, suggesting that women who use HRT, particularly oestrogen alone, may be at increased risk of DED. Therefore, gynaecologists should warn women taking or considering HRT about this potential complication [55]. Additionally, women with polycystic ovarian syndrome (PCOS) have been shown to suffer from more severe DED symptoms, further suggesting that hormonal imbalances may play a significant role in the progression and severity of the disease [56].

Endocrinologists

Hormonal imbalances, either physiological (menopause, ageing) or pathological, should be considered in the diagnosis and treatment planning of DED. Thyroid disorders have been implicated in the development of DED. Thyroid eye disease, thyroid-associated ophthalmopathy, thyroid-associated orbitopathy, and Graves' ophthalmopathy refer to an immune-mediated inflammatory condition that causes expansion of the extraocular muscles and fat in the orbit [57]. In particular, changes in the tear film and ocular surface are common in patients with thyroid-associated ophthalmopathy, while patients with active thyroid eye disease have evidence of severe MGD [57].

In severe cases, exposure keratopathy can complicate the course of thyroid eye disease. It results from inadequate lid closure due to axial proptosis (exophthalmos), a hallmark of Graves' disease, leading to dryness and corneal damage, particularly in the inferior region [58]. Effective diagnosis and treatment require a multidisciplinary team, including ophthalmologists, internists, radiologists, and head and neck surgeons [58]. Smoking cessation and euthyroid status help to prevent further exacerbations and reduce

the duration of active disease. The first and only US Food and Drug Administration (FDA)-approved prescription treatment for thyroid eye disease, teprotumumab, has been shown in clinical trials to reduce signs and symptoms in treated patients [59, 60]. For milder cases of corneal exposure, daily lubricant use and nightly patching are usually prescribed with success; for more severe cases, partial tarsorrhaphy or orbital decompression may be required [58]. Additionally, patients with diabetes mellitus show a high incidence of DED, further highlighting the connection between systemic conditions and dry eye symptoms [61].

Immunologists/Rheumatologists

Consultation with an immunologist or rheumatologist may be necessary when immune factors are suspected in the development of DED. In patients with immune system disorders, approximately 10–95% of cases are accompanied by dry eye [62]. Under normal conditions, the eye's intrinsic immune mechanisms are responsible for maintaining homeostasis of the microenvironment. When the immune system targets the eye, particularly in autoimmune diseases, the ocular immune response is over-stimulated by relevant immunoregulatory molecules. The result is an imbalance in the immunoregulatory mechanisms, leading to chronic ocular surface inflammation and DED [62]. For example, Sjögren's syndrome is a common autoimmune disease characterized by chronic inflammation, resulting in 'sicca' symptoms such as dry eyes and mouth that affect patients' QoL [63]. In addition, ocular manifestations are commonly seen in patients with systemic lupus erythematosus and rheumatoid arthritis [64, 65]. DED is the most common ocular manifestation in rheumatoid arthritis, and keratoconjunctivitis sicca is commonly seen in systemic lupus erythematosus [66]. Systemic sclerosis (SSc), a rare chronic connective tissue disease, also commonly presents with DED [66]. Other associated disorders include polyarteritis nodosa, Wegener's granulomatosis, primary biliary cholangitis (PBC), and mixed connective tissue disease [67].

Gastroenterologists

Alterations in the intestinal microbiome may be involved in the pathogenesis and/or maintenance of several ocular surface diseases, especially those in which inflammatory, neural, and/or hormonal factors are implicated [68, 69]. Many studies have reported imbalanced gut microbiota in patients with Sjögren's syndrome compared to controls [70, 71], and people with DED without autoimmune aetiology have shown changes in gut microbiome composition intermediate between Sjögren's syndrome and controls [71]. In addition, there may be a link between irritable bowel syndrome and DED, further suggesting that disturbances in gut health may have an impact on eye conditions, and that DED symptoms may be a source of further complications for patients with irritable bowel syndrome [72]. Inflammatory bowel disease (IBD), including Crohn's disease and ulcerative colitis, is linked to lacrimal gland dysfunction and ocular inflammation and serves as an independent risk factor for DED and corneal surface damage [73].

Dermatologists

People with dry skin often have dry eyes, and there is some statistical association between dry skin and DED [74]. Some evidence also suggests that patients with psoriasis have an increased risk of DED. However, the relationship between the two conditions remains unclear [75]. In addition, ocular rosacea is an underdiagnosed form of rosacea that can occur with or without the typical skin signs, and one of the most common symptoms is dryness [76]. Ocular rosacea is often associated with ocular conditions such as blepharitis and meibomitis, which can lead to MGD and subsequent atrophy [77], 30.

Oncologists

Modern cancer therapies are associated with ocular surface side effects that can cause severe

discomfort and even vision loss. The rapid detection of these adverse events is critical, as early intervention can aid in managing these conditions to prevent loss of vision and reduce the negative impact on QoL [78]. In particular, the use of aromatase inhibitors for breast cancer [79] or treatment with paclitaxel [80] can lead to DED. In addition, radiotherapy has many ophthalmic applications, and different studies have found a positive correlation between the incidence of DED and total radiation dose in patients with head and neck cancer [81, 82]. A recent study has also found a link between the presence of ovarian cancer and the development of DED [83].

Neurologists

Several neurological alterations including stroke, paralysis, migraine, myasthenia gravis, diabetic neuropathy, and neurotrophic keratitis are associated with tear film abnormalities and DED due to potential impairment of sensory and/or motor ocular innervation [84–86]. Therefore, neurologists should consider referring patients to an ophthalmologist for regular ocular surface evaluation. Neuropathic ocular pain, which is associated with injury to the corneal nerves, the terminal endings of the ophthalmic division of the trigeminal somatosensory system, can occur independently of dry eye, but it can also be a component of DED [87].

Psychiatrists

Some dry eye symptoms may be related to a mental health condition. Depression and anxiety are the most likely comorbidities and were found to be significantly correlated with the symptoms, but not signs, of DED [88]. Depression and anxiety may be both causes and effects of DED [88], along with the typical association of DED with sleep disturbance [89]. In some cases, depression can be a manifestation of the Sjögren's syndrome [90]. In addition, the use of antidepressants and tranquilizers can also worsen the signs and symptoms of DED [91]. The ocular discomfort and fatigue associated with DED can be distressing for patients,

limiting their ability to work and causing mood changes [88]. This suggests that appropriate medical management of psychological symptoms by a psychologist, in addition to DED therapy, may be beneficial for some patients with new-onset depression or anxiety symptoms following a diagnosis of DED.

Paediatricians

The prevalence of DED in paediatric patients is likely underestimated due to limited epidemiological data. Diagnosis is particularly challenging due to symptom identification difficulties, the lack of validated diagnostic criteria, and the absence of paediatric-specific tests [92]. Certain rare congenital disorders, including Riley–Day syndrome, ectodermal dysplasia syndromes, and epidermolysis bullosa, as well as autoimmune diseases, can contribute to reduced tear production and paediatric DED [93].

CONCLUSIONS

In conclusion, the effective management of DED requires an understanding of its complex pathogenesis, including dysfunctional para-inflammation and tear film instability, as well as lifestyle and environmental factors. This complexity makes patient care a collaborative effort that goes beyond symptom management. A multifaceted approach that combines pharmacological treatments, lifestyle modifications, and personalized patient education is essential in breaking the cycle of inflammation and tear instability that perpetuates the disease. The role of healthcare providers is not only to treat but also to educate patients about the chronic and often fluctuating nature of DED. By promoting understanding and adherence to care plans, they encourage active patient participation, leading to better outcomes, fewer exacerbations, and improved QoL. Advancements in diagnostics and a multidisciplinary approach that integrates the expertise of ophthalmologists with that of other healthcare professionals are essential in creating tailored strategies for each patient. Emerging therapies,

such as novel anti-inflammatory agents and tear-stabilizing technologies, show promise in addressing DED's unique and individualized challenges. These evolving tools and strategies underscore the potential for a more personalized and effective approach to managing DED, paving the way for better patient experiences and outcomes.

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Declarations

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Ethical Approval. This article is based on previously conducted studies and does not contain any new studies with human participants or animals performed by any of the authors.

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