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Modern Perspectives on Diagnosing and Treating Dry Eye Disease in Elderly Patients

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Abstract

Dry eye disease (DED) is a common multifactorial disorder of the ocular surface characterized by tear film instability, ocular discomfort, visual disturbance, and chronic inflammation. *The aim* of this review was to summarise current evidence regarding the pathophysiology, risk factors, diagnosis, and management of dry eye disease, with particular emphasis on its impact in the elderly population. *Material and methods*. A narrative literature review with PRISMA-informed methodology was conducted using PubMed, Web of Science, and Google Scholar. Literature published within the last 10 years concerning dry eye disease, ocular surface aging, meibomian gland dysfunction, and treatment strategies was analysed. *Results*. Dry eye disease is strongly associated with age-related changes in the lacrimal glands, meibomian glands, ocular surface epithelium, and neural regulation of tear secretion. Additional contributing factors include systemic comorbidities, polypharmacy, hormonal alterations, and environmental exposures. The most common clinical manifestations include ocular irritation, dryness, burning, foreign body sensation, fluctuating vision, and reduced visual quality, all of which can significantly impair daily functioning and quality of life. Diagnosis is based on a combination of patient-reported symptoms and clinical assessment of tear film stability and ocular surface integrity. Management strategies include environmental modifications, artificial tear supplementation, anti-inflammatory therapy, autologous serum eye drops, punctal occlusion, scleral lenses, and surgical options in severe or refractory cases. *Conclusions*. Dry eye disease in elderly patients is a progressive, multifactorial condition requiring early recognition and individualized, stepwise management to prevent disease progression and optimize quality of life.

Keywords: dry eye disease; ocular inflammation; elderly patients; tear film instability

1. Introduction

Dry eye disease, also known as keratoconjunctivitis sicca, is one of the most common ocular surface disorders encountered in clinical practice and represents a growing public health concern worldwide [1]. The condition is particularly prevalent among older adults, with age-related changes in tear production, tear film stability, and ocular surface function contributing to an increased risk of disease development. In addition to causing ocular discomfort, DED may impair visual function and negatively affect daily activities, thereby reducing quality of life [2].

DED is defined as a multifactorial disease of the ocular surface characterized by a loss of tear film homeostasis and accompanied by ocular symptoms, in which tear film instability, hyperosmolarity, inflammation, and neurosensory dysfunction are key pathogenic contributors [3]. Clinically, DED may present as episodic, typically triggered by environmental stressors or reduced blink rate during visual tasks, or as chronic, in which symptoms persist continuously and may be accompanied by ocular surface damage [4].

The tear film plays a crucial role in maintaining corneal epithelial integrity, providing lubrication, protecting against environmental stressors, and ensuring a smooth optical surface for clear vision [5]. Disruption of its homeostasis may result in epithelial damage and the development of characteristic symptoms, including dryness, irritation, and visual fluctuation. Consequently, a thorough understanding of DED pathophysiology, risk factors, and clinical manifestations is particularly important in aging populations, where disease burden is highest. Given its multifactorial nature, effective diagnosis and management require an integrated understanding of the underlying mechanisms affecting both the tear film and ocular surface [2,4].

The tear film is a trilaminar structure composed of lipid, aqueous, and mucin layers, each required in appropriate proportions for normal stability. The superficial lipid layer, secreted by the meibomian glands in the tarsal plates, reduces evaporation. The aqueous component, produced by the main and accessory lacrimal glands, supplies hydration and soluble proteins. The innermost mucin layer, secreted by conjunctival goblet cells, enhances tear adhesion to the ocular surface. More recent concepts describe the tear film as a hydrated mucin-based gel in which lipids are not restricted to a discrete surface layer but are integrated within a protein-rich matrix [6]. Blinking distributes the tear film across the cornea, while drainage occurs through the puncta into the nasolacrimal system. Eyelid malposition, such as ectropion or lid laxity, can

impair tear distribution and drainage, contributing to tear film instability [7,3]. In addition to its physical roles, the tear film contains antimicrobial factors including immunoglobulins, lysozyme, and leukocytes, providing protection against infectious keratitis [8].

Dry eye conditions have been classified into two main categories by the National Eye Institute: aqueous tear deficiency and evaporative dry eye. Evaporative dry eye is caused by deficient and/or alterations in lipid secretions by the meibomian glands resulting in increased evaporation of aqueous tears from the ocular surface. The leading cause is meibomian gland dysfunction. Although differentiating patients according to the main causative factor is useful for diagnosis and therapy, clinical presentation is often a mixture of both pathogenic pathways [1,9]. A detailed understanding of these interacting components is essential for accurate diagnosis and effective management of DED.

2. Materials and methods

2.1. Literature search strategy

A narrative literature review with PRISMA-informed methodology was conducted using PubMed, Web of Science and Google Scholar databases. Literature published between January 2016 and January 2026 was analysed in order to summarise current evidence regarding dry eye disease in the elderly. The search strategy included combinations of the following keywords and Medical Subject Headings (MeSH): “dry eye”, “ocular surface disease”, “keratoconjunctivitis sicca”, “elderly”, “aging”, “meibomian gland dysfunction”, “tear film”, “artificial tears”.

The review primarily focused on clinical studies, systematic reviews, meta-analyses, narrative reviews and international clinical guidelines related to DED in the elderly. Additional references were identified through manual screening of bibliographies from selected articles.

2.2. Eligibility criteria

Studies published in English between 2016 and 2026 involving elderly populations with dry eye disease or keratoconjunctivitis sicca were considered eligible for inclusion. Priority was given to high-quality evidence, including international guidelines, systematic reviews, meta-analyses and multicentre clinical studies. Articles lacking scientific relevance, conference abstracts without full-text availability and publications with insufficient methodological quality

were excluded from the analysis.

2.3. Data collection and narrative synthesis

Relevant publications were independently screened according to their titles, abstracts and full-text content. The selected studies were analysed with emphasis on epidemiology, pathophysiology, clinical manifestations, diagnostic approaches and therapeutic management of dry eye disease in the elderly.

Due to heterogeneity among the included studies, a narrative synthesis approach was applied. The collected evidence was organised into thematic sections to provide a comprehensive overview of current concepts in dry eye disease diagnosis and management.

2.3.1. Use of artificial intelligence

Artificial intelligence tools were used exclusively for linguistic refinement, structural organisation of the manuscript and improvement of academic English readability. All scientific content, interpretation of evidence, critical analysis and final conclusions were independently reviewed and verified by the authors. AI-assisted technologies were used solely as supportive instruments and did not replace human scientific judgement.

3. Results

3.1 Epidemiology and risk factors

Dry eye disease is a multifactorial disorder resulting from disruption of tear film homeostasis and the complex interactions between the ocular surface, tear-producing glands, eyelids, and neural pathways. Tear film instability is considered a central feature of DED pathophysiology and may arise from abnormalities in any component of the tear film, particularly dysfunction of the meibomian glands, which represent one of the most common causes of evaporative dry eye [9,10].

Inflammation represents another major mechanism involved in the development and progression of DED. Autoimmune disorders such as Sjögren syndrome and systemic lupus erythematosus may directly affect the lacrimal glands through inflammatory cell infiltration, resulting in severe aqueous tear deficiency [12]. Chronic ocular surface inflammation associated with blepharitis, meibomian gland dysfunction, and ocular rosacea may further

destabilize the tear film through damage to the eyelid margin and reduction of conjunctival goblet cell density [9,13].

Hormonal factors also play an important role in maintaining normal tear film homeostasis. Androgens support both lacrimal gland and meibomian gland function and appear to exert anti-inflammatory effects on the ocular surface, whereas age-related reductions in androgen levels have been associated with an increased prevalence of DED [10,13]. This relationship may partly explain the higher incidence of DED among postmenopausal women, although the influence of hormone replacement therapy remains controversial. Large epidemiological studies have shown that after the age of 50, the prevalence of DED in women and men increases every 5 years, and the prevalence rate of women is higher than that of men [14].

Aging itself is considered one of the strongest risk factors for DED, as age-related changes in the lacrimal glands, meibomian glands, ocular surface epithelium, and immune system contribute to reduced tear production, increased inflammation, and impaired tear film stability. This increased risk is further influenced by the higher prevalence of systemic diseases among older adults, which is particularly important when this requires systemic therapy that may reduce tear production. Common examples include medications with anticholinergic properties (e.g., amitriptyline), diuretics, and antihistamines [15].

Damage to the ocular surface may likewise result in severe tear film dysfunction. Chemical injuries, Stevens–Johnson syndrome, ocular cicatricial pemphigoid, and trachoma can cause extensive conjunctival scarring and loss of mucin-producing goblet cells, resulting in profound ocular surface disease and severe dry eye [16]. Neural mechanisms are also important because normal tear production depends on intact corneal innervation and a functional lacrimal reflex arc. Conditions such as herpes simplex infection, herpes zoster ophthalmicus, rheumatoid arthritis, prolonged contact lens wear, and corneal refractive surgery may impair corneal sensation and contribute to chronic tear deficiency and neurotrophic ocular surface disease [10,17].

In addition to these pathogenic mechanisms, numerous physical, environmental, and lifestyle-related factors may exacerbate dry eye symptoms. Incomplete blinking, eyelid malposition, lagophthalmos, facial nerve paralysis, and thyroid eye disease may increase tear evaporation despite normal tear production [18]. Environmental exposures including low humidity, air

conditioning, indoor heating, wind, pollution, and extreme temperatures have been shown to destabilize the tear film and increase ocular surface desiccation. Established risk factors for DED include advanced age, female sex, smoking, contact lens wear, prolonged digital device use, refractive surgery, and the use of medications [19]. These factors are particularly relevant in elderly individuals, who frequently present with systemic comorbidities, polypharmacy, and age-related ocular surface changes that collectively increase susceptibility to DED [15].

3.2 Clinical presentation

The instability of the tear film and its inability to adequately protect and lubricate the ocular surface are responsible for the characteristic symptoms of DED. Patients commonly report burning, stinging, grittiness, foreign body sensation, ocular fatigue, and a sensation of dryness. The severity of symptoms can vary considerably, ranging from mild discomfort and intermittent irritation to chronic pain and significant visual impairment in more severe cases [1,7].

Dry eye symptoms are frequently exacerbated by environmental and behavioral factors that increase tear evaporation or reduce blink frequency, such as exposure to wind, air conditioning, prolonged reading, and extended use of digital devices. A paradoxical symptom occasionally reported by patients is excessive tearing, which occurs as a reflex response to ocular surface irritation despite underlying tear film deficiency [7].

In addition to ocular discomfort, patients with DED frequently experience visual disturbances, including intermittent blurred vision, fluctuating visual acuity, and reduced visual quality resulting from disruption of the corneal optical surface and damage to the central corneal epithelium [3,20]. These symptoms are primarily attributable to tear film instability, which disrupts the smooth optical surface of the cornea and results in fluctuations in refractive power. Visual symptoms often become more pronounced during prolonged visual tasks, such as reading or digital device use, and may significantly impair daily functioning and quality of life [3].

3.3 Diagnostic evaluation

The diagnosis of DED is based on a combination of symptom assessment, comprehensive history taking, and objective evaluation of tear film and ocular surface abnormalities. The classical approach to diagnosis of the dry eye is to demonstrate decreased tear function (rapid tear break-up, decreased Schirmer secretion tests or low tear meniscus height) and evidence of

ocular surface damage (fluorescein staining of the cornea and conjunctiva, rose bengal or lissamine green staining of the conjunctiva) [4].The Schirmer test is one of the classic diagnostic tests for dry eye and evaluates tear production by measuring the wetting of filter paper strips placed in the inferior conjunctival fornix over a period of 1–5 minutes [4]. Detailed examination of patients with dry eye includes slit-lamp biomicroscopy, measurement of tear break-up time and ocular surface staining with vital dyes such as fluorescein dye or rose bengal. Additional diagnostic methods include tear osmolarity measurement, corneal sensitivity testing, impression cytology, and conjunctival surface biopsy [21]. Severe forms of dry eye syndrome associated with Sjögren syndrome or dermatological conditions such as rosacea may lead to vision-threatening complications, therefore, early differential diagnosis is essential [10,11]. In most patients, DED develops gradually over many years and is characterized by progressive reduction in tear production and increasing ocular surface damage in advanced stages of the disease [10].

3.4 Therapeutic management

The management of DED begins with identification and treatment of the underlying etiology whenever possible. Due to the multifactorial nature of the disease, management often requires a combination of therapeutic approaches. Treatment strategies emphasize addressing the specific causes of dry eye while minimizing factors that exacerbate symptoms [1]. An important initial step is patient education regarding environmental and behavioral factors that may worsen symptoms, including prolonged reading and video display terminal use, both of which reduce blink frequency and accelerate tear film break-up [4]. In addition, every effort should be made to identify and manage associated conditions contributing to the disease process, as symptomatic treatment alone does not address the underlying pathology; in many cases the dry eye improves as the patient goes into remission [7].

3.4.1 Lid hygiene and environmental modifications

Lid hygiene constitutes an important component of dry eye management, particularly in patients with blepharitis or meibomian gland dysfunction. Regular application of warm compresses and lid cleansing may improve meibomian gland function, reduce ocular surface inflammation, and decrease reliance on artificial tear supplementation [22]. When infectious blepharitis is suspected, topical antibiotic therapy, such as erythromycin or bacitracin ointment, may be used in conjunction with continued lid hygiene [22]. In cases associated with ocular rosacea, oral

tetracyclines, particularly doxycycline, may provide additional anti-inflammatory benefits and improve meibomian gland function [23].

Environmental modifications may also contribute to symptom relief by reducing tear evaporation. The use of humidifiers, moisture chamber spectacles, and avoidance of desiccating environments may help maintain ocular surface hydration, particularly in patients with evaporative DED [18].

3.4.2 Tear film supplementation

Regardless of the underlying cause, first-line treatment consists of tear film supplementation with artificial tears and lubricating ointments. Artificial tears are formulated to improve tear film stability, increase tear film break-up time, and enhance ocular surface lubrication. These preparations typically contain electrolytes, surfactants, viscosity-enhancing agents, and, in some cases, preservatives. Some formulations additionally contain lipids to improve tear film stability [1].

The primary goal of tear substitutes is to maintain ocular surface hydration and promote epithelial recovery. Clinical studies have demonstrated improvements in tear function, ocular surface staining, and overall ocular surface integrity following the use of artificial tear substitutes [24].

Artificial tear substitutes remain the cornerstone of first-line therapy for DED. Advances in the understanding of dry eye pathophysiology have led to the development of formulations designed to target specific abnormalities of the tear film and ocular surface, including tear film instability, hyperosmolarity, inflammation, and epithelial damage. Consequently, the selection of an artificial tear preparation should be guided by the predominant subtype of DED and the patient's individual clinical characteristics [20].

Hyaluronic acid remains one of the most widely used components of contemporary artificial tears due to its viscoelastic properties, high water-retention capacity, and ability to promote epithelial healing [25]. In addition, formulations containing trehalose have gained increasing attention because of their cytoprotective and osmoprotective effects, which may help protect ocular surface cells from desiccation-induced stress and inflammation [26].

Lipid-containing artificial tears have emerged as an important therapeutic option for patients with evaporative DED, particularly when meibomian gland dysfunction is present. By

replenishing the lipid layer of the tear film, these formulations reduce tear evaporation and improve tear film stability [27]. Recent clinical evidence has demonstrated the efficacy and safety of lipid-containing tears in improving symptoms and ocular surface parameters in patients with DED [28].

Another important trend in contemporary dry eye management is the increasing use of preservative-free artificial tears to avoid corneal toxicity, particularly in patients requiring frequent administration or those with moderate-to-severe disease [20]. Preservative-free tears come in individual disposable droppers for each application and are more expensive. These patients should also be given a preservative-free (and possibly lanolin-free) ocular lubricating ointment to be used in each eye at bedtime, especially if nocturnal lagophthalmos is suspected [7]. Although artificial tears remain a cornerstone of dry eye management, current evidence suggests that their greatest benefit is achieved when formulation characteristics are matched to the underlying pathophysiology of the disease [20].

Autologous serum eye drops (ASEDs) have emerged as an important therapeutic option for patients with moderate-to-severe DED, particularly in cases refractory to conventional artificial tear therapy. Unlike commercially available tear substitutes, ASEDs are derived from the patient's own blood serum and contain numerous biologically active components naturally present in tears, including epidermal growth factor (EGF), transforming growth factor-β (TGF-β), fibronectin, vitamin A, cytokines, and other proteins that support epithelial healing and ocular surface homeostasis [29]. As a result, ASEDs are considered not only lubricating agents but also biological therapies that may promote regeneration of the ocular surface and improve tear film function [29,30]. Consequently, they are most commonly used in severe DED and other ocular surface disorders characterized by persistent epithelial damage and inadequate response to conventional treatment [31,30].

3.4.3 Topical agents

Topical anti-inflammatory therapies play an important role in the management of DED, particularly in patients with chronic ocular surface inflammation. Cyclosporine is an immunomodulatory agent that inhibits T-cell activation and reduces inflammatory processes affecting the lacrimal glands and ocular surface [32]. Clinical studies have demonstrated that cyclosporine treatment can improve tear production, reduce ocular surface staining, and alleviate dry eye symptoms [32,33]. As a result, it is commonly prescribed for patients with

moderate-to-severe DED requiring long-term anti-inflammatory therapy.

Topical corticosteroids may be used for short-term management of acute exacerbations of DED due to their potent anti-inflammatory effects. They can rapidly reduce ocular surface inflammation and improve patient symptoms; however, prolonged use is limited by the risk of adverse effects, including elevated intraocular pressure, cataract formation, and increased susceptibility to infection [33].

Lifitegrast is a relatively recent therapeutic option that targets T-cell-mediated inflammation. Clinical trials have shown that lifitegrast can improve both the signs and symptoms of DED, making it an effective treatment for patients with inflammatory forms of the condition [34,35].

However, the therapeutic effect of topical agents intended to reduce tear hyperosmolarity may be transient, potentially limiting their long-term efficacy when used as monotherapy [4]. For patients with persistent symptoms despite conservative treatment, tear conservation strategies may be considered. Punctal occlusion, achieved through temporary collagen plugs or longer-lasting silicone plugs, reduces tear drainage and increases tear retention on the ocular surface. This approach is most commonly used in patients with aqueous-deficient DED [36].

Scleral contact lenses represent another therapeutic option for patients with severe or refractory DED. These large-diameter lenses create a fluid reservoir over the cornea, providing continuous hydration and protection of the ocular surface. In addition to improving visual function and patient comfort, scleral lenses may promote epithelial healing in individuals with advanced ocular surface disease [37].

3.4.4 Surgical management

Surgical intervention is generally reserved for severe or refractory cases. Tarsorrhaphy may be performed in patients with exposure keratopathy, neurotrophic keratopathy, or severe ocular surface disease to reduce tear evaporation and protect the cornea [38]. In advanced ocular surface disorders, including Stevens–Johnson syndrome, ocular cicatricial pemphigoid, and severe chemical injuries, reconstructive procedures such as conjunctival or ocular surface transplantation may be required to restore ocular surface integrity and preserve visual function [39].

3.4.5 Lifestyle and environmental modifications

In addition to pharmacological and procedural interventions, lifestyle and environmental modifications play an important role in the management of DED. Measures aimed at reducing tear evaporation, including the use of protective eyewear, optimization of indoor humidity, and avoidance of direct airflow from fans, air-conditioning systems, and heating vents, may help alleviate symptoms and improve ocular surface comfort [18]. Patients should also be advised to avoid exposure to tobacco smoke, which has been associated with tear film instability, ocular surface irritation, and worsening dry eye symptoms [19]. Dietary modifications may provide additional benefits, particularly through increased intake of omega-3 fatty acids, which have been shown to improve tear film parameters and reduce ocular surface inflammation in some patients with DED [40,41]. Furthermore, reducing alcohol consumption and, where clinically appropriate, minimizing the use of medications known to exacerbate dry eye symptoms, such as antihistamines and systemic diuretics, may contribute to improved disease control [18]. Collectively, these measures complement conventional therapies and support a comprehensive, individualized approach to the management of DED.

Discussion

Dry eye disease is a multifactorial and increasingly prevalent disorder of the ocular surface that may significantly impair visual performance and overall quality of life, especially among elderly individuals [1,2]. Population aging, increasing environmental exposures, prolonged screen use, and the growing prevalence of systemic diseases have all contributed to the expanding global burden of DED [2,15]. Physiological age-related alterations involving the lacrimal glands, meibomian glands, ocular surface epithelium, and neural regulation of tear secretion are considered major contributors to disease susceptibility in older adults [2,10].

This narrative review summarises contemporary evidence concerning the epidemiology, pathophysiology, clinical presentation, diagnostic evaluation, and treatment of DED in the elderly population. Current literature highlights that DED is not a single pathological entity but rather a heterogeneous condition involving multiple interacting mechanisms, including tear film instability, chronic inflammation, hormonal imbalance, neurosensory dysfunction, and environmental influences [3,10]. Persistent ocular surface inflammation and hyperosmolarity appear to play a central role in maintaining the vicious cycle responsible for disease progression and symptom persistence [3,10].

Among the recognised pathogenic mechanisms, meibomian gland dysfunction remains one of the most common causes of evaporative DED, particularly in ageing populations [9,10]. Autoimmune diseases such as Sjögren syndrome, as well as chronic blepharitis and ocular rosacea, may further contribute to ocular surface inflammation and tear film disruption [11,13]. Hormonal changes associated with aging, especially reductions in androgen levels, have also been linked to impaired lacrimal and meibomian gland function [13]. Furthermore, elderly patients are frequently affected by systemic comorbidities requiring medications that may decrease tear production, including antihistamines, antidepressants, and diuretics [15]. Environmental conditions such as low humidity, pollution, air-conditioning exposure, and prolonged visual tasks additionally exacerbate tear evaporation and symptom severity [19].

Prompt recognition of DED is particularly important because untreated disease may progressively impair ocular surface integrity and visual quality [3,4]. Nevertheless, diagnosis remains challenging due to the often inconsistent relationship between patient-reported symptoms and objective clinical findings [10,21]. Many patients experience burning, foreign body sensation, fluctuating vision, reflex tearing, or ocular fatigue, all of which may interfere with reading, driving, and other daily activities essential for maintaining independence in older age [3,20]. Consequently, current diagnostic strategies combine symptom assessment with objective evaluation of tear film stability and ocular surface damage using methods such as Schirmer testing, tear break-up time measurement, ocular surface staining, and tear osmolarity analysis [4,21].

Artificial tears continue to represent the foundation of first-line therapy for most patients with DED [20]. Improved understanding of tear film composition and ocular surface physiology has led to the development of newer formulations specifically designed to address tear instability, lipid deficiency, hyperosmolarity, and epithelial injury [20,25,27]. Hyaluronic acid–based preparations and lipid-containing tear substitutes have demonstrated favourable effects on ocular surface hydration and tear film stability [25,27,28]. At the same time, preservative-free formulations are increasingly recommended for patients requiring long-term or frequent treatment because of their lower risk of ocular surface toxicity [20].

Anti-inflammatory therapies have become increasingly important in contemporary DED management, particularly in patients with chronic inflammatory disease [32,33]. Topical cyclosporine and lifitegrast have demonstrated efficacy in reducing ocular surface inflammation and improving both clinical signs and symptoms [32,34]. Short-term

corticosteroid therapy may provide rapid symptom relief during acute exacerbations, although prolonged administration remains limited by the risk of complications such as cataract formation and elevated intraocular pressure [33]. In more advanced or treatment-resistant cases, additional therapeutic strategies including autologous serum eye drops, punctal occlusion, scleral lenses, and surgical procedures may be necessary to preserve ocular surface integrity and maintain visual function [29–31,36–39].

Non-pharmacological measures also play a substantial role in symptom management and long-term disease control. Environmental optimisation, smoking cessation, reduction of direct airflow exposure, and the use of protective eyewear may help reduce tear evaporation and improve patient comfort [18,19]. Moreover, emerging evidence suggests that omega-3 fatty acid supplementation may exert beneficial anti-inflammatory effects and improve selected tear film parameters in some patients with DED [40,41].

Recent international recommendations increasingly support a multidisciplinary approach to DED management, particularly in elderly individuals with systemic autoimmune disease or multiple comorbidities [11,18]. Cooperation between ophthalmologists, rheumatologists, dermatologists, and primary care physicians may facilitate earlier diagnosis, more effective treatment selection, and improved long-term disease control.

Despite considerable progress in understanding DED pathophysiology and treatment, important limitations remain within the available literature. Many studies demonstrate heterogeneity regarding diagnostic criteria, patient populations, therapeutic protocols, and outcome measures, which complicates direct comparison of results [20,21]. Additional prospective multicentre studies are therefore required to establish standardised diagnostic algorithms and evaluate the long-term efficacy of emerging therapies in elderly populations.

Several limitations of this review should be acknowledged. Owing to its narrative design, the review may be associated with potential selection bias and heterogeneity among the included publications. Furthermore, variations in study populations, diagnostic criteria, disease severity, and therapeutic approaches across the analysed studies may restrict direct comparison of the reported findings. Despite these limitations, the review summarises current knowledge and provides an up-to-date overview of contemporary concepts related to the diagnosis and management of DED in elderly patients.

5. Conclusion

Dry eye disease is a prevalent and increasingly important ocular disorder, particularly among older adults, in whom age-related physiological changes, systemic comorbidities, and medication use contribute to a heightened risk of disease development. Although often perceived as a minor ocular complaint, DED is a chronic disease that can substantially affect visual function, daily activities, psychological well-being, and overall quality of life.

The impact of DED extends beyond ocular discomfort. Persistent symptoms may interfere with reading, driving, computer use, and other activities essential for maintaining independence in older age. In severe cases, chronic symptoms may contribute to social withdrawal, reduced productivity, sleep disturbances, and increased levels of anxiety and depression. Consequently, the burden of DED should be considered not only in terms of ocular health but also in relation to its broader effects on physical, emotional, and social functioning.

Accurate diagnosis requires careful assessment of symptoms and objective evaluation of tear film and ocular surface abnormalities. Because DED is a multifactorial disease, management should be individualized and directed not only toward symptomatic relief but also toward addressing the underlying pathogenic mechanisms through the use of evidence-based contemporary therapeutic options.

As the global population continues to age, the prevalence and societal burden of DED are expected to increase. Greater awareness of risk factors, early diagnosis, and implementation of targeted therapeutic strategies are essential for reducing disease-related morbidity, preserving visual function, and improving quality of life among elderly patients. Continued research into the mechanisms and treatment of DED will further contribute to more effective long-term management of this common and often underestimated condition.

Declarations

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