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Dry Eye Disease: An Increasing Ocular Surface and Public Health Challenge

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Abstract: Background: Dry eye disease is a common, multifactorial ocular surface disorder characterized by symptoms and loss of tear-film or ocular-surface homeostasis. Its burden is increasing because of population ageing, digital-device use, environmental pollution, contact-lens wear, systemic disease and ocular procedures. Although rarely sight-threatening in its mild forms, persistent disease can substantially impair visual function, productivity, sleep, mental well-being and quality of life. **Objective:** This narrative review critically examines the epidemiology, pathophysiology, diagnosis, management and public health significance of dry eye disease, with particular attention to recent advances and the Indian context. **Key findings:** Dry eye includes overlapping aqueous-deficient, evaporative and neurosensory mechanisms rather than two mutually exclusive categories. Symptoms and clinical signs correlate poorly, making diagnosis dependent on a structured assessment rather than a single test. Meibomian gland dysfunction, ageing, female sex, autoimmune disease, contact-lens use, ocular surgery, medications, low-humidity environments and prolonged digital-device use are important contributors. Management should be individualized and may include environmental modification, eyelid therapy, tear supplementation, anti-inflammatory treatment, tear conservation, secretagogues and selected device-based procedures. Evidence does not support routine omega-3 supplementation for all patients. India faces a substantial but poorly quantified burden, with clinic-based studies reporting high prevalence and earlier presentation among digital-device users. **Conclusion:** Dry eye disease should be approached as a chronic, heterogeneous disorder requiring identification of dominant mechanisms and associated conditions. Public health action should improve awareness, occupational and digital-eye-health practices, primary-care recognition, rational medicine use and access to affordable ophthalmic assessment.

Keywords: Dry eye disease; ocular surface; meibomian gland dysfunction; digital eye strain; tear film; India

INTRODUCTION

Dry eye disease is among the most frequent reasons for seeking eye care. Patients may report burning, grittiness, foreign-body sensation, fluctuating vision, photophobia, watering, eye fatigue or difficulty keeping the eyes open. Paradoxical tearing is common because ocular-surface irritation can stimulate reflex lacrimation even when the underlying tear film is unstable.

The Tear Film and Ocular Surface Society Dry Eye Workshop III defines dry eye as a multifactorial, symptomatic disease characterized by loss of homeostasis of the tear film and/or ocular surface, in which tear-film instability and hyperosmolarity, ocular-surface inflammation and damage, and neurosensory abnormalities are etiological factors.[1] This formulation represents an important conceptual

development. Dry eye is not simply insufficient tear volume, and symptoms are central to the diagnosis. Individuals with abnormal tests but no symptoms may have ocular-surface dysfunction or be at risk of future disease, but they do not necessarily meet the contemporary definition of symptomatic dry eye.

The term "dry eye syndrome" is still used, although "dry eye disease" more accurately reflects its chronic and biologically complex nature. The condition includes aqueous-deficient dry eye, evaporative dry eye and mixed forms. These categories frequently overlap. A patient with meibomian gland dysfunction may develop tear hyperosmolarity and inflammation, while chronic inflammation may subsequently impair lacrimal secretion.

Dry eye is often regarded as a minor inconvenience because it usually does not cause blindness. This view underestimates its impact. Stable tears are essential for maintaining a smooth refractive surface. Tear-film disruption can therefore cause fluctuating vision, particularly during reading, driving and screen work. Severe disease may produce epithelial defects, infection, scarring or visual impairment, especially in autoimmune and cicatrizing disorders.

Epidemiology and Disease Burden

The reported prevalence of dry eye varies from below 10% to more than 50%, depending on population, age, geography and diagnostic definition. Studies based only on symptom questionnaires usually produce different estimates from those requiring both symptoms and objective signs. A recent epidemiological review concluded that variation in diagnostic criteria remains a major obstacle to reliable global comparisons.[3]

Age is one of the most consistent risk factors. Lacrimal function, corneal sensitivity, blink dynamics and meibomian gland structure may deteriorate with ageing. Women, particularly after menopause, are frequently affected more than men, probably through interactions among sex hormones, immune regulation and glandular function. The association is not explained by oestrogen deficiency alone, and hormonal treatment does not uniformly improve disease.

Dry eye is increasingly recognized among children and young adults. A 2025 systematic review of 42 paediatric cohorts estimated an overall prevalence of 23.7%, but the estimate varied substantially according to diagnostic method. Prevalence based on symptoms was approximately twice that based on clinical signs, illustrating the uncertainty created by inconsistent case definitions.[4]

Population prevalence should not be inferred from eye-clinic data. Patients attending ophthalmology services are more likely to have symptoms, ocular disease, contact-lens use or previous surgery. Conversely, community surveys may miss mild or intermittent disease. Meaningful surveillance therefore requires standardized symptom instruments, objective tests and population-based sampling.

Tear-Film Homeostasis and Pathophysiology

The tear film is maintained by the lacrimal glands, ocular-surface epithelium, meibomian glands, eyelids, corneal nerves and immune system. These components form an integrated lacrimal functional unit. Dysfunction of one component can destabilize the entire system.

Tear-film instability and hyperosmolarity

Tears must remain evenly distributed between blinks. Reduced tear secretion, excessive evaporation or abnormal lipid composition shortens tear-film breakup time. Evaporation and reduced tear volume increase tear osmolarity, placing osmotic stress on epithelial cells.

Hyperosmolarity activates inflammatory pathways and promotes release of cytokines and matrix-degrading enzymes. Ocular-surface damage then reduces mucin expression, impairs epithelial wettability and further destabilizes the tear film. This self-perpetuating interaction is frequently described as the dry-eye vicious cycle.

Aqueous-deficient dry eye

Aqueous deficiency results from reduced secretion by the lacrimal glands. It may be associated with ageing, Sjögren disease, graft-versus-host disease, lacrimal gland injury, systemic medications or sensory impairment.

Sjögren disease is particularly important because dry eye may precede recognition of systemic autoimmunity. Patients with marked aqueous deficiency, dry mouth, salivary swelling, arthralgia or systemic features require evaluation for autoimmune disease. Severe dry eye should not be treated indefinitely with lubricants without considering an underlying systemic diagnosis.

Evaporative dry eye and meibomian gland dysfunction

Meibomian glands produce the lipid layer that limits tear evaporation. Gland obstruction, altered secretion and structural loss increase evaporative stress. Meibomian gland dysfunction is one of the most common contributors to dry eye.

Blepharitis, rosacea, contact-lens wear, ageing, incomplete blinking and some cosmetic practices may aggravate gland dysfunction. Digital tasks can reduce blink frequency and increase incomplete blinking, prolonging exposure of the ocular surface.

The distinction between aqueous-deficient and evaporative disease is clinically useful but not absolute. Many patients have mixed disease, and treatment aimed at only one mechanism may fail.

Neurosensory abnormalities

Symptoms may be disproportionately severe despite minimal staining or apparently normal tear tests. In some patients, peripheral nerve injury or central sensitization produces neuropathic ocular pain. Burning, light sensitivity, wind sensitivity and pain described as electric or severe may suggest a neurosensory component.

The opposite pattern also occurs: marked ocular-surface damage may produce relatively few symptoms when corneal sensitivity is reduced, as in diabetes, neurotrophic disease or following ocular surgery. Poor agreement between signs and symptoms is therefore a biological feature rather than merely measurement error.

Risk Factors and Associated Conditions

Dry eye usually results from several interacting factors. Non-modifiable risks include ageing, female sex, autoimmune disease and certain anatomical characteristics. Modifiable contributors include contact-lens misuse, smoking, poor eyelid hygiene, prolonged uninterrupted visual tasks and exposure to air conditioning, fans, dust or low humidity.

Systemic medications associated with dry-eye symptoms include antihistamines, anticholinergic medicines, some antidepressants, diuretics, retinoids and other agents that reduce secretion or alter ocular-surface function. Medication review is particularly important in older adults receiving multiple drugs.

Topical ophthalmic medicines can also cause or worsen disease. Preservatives, especially with frequent or long-term exposure, may damage epithelial cells and increase inflammation. Patients using several glaucoma drops are particularly vulnerable. Preservative-free formulations may reduce exposure but are often more expensive.

Ocular surgery can disrupt corneal nerves and destabilize the tear film. Cataract, refractive, corneal and glaucoma procedures may aggravate pre-existing disease. The ocular surface should therefore be assessed and optimized before surgery, both to improve patient comfort and to enhance the accuracy of keratometry and intraocular-lens calculations.

Contact lenses alter tear distribution and evaporation. Proper lens selection, cleaning, replacement and wearing schedules are essential. Persistent pain or redness in a lens wearer requires urgent assessment because infectious keratitis may initially resemble dry eye.

Environmental air pollution may contribute through oxidative stress and ocular-surface inflammation, although exposure assessment remains inconsistent. This issue is particularly relevant in densely populated urban and industrial regions.

Digital-Device Use and Dry Eye

Digital-device use is strongly associated with eye fatigue and dry-eye symptoms, but the relationship is more nuanced than the simple claim that screens "damage the eyes." Concentrated near work reduces blink frequency and increases incomplete blinking. Small fonts, glare, poor contrast, prolonged sessions and an elevated monitor position may intensify exposure.

Screen time is difficult to measure accurately and is correlated with indoor air conditioning, reduced outdoor activity, sleep disruption and occupational stress. Most studies are cross-sectional, so symptomatic individuals may also use devices differently or report exposure more readily.

Prevention should focus on feasible behavioural and ergonomic measures. Users should take regular visual breaks, blink fully, position screens slightly below eye level, reduce glare, use appropriate refractive correction and avoid direct airflow towards the face. The widely promoted "20-20-20 rule"—looking approximately 20 feet away for 20 seconds every 20 minutes—is easy to remember, but its specific superiority over other regular-break strategies is not firmly established.

Rigid recommendations to eliminate screen use are unrealistic for students and workers. Workplace approaches should include adjustable screens, adequate font size, humidity control, scheduled breaks and access to eye assessment when symptoms persist.

Diagnosis: Beyond a Single Test

Dry eye cannot be diagnosed reliably using one symptom, one questionnaire or one clinical measurement. TFOS DEWS III recommends a structured process combining symptoms, history, observation, tests of tear-film homeostasis and assessment of contributing mechanisms.[1]

Validated questionnaires such as the Ocular Surface Disease Index or the Dry Eye Questionnaire can quantify symptoms and monitor change. They are screening and outcome tools rather than stand-alone diagnostic tests.

Tear-film breakup time assesses stability. Fluorescein, lissamine green or rose bengal staining demonstrates epithelial damage, although staining patterns vary. Tear osmolarity and inflammatory-marker tests may provide additional information, but variability between eyes and over time limits interpretation.

The Schirmer test estimates aqueous tear production but has limited reproducibility and can be influenced by reflex tearing. Meibomian glands should be assessed through lid-margin examination, expressibility, secretion quality and, where available, meibography.

Clinicians must exclude conditions that mimic dry eye, including allergy, conjunctivitis, blepharitis, recurrent corneal erosion, exposure keratopathy, contact-lens complications, infection, episcleritis and medication toxicity. Severe pain, sudden visual loss, marked photophobia, corneal opacity, trauma or unilateral redness requires urgent assessment rather than empirical lubricant treatment.

The mismatch between signs and symptoms has practical implications. Treatment should not be escalated solely because one test is abnormal, nor should severe symptoms be dismissed when staining is minimal. Repeated assessment and attention to neurosensory pain may be necessary.

Management: A Mechanism-Based Approach

The objective of treatment is to restore homeostasis, reduce symptoms, protect the ocular surface and improve visual function. Because dry eye is heterogeneous, no single therapy is effective for every patient.

Table 1. Major Dry-Eye Mechanisms, Clinical Features and Management Priorities

Mechanism or clinical context	Typical findings	Important contributors	Principal management priorities	Important cautions
Aqueous-deficient dry eye	Low tear volume, reduced meniscus, short breakup time and epithelial staining	Ageing, Sjögren disease, lacrimal injury and systemic medications	Lubrication, tear conservation, anti-inflammatory treatment and investigation of systemic disease	Severe deficiency may cause corneal damage and should not be managed with lubricants alone
Evaporative dry eye	Rapid breakup, lid-margin abnormalities and poor meibum quality	Meibomian gland dysfunction, blepharitis, rosacea, incomplete blinking and environmental exposure	Warm compresses, eyelid therapy, blink training, lipid-containing tears and selected gland-directed procedures	Excessive heat or aggressive lid manipulation can irritate the ocular surface
Mixed dry eye	Features of both aqueous and evaporative disease	Chronic disease, ageing, surgery and multiple exposures	Treat each contributing mechanism rather than assigning a single label	Failure to recognize mixed disease commonly leads to incomplete response
Digital-task-related disease	Symptoms worsen during screen work; reduced or incomplete blinking	Prolonged concentration, glare, small text, air conditioning and poor ergonomics	Regular breaks, complete blinking, ergonomic adjustment and environmental modification	Screen duration alone does not establish causation or disease severity
Inflammatory ocular-surface disease	Persistent symptoms, staining and conjunctival or lid inflammation	Chronic tear instability, autoimmune disease, allergy and preservative exposure	Short-term corticosteroid when appropriate, longer-term immunomodulation and treatment of associated disease	Corticosteroids require monitoring for pressure elevation, cataract and infection
Contact-lens-associated disease	End-of-day discomfort, reduced wearing time and fluctuating vision	Lens material, poor fit, deposits, overwear and cleaning solutions	Review lens fit and regimen, improve surface disease and consider daily disposable lenses	Pain, infiltrate or reduced vision may indicate microbial keratitis
Postoperative dry eye	New or worsened symptoms after cataract or refractive surgery	Corneal nerve disruption, topical medicines and pre-existing disease	Preoperative detection, perioperative lubrication and mechanism-based treatment	Untreated disease can affect surgical measurements and satisfaction
Neuropathic ocular pain	Severe burning, photoallodynia or wind sensitivity with limited surface signs	Nerve injury, chronic inflammation and central sensitization	Exclude active surface disease, assess pain phenotype and use multidisciplinary care	Repeated topical escalation may fail when central sensitization predominates
Sjögren-associated disease	Severe aqueous deficiency, dry mouth and systemic symptoms	Autoimmune exocrinopathy	Rheumatological evaluation, ocular-surface protection and systemic care	Risk of epithelial breakdown, infection and progressive corneal damage
Medication-related disease	Symptoms begin or worsen after systemic or topical treatment	Anticholinergic drugs, antihistamines, retinoids and preserved eye drops	Medication review, dose or formulation adjustment and preservative reduction	Essential medicines should not be stopped without consultation

Education and environmental modification

Patients should understand that dry eye is often chronic and fluctuating. Treatment expectations should be realistic. Humidification, avoidance of direct fan or air-conditioner flow, smoking cessation, adequate sleep and management of prolonged visual tasks can reduce exposure.

Medication review and treatment of associated allergy, blepharitis or rosacea may be more valuable than repeatedly changing lubricant brands.

Tear supplementation

Artificial tears remain the foundation of symptomatic care.[2] Formulations differ in viscosity, osmolarity, lipid content and preservatives. Low-viscosity products interfere less with vision but may require frequent use; gels and ointments last longer but can blur vision.

Preservative-free products are preferable when drops are required frequently, in severe disease, after surgery or when the ocular surface is sensitive. Artificial tears improve lubrication but may not correct inflammation, gland obstruction or neuropathic pain.

Autologous serum and platelet-derived products contain epitheliotrophic factors and may benefit severe or refractory disease. Their use requires standardized preparation, microbiological safety and appropriate storage.

Eyelid and meibomian gland therapy

Warm compresses can soften abnormal meibum, but adequate temperature and duration are difficult to achieve consistently with household methods. Lid hygiene is useful when crusting or blepharitis is present; excessive cleansing can worsen irritation.

In-office thermal pulsation, intense pulsed light and other gland-directed technologies have expanded. Some trials report improvements in symptoms or gland function, particularly in selected meibomian gland dysfunction. However, devices are expensive, protocols vary and high-quality comparative evidence remains limited. They should not be promoted as universally curative.

Anti-inflammatory treatment

Inflammation is central to many forms of persistent disease. Short courses of topical corticosteroids may rapidly suppress inflammation but require ophthalmic supervision because prolonged use can elevate intraocular pressure, promote cataract and worsen infection.

Topical cyclosporine and lifitegrast reduce inflammatory signalling and can improve selected symptoms or signs. Response may take weeks or months, and burning on instillation can reduce adherence. No currently approved anti-inflammatory agent has demonstrated universal superiority in direct comparisons.[5]

Treatment should be guided by clinical phenotype and response rather than commercial availability alone. Patients with allergy require appropriate anti-allergic therapy, while those with autoimmune disease may need coordinated systemic management.

Tear conservation and stimulation

Punctal plugs reduce tear drainage and may benefit aqueous-deficient disease. Inflammatory ocular-surface disease should generally be controlled before occlusion because retaining highly inflammatory tears may aggravate symptoms. Complications include extrusion, irritation, infection and, rarely, migration.

Secretagogues stimulate tear production through neural or pharmacological pathways. Varenicline nasal spray activates the trigeminal parasympathetic pathway and has shown improvement in tear production in clinical trials, although availability, nasal adverse effects and cost may limit use.

Omega-3 fatty acids

Omega-3 supplements have been widely recommended because of proposed anti-inflammatory effects. The multicentre DREAM randomized trial found that high-dose n-3 fatty-acid supplementation was not superior to an olive-oil placebo for improving dry-eye symptoms or signs over one year.[6]

This does not prove that dietary fatty-acid quality is irrelevant or that no subgroup could benefit. It does mean that routine high-dose supplementation should not be presented as an evidence-based treatment for all patients. Product composition, cost, bleeding risk and possible interactions should be considered.

Severe disease and advanced care

Severe ocular-surface disease may require scleral lenses, moisture-chamber spectacles, amniotic membrane, tarsorrhaphy or other protective procedures. Scleral lenses create a fluid reservoir over the cornea and can markedly improve comfort and vision, but fitting requires expertise and meticulous hygiene.

Persistent pain despite adequate surface treatment should prompt evaluation for neuropathic pain. Management may require pain specialists, neurologists, psychiatrists or psychologists. This does not imply that symptoms are imaginary; it recognizes that chronic ocular pain may involve peripheral and central neural pathways.

Public Health Significance

Dry eye affects daily functioning beyond ophthalmic discomfort. Fluctuating vision reduces reading speed and accuracy, while burning and photophobia interfere with driving and screen work. Productivity loss may occur through reduced concentration, repeated breaks and health-care visits.

The condition is associated with poorer sleep, anxiety and depressive symptoms. Directionality is uncertain: chronic symptoms may impair mental well-being, while anxiety and heightened sensory processing may increase symptom burden. Integrated assessment is appropriate when distress is substantial.

Dry eye also has implications for surgical outcomes. Unrecognized disease can compromise preoperative measurements and contribute to dissatisfaction after otherwise successful cataract or refractive surgery. Ocular-surface evaluation should therefore become part of routine surgical planning.

From a health-system perspective, indiscriminate use of over-the-counter drops can delay diagnosis, increase cost and expose patients to preservatives or inappropriate vasoconstrictors. Public education should emphasize that persistent symptoms require examination rather than indefinite self-medication.

Indian Perspective

India has several factors likely to increase dry-eye burden: a large ageing population, rapid expansion of smartphone and computer use, substantial air pollution, hot or arid climates in many regions, widespread two-wheeler travel, and increasing cataract and refractive surgery volumes.

A hospital-based study from North India reported dry eye in 32% of more than 31,000 patients attending a tertiary eye centre. Digital-screen exposure, smoking and contact-lens use were associated with higher odds of disease.[7] The study's large sample is valuable, but it represents care-seeking patients rather than the general population and should not be interpreted as national prevalence.

A 2025 prospective database study at a tertiary centre in central India estimated dry eye in approximately one-quarter of attendees and identified associations with several occupations, ocular allergy, smoking, uncorrected refractive error and contact-lens use.[8] Again, referral and selection effects limit population generalizability.

Available Indian studies differ in questionnaires, staining protocols, breakup-time thresholds and Schirmer criteria. Estimates from different centres are therefore not directly comparable. Nationally representative population-based studies using standardized contemporary criteria are lacking.

The disease may be under-recognized in rural areas, where symptoms can be attributed to dust, smoke or ageing and access to slit-lamp examination is limited. Conversely, urban commercial eye care may overdiagnose disease through isolated abnormal tests or device-based screening.

Primary and community eye-care workers can identify persistent symptoms, review environmental and medication risks and refer patients with pain, visual loss, autoimmune symptoms or corneal abnormalities. However, diagnosis and prescription of anti-inflammatory treatment require trained eye-care professionals.

Occupational health is particularly relevant. Employers in information technology, education, banking and other screen-intensive sectors should provide ergonomic workstations, appropriate lighting, regular breaks and referral pathways. Outdoor workers may benefit from protective eyewear reducing wind, dust and ultraviolet exposure.

Recent Advances

The most important conceptual advance is the move towards phenotype-based treatment. Instead of prescribing the same lubricant to every patient, clinicians increasingly assess aqueous secretion, evaporation, meibomian function, inflammation, lid closure and neural pain.

Non-invasive imaging has expanded through meibography, tear-film interferometry, thermography and high-resolution ocular-surface imaging. These technologies may improve phenotyping, although their additional value over careful clinical examination and their cost-effectiveness remain uncertain.

Home-based and digital monitoring tools are being developed to measure blinking, symptoms and treatment adherence. Artificial intelligence may assist image analysis or classification, but dry eye lacks a single objective reference standard. Algorithms trained against inconsistent labels may reproduce diagnostic uncertainty rather than resolve it.

New therapies include tear neurostimulation, water-free drug-delivery systems, novel anti-inflammatory agents and regenerative approaches. Recent approvals broaden treatment options but do not eliminate the need to identify underlying mechanisms.

The TFOS DEWS III reports also place greater emphasis on symptom–sign discordance, neurosensory abnormalities, lifestyle exposures and individualized care.[1,2] This represents a shift away from viewing dry eye as a simple tear-deficiency disorder.

Challenges and Limitations

The greatest research limitation is the absence of a universally applied diagnostic standard. Studies use different symptom scales and thresholds for breakup time, osmolarity, staining and tear production. This creates extreme heterogeneity in prevalence estimates and treatment trials.

Clinical endpoints are also problematic. Symptoms fluctuate with weather, work, sleep and expectations. Signs vary between visits and may improve without corresponding symptom relief. Trials frequently test multiple outcomes, increasing the possibility of selective positive findings.

Placebo effects can be substantial because control drops may themselves lubricate the eye. Masking is difficult for procedures, and industry funding is common in device and pharmaceutical studies. Transparent protocols and independent comparative trials are needed.

Another challenge is overcommercialization. Patients may be offered costly imaging, nutritional products or repeated device-based procedures without clear evidence that these will improve patient-important outcomes. More technology does not automatically mean more precise or effective care.

Finally, prevention evidence is less developed than treatment evidence. Common recommendations concerning hydration, screen breaks and dietary supplements are often biologically plausible but supported by limited controlled research.

Future Directions

Population-based studies should apply standardized TFOS-compatible definitions and report symptoms, signs and mechanisms separately. India requires nationally coordinated surveys across rural, urban, climatic and occupational settings.

Longitudinal studies are needed to determine whether digital exposure, pollution and childhood disease predict persistent dry eye rather than transient symptoms. Objective device-use data and personal environmental monitoring would reduce recall error.

Treatment trials should recruit clearly defined phenotypes and use core outcome sets incorporating symptoms, visual function, tear stability, corneal integrity, adverse effects and quality of life. Head-to-head comparisons are more useful than repeated comparisons with inert or lubricating controls.

Affordable diagnostic and treatment pathways are essential for low-resource settings. Research should determine which limited set of tests provides the greatest clinical value in primary and secondary eye care.

Ocular-surface health should be incorporated into workplace wellness, school eye health and preoperative ophthalmic protocols. Public communication should encourage regular blinking and breaks without generating fear that ordinary screen use inevitably damages the eyes.

Greater attention is also required for neuropathic ocular pain and mental-health comorbidity. Patients with severe symptoms and minimal signs should receive careful evaluation rather than dismissal or endless escalation of topical drops.

CONCLUSION

Dry eye disease is a common, chronic and heterogeneous disorder affecting the tear film, ocular surface, eyelids, lacrimal glands and sensory nervous system. Its clinical importance extends beyond discomfort to fluctuating vision, impaired productivity, reduced quality of life and, in severe cases, corneal damage.

Diagnosis requires both symptoms and structured evaluation. No single questionnaire or tear test is sufficient, and discordance between signs and symptoms is common. Management should address the dominant mechanisms, including aqueous deficiency, evaporation, meibomian gland dysfunction, inflammation, environmental exposure and neuropathic pain.

Artificial tears remain central, but persistent disease may require eyelid therapy, anti-inflammatory treatment, tear conservation, secretagogues, scleral lenses or other specialized care. Routine omega-3 supplementation is not supported for all patients, and expensive device-based treatment should be offered selectively.

India likely carries a large and increasing burden, but national prevalence remains uncertain because current evidence is dominated by tertiary-centre studies using variable criteria. Standardized community research, occupational prevention, improved primary-care recognition and affordable specialist services are needed.

Dry eye should not be trivialized as an inevitable consequence of screens or ageing. Equally, it should not be overdiagnosed through isolated tests. A balanced, evidence-based and mechanism-oriented approach offers the best prospect of protecting ocular comfort, vision and everyday functioning.

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