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Meibomian Gland Dysfunction: Emerging Diagnostic Technologies and Novel Therapeutic Approaches

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Abstract

Meibomian gland dysfunction (MGD) is a chronic disorder of the eyelid margin and the leading cause of evaporative dry eye disease (EDE). Alterations in meibum secretion impair tear film stability, increase tear evaporation, and contribute to ocular surface inflammation and visual disturbances. This narrative review summarizes current knowledge regarding the anatomy and physiology of the meibomian glands, the pathophysiology of MGD, its relationship with dry eye disease (DED), contemporary diagnostic methods, and available treatment options. Recent advances in meibography, interferometry, lipid layer assessment, and artificial intelligence have improved diagnostic capabilities. In addition to conventional therapies, including eyelid hygiene, warm compresses, and anti-inflammatory treatment, device-based interventions such as thermal pulsation therapy, intense pulsed light, and low-level light therapy have expanded therapeutic possibilities. Emerging regenerative approaches may further improve future management strategies. Continued advances in diagnosis and treatment are expected to support more personalized and mechanism-based care for patients with MGD.

Keywords: meibomian gland dysfunction; dry eye disease; evaporative dry eye; meibography; tear film; treatment.

1. Introduction

1.1 Anatomy and Physiology of the Meibomian Glands

Meibomian glands are modified sebaceous glands embedded within the tarsal plates of the upper and lower eyelids, arranged in a vertical orientation relative to the eyelid margin. They were first described by Heinrich Meibom in 1666 and represent the largest sebaceous glands in the human body. Histologically, they exhibit a lobular architecture composed of multiple acini that drain into a branched ductal system culminating in a central excretory duct, which opens at the posterior eyelid margin. The upper eyelid contains a higher density of meibomian glands and a greater lipid reservoir compared to the lower eyelid. These glands secrete meibum, a lipid-rich secretion delivered through holocrine secretion that forms the outermost layer of the tear film. Meibum reduces tear evaporation, contributes to tear film stability, regulates tear film surface tension, and supports ocular surface homeostasis. Because of these essential functions, proper meibomian gland activity is critical for maintaining eye comfort and visual quality [1-3].

1.2 Meibomian Gland Dysfunction: Definition and Clinical Significance

Disruption of normal meibomian gland function leads to MGD, a chronic and multifactorial disorder that represents the leading cause of EDE. MGD is characterized by obstruction of the gland ducts and/or alterations in the quantity or quality of meibum secretion. These abnormalities compromise tear film stability, resulting in increased tear evaporation, ocular surface inflammation, and symptoms such as dryness, burning, irritation, discomfort, and foreign body sensation. The condition is highly prevalent in clinical practice and is estimated to account for approximately 40-60% of dry eye cases [2,3]. In addition to diagnostic challenges, MGD encompasses several clinical subtypes that differ in their underlying mechanisms and patterns of meibum secretion. MGD can be classified into low-delivery and high-delivery forms based on the amount of meibum reaching the ocular surface. Low-delivery MGD, which includes obstructive and hyposecretory subtypes, is the most common form encountered in clinical practice. Obstructive MGD is characterized by blockage of the gland ducts, whereas hyposecretory MGD involves reduced meibum production. In contrast, high-delivery MGD is associated with excessive lipid secretion and is observed less frequently. Among these subtypes, obstructive MGD is the primary cause of EDE. Reduced lipid delivery to the tear film compromises its stability and increases tear evaporation, leading to ocular surface inflammation and symptoms such as dryness, irritation, and visual discomfort [4].

1.3 Pathophysiology of MGD

The pathophysiology of MGD is complex and not yet fully understood. Several factors have been implicated in its development, including congenital abnormalities, reduced blink frequency, chronic ocular surface inflammation, age-related changes, and hormonal imbalances. Among these factors, sex hormones, particularly androgens have attracted considerable attention because of their regulatory effects on meibomian gland structure and function. Consequently, androgen replacement therapy and other hormone-based approaches are being investigated as potential treatment strategies [2]. Given the critical role of meibomian gland-derived lipids in maintaining tear film stability, dysfunction of these glands is closely associated with tear film instability and the development of EDE which represents one of the most common clinical consequences of MGD [5].

2. Dry Eye Disease

2.1 Definition and Clinical Classification

DED is a multifactorial disorder of the ocular surface characterized by loss of tear film homeostasis and ocular symptoms such as dryness, irritation, burning sensation, and visual disturbance [6]. Current evidence indicates that DED is primarily driven by tear film instability, hyperosmolarity, inflammation, and neurosensory abnormalities [7]. DED is broadly classified into aqueous-deficient and evaporative forms, with MGD representing the leading cause of EDE [5,7].

2.2 Pathophysiology and Link to Meibomian Gland Dysfunction

The tear film is a highly organized and dynamic structure composed of lipid, aqueous, and mucin layers that work in close coordination to maintain ocular surface integrity, ensure optical stability, and provide comfortable vision. Each component plays a distinct but interdependent role, and disruption of any of these layers may compromise tear film stability, ultimately resulting in ocular surface disease. EDE is most frequently associated with MGD, which represents the major cause of this subtype [4]. In MGD, abnormalities in meibomian gland function result in altered quantity and quality of meibum secretion. This leads to an unstable or insufficient lipid layer, which increases tear evaporation and contributes to tear film hyperosmolarity. As a consequence, ocular surface homeostasis is disrupted, predisposing to clinical symptoms and disease progression. Given the central role of the meibomian glands in maintaining the stability of the tear film lipid layer, MGD is widely recognized as the primary etiological factor in EDE and a key target in both understanding and managing ocular surface disorders [4,5].

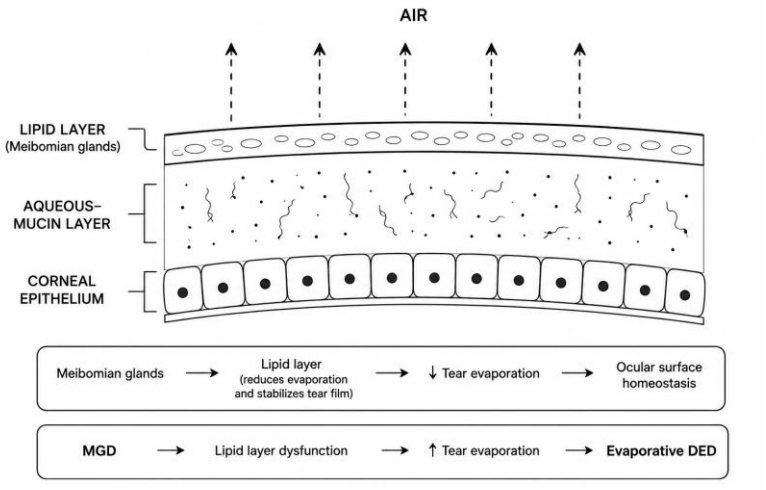

Figure 1. Schematic representation of the tear film structure. The outer lipid layer, produced by the meibomian glands, reduces tear evaporation and stabilizes the tear film. Dysfunction of

the meibomian glands results in lipid layer deficiency, increased evaporation, and development of EDE.

3. Risk Factors of Dry Eye Disease

Age, female sex, prolonged digital screen use, contact lens wear, low humidity, rosacea, blepharitis, and autoimmune diseases are recognized risk factors for DED and may contribute to the development or progression of MGD [4-8].

4. Clinical Presentation and Diagnosis

According to the TFOS DEWS III diagnostic methodology report, DED is a multifactorial, symptomatic disorder characterized by loss of homeostasis of the tear film and/or ocular surface, in which tear film instability, hyperosmolarity, ocular surface inflammation and damage, and neurosensory abnormalities contribute to disease development [9]. Contemporary diagnostic approaches therefore follow a stepwise strategy that first confirms the presence of DED and subsequently identifies the underlying etiological drivers, including MGD, which represents the leading cause of EDE [3,9].

4.1 Diagnosis of Dry Eye Disease

The diagnostic process begins with a detailed medical history and symptom evaluation. Patients with DED commonly report ocular dryness, burning, stinging, foreign body sensation, itching, fluctuating vision, visual fatigue, photophobia, redness, excessive tearing, and increased mucus secretion. Symptoms often worsen during prolonged visual tasks, such as reading or computer use, and in environments characterized by low humidity or increased airflow. Importantly, symptom severity does not always correlate with objective clinical findings, highlighting the need for both subjective and objective assessment [3,10]. Symptom burden is commonly assessed using validated questionnaires such as OSDI, DEQ-5, and SPEED, which support diagnosis and evaluation of disease impact on daily functioning [9,11]. Current recommendations require the presence of symptoms together with at least one sign indicating loss of tear film homeostasis. Consequently, diagnostic testing should be performed following symptom assessment. To minimize interference between measurements, non-invasive procedures should be carried out before tests that may alter tear film properties, such as fluorescein instillation. Recommended examinations include tear film break-up time, ocular surface staining, and tear osmolarity assessment [9]. Tear film stability is typically evaluated using either fluorescein tear break-up time (TBUT) or non-invasive tear break-up time

(NIBUT). NIBUT is increasingly preferred because it avoids disruption of the tear film associated with fluorescein instillation. Reduced break-up time supports the diagnosis of DED by indicating tear film instability; however, it does not independently differentiate between evaporative and aqueous-deficient forms of disease [10]. Ocular surface staining provides valuable information regarding epithelial damage resulting from tear film instability and ocular surface inflammation. Fluorescein is most commonly used for corneal staining, whereas lissamine green facilitates evaluation of conjunctival involvement. The extent and distribution of staining correlate with disease severity and constitute important markers of ocular surface homeostasis loss. Tear osmolarity represents another quantitative marker of tear film homeostasis. Increased osmolarity reflects tear film instability and hyperosmolar stress, both of which are central features of DED pathophysiology. Although osmolarity testing may support diagnosis, results should always be interpreted in conjunction with symptoms and other clinical findings. The frequently observed discordance between symptoms and clinical signs may be related to neurosensory abnormalities, variable inflammatory activity, and the multifactorial nature of DED [9,10].

4.2 Identification of MGD as the Cause of Evaporative Dry Eye

After confirmation of DED, evaluation should identify the underlying etiological drivers, including tear film deficiencies, eyelid abnormalities, ocular surface pathology, and systemic contributors. Within this framework, MGD represents an eyelid-related driver and the principal cause of lipid-deficient evaporative dry eye [9,10]. Evaluation of tear volume remains important for excluding or identifying an aqueous-deficient component. This may include measurement of tear meniscus height and performance of the Schirmer test. Although Schirmer values are frequently normal in isolated MGD, assessment of tear production remains useful for differentiating evaporative and mixed forms of DED. Therefore, Schirmer testing continues to play an important role in DED subtype classification. Nevertheless, the Schirmer test is characterized by considerable inter- and intra-observer variability, which may limit its reproducibility in both clinical practice and research settings [12]. Slit-lamp biomicroscopy remains the cornerstone of MGD diagnosis. Examination should include evaluation of the eyelid margins, meibomian gland orifices, blink pattern, and ocular surface. Characteristic findings include lid margin thickening, telangiectasia, vascular engorgement, mucocutaneous junction displacement, gland orifice obstruction, posterior blepharitis, and foamy secretions. Particular attention should also be paid to dermatological conditions such as rosacea and

seborrheic dermatitis, which are frequently associated with MGD [3,10]. Direct evaluation of meibomian gland function is essential for confirming the diagnosis. Meibum expressibility is determined by applying standardized pressure to the eyelid and assessing the number of glands capable of producing secretions. Reduced expressibility reflects ductal obstruction and impaired gland function and is considered a hallmark of obstructive MGD. However, despite its clinical utility, assessment of meibum expressibility remains partly examiner-dependent, and differences in the force applied during gland expression may affect measurement reproducibility and interobserver agreement [13]. The quality of expressed meibum is evaluated according to its appearance and consistency. Healthy glands typically produce clear and fluid secretions, whereas MGD is associated with cloudy, particulate, thickened, or toothpaste-like meibum. These changes reflect alterations in lipid composition and increased viscosity of glandular secretions, contributing to tear film instability and accelerated evaporation [10,13].

4.3 Advanced Clinical Diagnostic Techniques

Although conventional clinical examination remains sufficient for diagnosing most cases of MGD, several advanced diagnostic techniques have emerged in recent years and provide valuable information regarding meibomian gland structure, tear film function, and disease severity. Among these, meibography has become one of the most important imaging modalities in MGD assessment. Infrared meibography enables direct visualization of meibomian gland architecture and allows evaluation of gland shortening, tortuosity, distortion, dilation, and gland dropout. Structural abnormalities identified by meibography have been shown to correlate with disease severity and may detect structural gland alterations in patients with relatively mild clinical findings, making this technique particularly valuable for diagnosis and longitudinal monitoring of disease progression. [10,14]. Beyond morphological evaluation, increasing attention has been directed toward objective assessment of the tear film lipid layer. Interferometry provides a non-invasive method for analyzing interference patterns generated by reflected light, enabling both qualitative and quantitative characterization of lipid layer properties and tear film dynamics. This technique may facilitate differentiation between evaporative and aqueous-deficient mechanisms of dry eye and offers insight into tear film stability and lipid layer behavior [15]. One of the most widely used quantitative parameters derived from interferometric assessment is lipid layer thickness (LLT), commonly measured using devices such as LipiView. Reduced LLT has been associated with MGD, increased tear evaporation, gland dropout, and tear film instability. Because lipid deficiency is now recognized as one of the principal drivers of EDE, LLT measurement may offer useful information

regarding disease severity and treatment response [9,16]. Recent studies have also highlighted the value of objective optical quality assessment systems. Among these, double-pass imaging systems such as the Optical Quality Analysis System (OQAS) enable quantitative assessment of intraocular light scatter and dynamic changes in optical quality associated with tear film instability. Parameters derived from these technologies correlate with tear film instability, meibomian gland expressibility, gland dropout, ocular surface abnormalities, and overall disease severity. A comparative study demonstrated significantly higher objective scatter index (OSI), ΔOSI, ΔOSI/time, and blinking-related parameters in patients with MGD than in those with aqueous-deficient dry eye (all $P < 0.01$), suggesting that MGD may exert a particularly pronounced impact on visual quality through disruption of tear film stability. By quantifying the impact of tear film disruption on visual quality, these systems may complement traditional clinical metrics and provide additional insight into the functional consequences of MGD-related tear film instability. Together, these techniques provide complementary information regarding gland morphology, tear film stability, lipid layer characteristics, and visual quality [17].

4.4 Emerging and Research-Oriented Diagnostic Technologies

Additional emerging diagnostic techniques include tear biomarker analysis, MMP-9 testing, impression cytology, in vivo confocal microscopy, AS-OCT, and AI-assisted meibography analysis. These methods may improve characterization of ocular surface inflammation, meibomian gland morphology, and tear film dynamics [18,19]. Tear biomarker analysis, particularly assessment of MMP-9 and inflammatory cytokines, may provide additional information regarding ocular surface inflammation; however, lack of standardization currently limits routine clinical implementation [18]. Other advanced imaging modalities may provide additional structural and microstructural information regarding the ocular surface and meibomian glands. In vivo confocal microscopy enables high-resolution visualization of meibomian gland acini, inflammatory cell infiltration, and microscopic tissue alterations that are not detectable by routine slit-lamp examination. Similarly, AS-OCT may be used to assess tear meniscus parameters and ocular surface anatomy, providing complementary information regarding tear volume and tear film dynamics. However, the widespread use of these technologies remains limited by equipment availability, examination time, cost, and the need for specialized expertise in image acquisition and interpretation [9,10]. In particular, growing interest has focused on the application of artificial intelligence algorithms for automated meibography analysis, gland segmentation, and quantitative assessment of meibomian gland

dropout. Artificial intelligence-based approaches have demonstrated the potential to improve the objectivity, reproducibility, and efficiency of MGD evaluation while facilitating automated severity classification and large-scale image analysis. Nevertheless, most currently available models remain under clinical validation, and their performance across different imaging platforms, patient populations, and clinical settings requires further investigation before widespread implementation can be recommended. Additionally, the lack of standardized datasets and external validation remains an important limitation of many currently published algorithms [19,20]. Overall, these emerging technologies may improve disease phenotyping, facilitate more individualized therapeutic decision-making, and enhance understanding of the complex pathophysiology of MGD-related DED. At present, however, they should be regarded primarily as adjunctive or research-oriented tools rather than first-line diagnostic methods [18-20].

5. Treatment of Meibomian Gland Dysfunction

5.1 Conventional Management

The management of MGD aims to restore meibomian gland function, improve tear film stability, alleviate symptoms, and prevent progressive ocular surface damage. Because MGD is a chronic and multifactorial disorder, treatment often requires a long-term and individualized approach. Current management strategies combine patient education, lifestyle modifications, eyelid hygiene, pharmacological therapy, and device-based interventions according to disease severity and underlying pathogenic mechanisms [3]. Conservative measures remain the foundation of MGD treatment. Patient education regarding the chronic nature of the disease, environmental risk factors, screen use habits, and blinking behavior is essential for long-term therapeutic success. Patients are advised to minimize exposure to factors that increase tear evaporation, including low humidity, air conditioning, wind, and prolonged digital screen use. Particular attention should be paid to blink dynamics, as incomplete or infrequent blinking may impair meibum distribution across the ocular surface, disrupt lipid layer spreading, and contribute to tear film instability [10,16]. Daily eyelid hygiene and warm compresses are commonly recommended as first-line therapy. Heat application facilitates liquefaction of altered meibum, improves glandular secretion, and promotes relief of ductal obstruction. Lid massage and gland expression may further enhance meibum outflow, improve gland patency, and contribute to tear film stabilization. These interventions are inexpensive, widely available, and remain a cornerstone of treatment across all stages of disease severity [3]. Maintenance of tear film homeostasis requires adequate interaction between the lipid and aqueous components

of the tear film. The ocular surface environment may therefore be supported by artificial tears, particularly lipid-containing formulations designed to supplement the deficient tear film lipid layer and reduce excessive evaporation. Restoration of lipid layer integrity remains a principal therapeutic goal in MGD because lipid deficiency plays a central role in the pathogenesis of EDE [12,16]. Dietary supplementation with omega-3 fatty acids has also been investigated as a potential adjunctive therapy because of its proposed anti-inflammatory effects and possible influence on meibomian gland secretions; however, available evidence remains inconsistent, and its role in routine management continues to be debated [3]. Pharmacological interventions may be considered in patients with moderate-to-severe disease or significant inflammatory involvement. Topical azithromycin and oral tetracyclines, including doxycycline and minocycline, exert both antimicrobial and anti-inflammatory effects and may improve meibomian gland function by reducing eyelid margin inflammation and modifying meibum composition. In selected cases, short-term topical corticosteroids may be used to control acute ocular surface inflammation, whereas immunomodulatory therapies such as cyclosporine A can be considered when chronic inflammation contributes to disease persistence. Nevertheless, these therapies primarily target the inflammatory consequences of gland dysfunction rather than directly restoring normal meibomian gland structure and function [3,10]. Despite their benefits, conventional therapies often require long-term adherence and may not adequately address established gland obstruction. Persistent tear film instability may also impair optical quality and visual performance, supporting the development of device-based therapies designed to improve gland function and provide longer-lasting benefits [10,17].

5.2 Device-Based Therapies

Among contemporary procedural interventions, thermal pulsation therapy has become one of the most extensively investigated treatments for MGD. Systems such as LipiFlow combine controlled heating of the inner eyelid surface with simultaneous pulsatile pressure applied to the outer eyelid. This approach aims to liquefy obstructed meibum while mechanically evacuating gland contents. Clinical studies suggest potential improvements in meibomian gland secretion, symptom scores, and tear film stability following treatment. However, recent systematic reviews have reported considerable variability among studies and emphasized the need for further high-quality evidence regarding long-term efficacy, comparative effectiveness, and optimal patient selection [21]. Intense pulsed light (IPL) therapy has emerged as an increasingly investigated treatment modality, particularly in patients with obstructive MGD and associated eyelid inflammation. Initially developed for dermatological applications, IPL

delivers high-intensity pulses of non-coherent light to the periocular skin. Several mechanisms have been proposed to explain its therapeutic effects, including reduction of abnormal telangiectatic vessels, decreased inflammatory mediator release, eradication of Demodex mites, and improvement in meibum quality. Clinical studies suggest improvements in symptom severity, tear film stability, meibum expressibility, and meibomian gland function following IPL treatment. Reductions in inflammatory tear cytokines, including IL-6 and TNF-α, have also been reported, suggesting that anti-inflammatory mechanisms may contribute to its therapeutic effects. Although systematic reviews support the potential benefits of IPL, the overall certainty of evidence remains limited because of heterogeneity in treatment protocols, outcome measures, and study designs [13,22]. Another emerging light-based modality is low-level light therapy (LLLT), which utilizes low-energy light-emitting diodes to induce photobiomodulation. Experimental and clinical studies suggest that LLLT may exert anti-inflammatory effects, improve cellular metabolism, and enhance tissue repair. A randomized controlled trial demonstrated improvements in ocular surface staining, Schirmer test values, and selected meibomian gland parameters while maintaining a favorable safety profile. Although current evidence remains more limited than for IPL, LLLT may represent a useful adjunctive therapy for selected patients with MGD-associated dry eye [23]. Intraductal meibomian gland probing has also attracted increasing interest as a potential treatment for obstructive MGD. This procedure involves insertion of a fine probe into the gland duct with the aim of mechanically relieving obstruction and restoring gland patency. Although several studies have reported improvements in symptoms and gland function following probing, controlled trials have produced inconsistent results. Current evidence suggests that the procedure appears relatively safe; however, its effectiveness remains uncertain, and larger placebo-controlled studies are required before widespread adoption can be recommended [24].

5.3 Emerging and Future Therapeutic Approaches

Recent years have witnessed growing interest in regenerative and biologically based therapies for DED and MGD. Among these, autologous serum and platelet-rich plasma (PRP) eye drops have received considerable attention. Unlike conventional lubricants, these biological preparations contain growth factors, cytokines, vitamins, and other bioactive molecules that more closely resemble the composition of natural tears. Clinical studies and recent meta-analyses suggest improvements in tear film stability, ocular surface staining, symptom severity, and tear production following treatment. Although most available evidence concerns DED in general rather than isolated MGD, these therapies may be particularly valuable in patients with

significant ocular surface damage and treatment-refractory disease. Furthermore, blood-derived products may support epithelial regeneration and restoration of ocular surface homeostasis through mechanisms extending beyond simple lubrication, highlighting their potential role as regenerative therapies rather than conventional tear substitutes [25,26]. A first-in-human pilot study of umbilical cord-derived mesenchymal stem cell eye drops demonstrated improvements in symptoms, tear production, tear film stability, and meibomian gland function in patients with refractory DED. However, larger controlled studies are required to confirm these findings [27]. Novel drug-delivery systems are also under development. One particularly innovative approach involves photothermal-responsive soluble microneedle patches designed to deliver therapeutic agents directly through the eyelid. Experimental studies have shown that these systems can simultaneously liquefy obstructed meibum and provide targeted drug delivery to meibomian glands, resulting in improved gland morphology, reduced inflammation, and restoration of glandular function in animal models. The incorporated PPAR-γ agonist rosiglitazone was shown to promote meibocyte differentiation and lipid synthesis, directly targeting mechanisms involved in gland dysfunction. Although currently at a preclinical stage, such technologies illustrate the growing trend toward targeted and precision-based therapies for MGD [28]. Overall, the management of MGD is evolving from traditional symptom-oriented approaches toward personalized, mechanism-based therapeutic strategies. While eyelid hygiene and warm compresses remain fundamental components of care, growing evidence supports the use of device-based therapies and emerging regenerative interventions. Future advances will likely focus on earlier intervention, improved patient stratification, targeted drug delivery, and therapies capable of restoring meibomian gland structure and function rather than simply alleviating symptoms. The integration of regenerative medicine and advanced drug-delivery platforms may further accelerate the transition toward precision medicine in MGD management [29].

Conclusion

MGD is the leading cause of EDE and a major contributor to ocular surface instability and visual discomfort. Advances in diagnostic technologies have improved the assessment of meibomian gland structure and function, while emerging therapeutic approaches have expanded treatment options beyond conventional management. Although eyelid hygiene and warm compresses remain the foundation of therapy, device-based interventions and regenerative strategies show growing potential. Future research should focus on validating emerging technologies and developing therapies that restore meibomian gland function.

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