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DRY EYE DISEASE AND MEIBOMIAN GLAND DYSFUNCTION A CHAIN REACTION

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ABSTRACT

The most prevalent cause of dry eye disease (DED) is dysfunction of the meibomian glands (MGD). MGD is a multifactorial disorder with eyelid inflammation, microbial growth, skin disorders associated with it, and potentially severe corneal complications. MGD may be the result of any combination of the five distinct pathophysiological mechanisms listed below, making it likely that it is a heterogeneous condition: eyelid inflammation, conjunctival inflammation, damage to the cornea, changes in the microbiome, and DED brought on by unstable tear film A "vicious circle" can be used to describe both MGD and DED's pathogenesis: DED and MGD's underlying pathophysiological mechanisms interact, creating a double-whammy. Changes in microbiology can self-stimulate the MGD cycle, causing meibum to melt at a higher temperature and meibomian gland blockage, both of which reinforce the MGD cycle. The two vicious circles are directly connected by inflammation, dropout, and blockage of the meibomian gland. The hyperosmolarity and inflammation that are both a cause and a result of DED are brought on by MGD-associated tear film instability, which serves as a doorway into the DED cycle. To better pinpoint the underlying pathological mechanisms and enable more precise therapeutic targeting, we present a brand-new pathophysiological model for MGD. MGD may be considered a disease rather than merely a dysfunction if this scheme is better understood.

INTRODUCTION

Dry eye disease (DED) is one of the most prevalent ocular surface diseases and can significantly impair a patient's quality of life. Over the course of the past few decades, various definitions of DED have been developed. The Tear Film and Ocular Surface Society (TFOS) Dry Eye Workshop (DEWS) aims to develop an evidence-based definition, a well-defined classification system, and an appropriate diagnosis and management algorithm for dry eye disease (DED).1

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The TFOS DEWS definition of DED was first published in 2007.2 In 2017, the TFOS DEWS II revised the definition to include the phrase "a multifactorial disease of the ocular surface, characterised by a loss of homeostasis of the Conditions that affect the function of the lacrimal gland, such as Sjögren's syndrome, lacrimal gland duct obstruction or deficiencies, and adverse effects of systemic medications are the primary causes of aqueous deficient dry eye. Meibomian glands are located in the upper and lower eyelids, where they secrete lipids (meibum) onto the ocular surface, forming the outermost layer of the tear film. Epidemiological evidence suggests that DED is primarily evaporative in nature and is frequently associated with MGD.5,6 Spreading readily, these lipids aid in tear film stability and prevent evaporation. "a chronic, diffuse abnormality of the meibomian glands, commonly characterized by terminal duct obstruction and/or qualitative/quantitative changes in the glandular secretion" is the definition of meibomian gland dysfunction. It could alter the tear film, cause irritation, inflammation that is clinically evident, and ocular surface disease

PATHOPHYSIOLOGY OF DRY EYE DISEASE

All forms of dry eye disease are primarily caused by the loss of water from the tear film. This causes tear hyperosmolarity in evaporative dry eye and/or aqueous deficient dry eye.10 In evaporative dry eye, hyperosmolarity is caused by too much evaporation of tears in the context of normal lacrimal function. In contrast, hyperosmolarity results from an inadequate rate of lacrimal secretion in the context of a normal rate of evaporation in aqueous deficient dry eyes. The presence of hyperosmolarity on the ocular surface is influenced by the environment, which can either set off the onset of DED or make the condition worse.

SYMPTOMS

There are many DED and MGD symptoms that are similar. However, the majority of MGD patients do not exhibit any symptoms at all; If they are symptomatic, the severity of the ocular surface disturbance is frequently not directly correlated with their specific symptoms. In a population-based study in China, 22% of the participants had asymptomatic MGD, while 9% had symptomatic MGD.8 In cases of symptomatic MGD, patients may experience dryness, itching, foreign body sensation, and/or photosensitivity7. These symptoms may be caused by mechanical friction between the ocular surface and meibum that has accumulated in the gland orifices or by chronic inflammation.

PATHOPHYSIOLOGY OF MEIBOMIAN GLAND DYSFUNCTION

The rate of gland secretion is used to classify meibomian gland dysfunction. Meibomian gland obstruction or hyposecretion are signs of a low delivery state, while meibomian gland hypersecretion is a sign of a high delivery state. Epithelial hyperkeratinization is the most

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common cause of duct obstruction, which results in meibum accumulation, chronic inflammation, and eventually gland dropout12—importantly in the quantitative and qualitative abnormalities of glandular secretions. Of these two categories, the most common mechanism is a low delivery state. There is a high commonness of MGD in skin break out rosacea, which is an ongoing cutaneous problem.

ASSOCIATION BETWEEN DRY EYE DISEASE AND MEIBOMIAN GLAND DYSFUNCTION

The tear film comprises of three layers: the layers of mucus, water, and lipids. Meibomian glands are the source of the lipid layer, a crucial component of the tear film. The lipid layer keeps water dissipation from the visual surface and is in this way critical in the upkeep of a solid visual surface. Patients with MGD reportedly exhibit a higher rate of ocular surface evaporation than normal subjects13, demonstrating that DED is directly correlated with the integrity and quality of meibum on the ocular surface. Meibomian gland dysfunction results in unbalanced lipid secretion, which increases the rate of ocular surface evaporation and causes tear hyperosmolarity.

RISK FACTORS

There are many DED-related risk factors that also contribute to MGD. As a result, modifying risk factors may improve both disease states. As androgen and oestrogen receptors are both present in the meibomian glands, androgens have been reported to stimulate meibum secretion and suppress inflammation, whereas oestrogens reduce meibum secretion and increase inflammation.15Dysfunctional meibomian gland secretion and concurrent alterations in the lipid layer have been observed in patients with androgen depletion.16Additionally, female sex has been identified as a risk factor for the development of autoimmune diseases that lead to DED, such as Sjögren's syndrome.17

MANAGEMENT

The TFOS DEWS II developed an algorithm to implement various management options based on disease severity.40 At the outset, patients must be educated about environmental and dietary modifications, including essential fatty acid supplements. This is necessary to standardize the disease's management. Additionally, patients must be instructed to eliminate the use of both topical and systemic medications, contact lens wear, and other factors that can trigger the onset of DED. Several adjustments to one's way of life, such as getting enough sleep or rest, drinking enough water, and quitting smoking, may help alleviate symptoms. For mild DED, lubricants for the eyes are recommended; Preferably, these should not be preserved with benzalkonium

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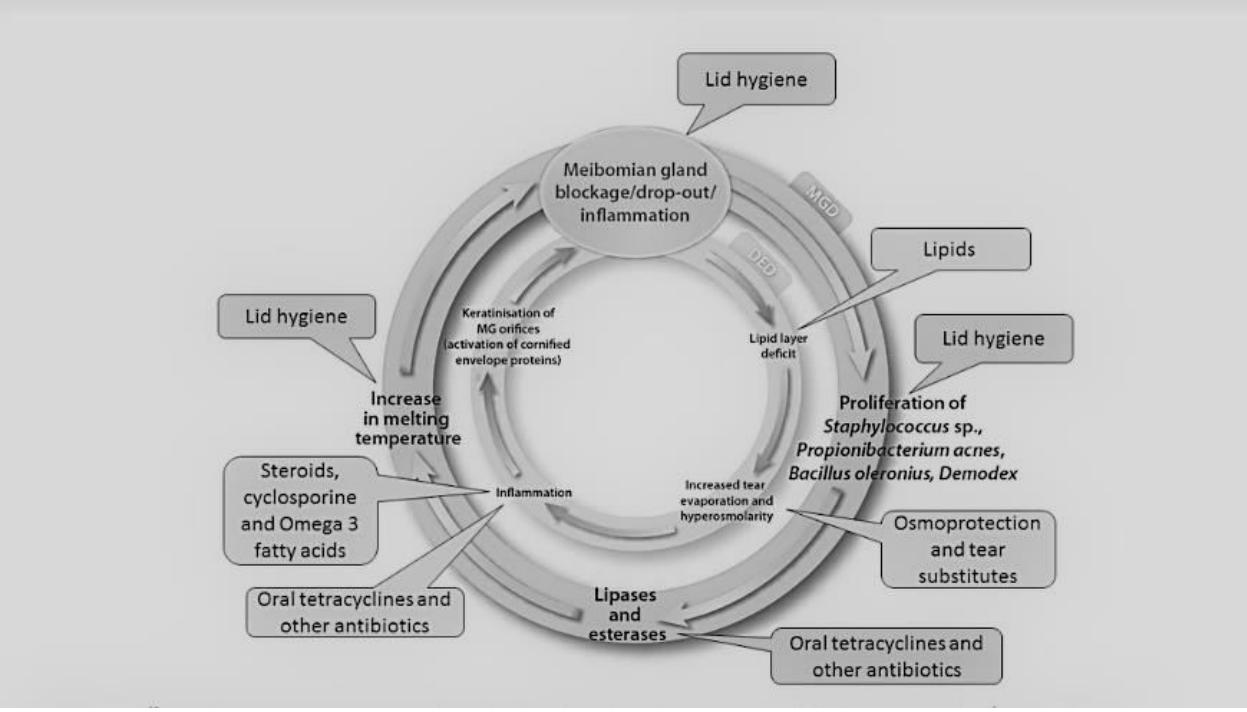
chloride. An overview of the newest treatment options and devices as well as some of these modification strategies are provided below.

1.Eyelid cleanliness

2.Lipid containing Fake tears

3.Antiinflammatory prescriptions

4.Omega 3 dietary supplementation



Above figure delineate show treatments that can possibly break one of the horrendous circles, for example, tear substitutes, may influence on the other by means of circuitous impacts on meibomian organ blockage, dropout and irritation

CONCLUSION

Dry eye sickness is a typical ophthalmic issue, with a reason that is frequently multifactorial. Due to an imbalance in lipid secretion that has an effect on the rate of tear evaporation, meibomian gland dysfunction is a major cause of DED. DED occurs when tears evaporate

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quickly and their osmolarity rises. There are numerous risk factors that can lead to the onset of both DED and MGD, many of which may be shared by both diseases. When dealing with DED, a precise diagnosis is essential. For DED and MGD, there are a variety of treatment options, and a step-by-step, staged approach is frequently necessary to ensure proper management.

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