**LEARNING METHOD AND MEDIUM**

This educational activity consists of a supplement and 30 study questions. The participant should, in order, read the learning objectives contained at the beginning of this supplement, read the supplement, answer all questions in the post test, and complete the evaluation form. To receive credit for this activity, please follow the instructions provided on the post test and evaluation form. This educational activity should take a maximum of 3.0 hours to complete.

**CONTENT SOURCE**

This continuing education (CE) activity is based on a review of the literature and an expert panel discussion.

**TARGET AUDIENCE**

This educational activity intends to educate optometrists.

**OVERVIEW**

Previously, there had not been a consistent and thorough discussion of meibomian gland anatomy and pathophysiology. An international working group of the Tear Film and Ocular Surface Society (TFOS) convened and developed a new definition and new terminology on meibomian gland dysfunction (MGD), clarifying the differences between it and other ocular surface diseases, and introduced a new diagnosis and management algorithm. This CE monograph captures proceedings from a roundtable discussion of a group of optometrists, some of whom were directly involved in the MGD workshop, others who were not, who gathered to evaluate the clinical relevance of the TFOS MGD Workshop’s findings, particularly as they apply to patients seeking treatment for contact lens-related or general ocular surface complaints, as well as to those patients cared for before and after ocular surgery.

**LEARNING OBJECTIVES**

Upon completion of this activity, optometrists will be better able to:

1. Describe new terminology and classifications of blepharitis and meibomian gland dysfunction
2. Distinguish blepharitis/meibomian gland dysfunction from other ocular conditions
3. Describe treatment implications for meibomian gland dysfunction with or without the presence of additional ocular surface disorders

**ACCREDITATION DESIGNATION STATEMENT**

This course is COPE approved for 3.0 hours of CE credit. COPE Course ID: 34224-AS. Check with your local state licensing board to see if this counts toward your CE requirement for relicensure.

**DISCLOSURES**

Gary N. Foulks, MD, FACS, has had a financial agreement or affiliation during the past year with the following commercial interests in the form of Honoraria: InSite Vision Incorporated; and Santen Pharmaceutical Co, Ltd; Consultant/Advisory Board: Bausch + Lomb Incorporated; Inspire Pharmaceuticals, Inc; and Scynexis, Inc; Contracted Research: Alcon, Inc.

Alan G. Kabat, OD, FAAO, has had a financial agreement or affiliation during the past year with the following commercial interests in the form of Consultant/Advisory Board: Alcon, Inc; Exulus of America, Inc; Inspire Pharmaceuticals, Inc; and ISTA Pharmaceuticals, Inc; Contracted Research: TearLab Corporation.

Paul M. Karpecki, OD, FAAO, has had a financial agreement or affiliation during the past year with the following commercial interests in the form of Consultant: Alcon, Inc; Allergan, Inc; Bausch + Lomb Incorporated; Inspire Pharmaceuticals, Inc; and ISTA Pharmaceuticals, Inc; and OcuSOFT, Inc.

Jason J. Nichols, OD, MPH, PhD, FAAO, has had a financial agreement or affiliation during the past year with the following commercial interests in the form of Advisory Board/Reviewer: Bausch + Lomb Incorporated; and Vistakon; Honoraria/Editorship: Wolters Kluwer; Contracted Research: Alcon, Inc; CIBA VISION Corporation; Inspire Pharmaceuticals, Inc; and Vistakon, Inc.

Kelly K. Nichols, OD, MPH, PhD, FAAO, has had a financial agreement or affiliation during the past year with the following commercial interests in the form of Honoraria: Promotional: Alcon, Inc; Allergan, Inc; and Inspire Pharmaceuticals, Inc; Consultant/Advisor: Alcon, Inc; Allergan, Inc; Bausch + Lomb Incorporated; Celsic Pharma; Forest Laboratories, Inc; ISTA Pharmaceuticals, Inc; InSite Vision Incorporated; Merck & Co, Inc; SAICode BioScience, Inc; and TearLab Corporation; Contracted Research: Alcon, Inc; Inspire Pharmaceuticals, Inc; and TearLab Corporation.

**DISCLOSURE ATTESTATION**

Each of the contributing physicians listed above has attested to the following:

1. that the relationships/affiliations noted will not bias or otherwise influence his or her involvement in this activity;
2. that practice recommendations given relevant to the companies with whom he or she has relationships/affiliations will be supported by the best available evidence or, absent evidence, will be consistent with generally accepted medical practice; and
3. that all reasonable clinical alternatives will be discussed when making practice recommendations.

**OFF-LABEL DISCUSSION**

This activity includes off-label discussion of azithromycin for MGD, steroids for MGD and for dry eye.

**PEER REVIEW DISCLOSURES**

Katherine M. Mastrotta, OD, has had a financial agreement or affiliation during the past year with the following commercial interests in the form of Advisory Board: Allergan, Inc; Bausch + Lomb Incorporated; Focus Laboratories; ISTA Pharmaceuticals, Inc; and Merck & Co, Inc; Board Member: Noble Vision Group, LLC; and OcuSOFT, Inc; Ownership Interest: TearLab Corporation.

Leo Semes, OD, has had a financial agreement or affiliation during the past year with the following commercial interests in the form of Consultant: Alcon, Inc; Allergan, Inc; Merck & Co, Inc; Speaker: Optovue, Inc.

**EDITORIAL SUPPORT DISCLOSURES**

Michelle Dalton, ELS; Cynthia Tornayy, RD, MBA, CCMEP; and Barbara Lyon have no relevant commercial relationships to disclose.

**GRANTOR STATEMENT**

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Meibomian Gland Dysfunction: No Longer in the Shadows

In the mid 1990s, research in ocular surface disease was in its infancy, with disjointed descriptions of patient-reported symptoms failing to correlate well with clinically observed signs. Terminology was intermingled and not qualified, or sufficiently specific. Around the world, clinical studies devised their own sets of inclusion criteria, limiting the ability to generate consensus. The pathogenic relationship of dry eye alone or in conjunction with contact lens (CL) wear was uncertain. In an important update to a pivotal report on dry eye published in 1995, the 2007 Dry Eye Workshop (DEWS) published its findings, providing a consensus overview of dry eye disease as it currently stood. A revised algorithm for diagnosis and management of dry eye disease was included in the report, and became the severity grading scale standard for describing the disease.

Following the publication of the DEWS report, the Tear Film and Ocular Surface Society (TFOS) undertook a similar task, this time on the equally ambiguous topic of meibomian gland dysfunction (MGD). While MGD was mentioned in the DEWS report, the description of MGD and its association with dry eye disease was limited. This fact was apparent in the disease severity grading diagram, in which the discussion of meibomian gland involvement was “variable” across most disease severity grading levels. Before the TFOS evaluation of MGD, not many researchers were actively involved in studying the disorder, although the condition itself had been described one way or another in the literature consistently since the 1980s. At that time, clinicians likely did not include MGD in findings of dry eye, except in severe cases of the disease. MGD was most often referred to in overview reports on dry eye or in treatment regimens and clinical trials evaluating blepharitis. Although ophthalmic researchers have since come to respect the vital role MGD plays in evaporative dry eye, the association between MGD and aqueous-deficient dry eye and the parallel relationship between MGD and age, conveying findings to clinicians worldwide had not been a priority. Even among those concerned with the condition, broad and inconsistent terminology has been banded about, all presuming to describe the same observations. The term “meibomian gland dysfunction” was first coined in the 1980s; additional terminology, including posterior blepharitis, meibomitis, meibomian gland disease, and meibomian keratoconjunctivitis, among others, has been used to describe the condition.

After 2 years and input from more than 50 experts around the world, the TFOS/International Workshop on Meibomian Gland Dysfunction defined the relationship between MGD and dry eye, clarified the differences between MGD and other ocular surface diseases, proposed a new consensus definition for MGD, and introduced a new diagnosis and management algorithm. It was the hope of the steering committee to advance the study of MGD in one large uniform step. To our knowledge, the MGD Workshop report represents the first time a global consensus has been reached on the definition of MGD, and as such, allows a general consensus on how to diagnose and treat patients with MGD.

Previously, there had not been a consistent and thorough discussion of meibomian gland anatomy and pathophysiology. In an effort to now correct this lack of information, the committee members proposed that meibomian gland duct obstruction be considered a leading cause of MGD that may contribute to the bulk of dry eye disease. Given that, should clinicians and researchers consider MGD a separate entity with its own set of diagnostics and treatments, or should it remain sufficient to evaluate MGD only within the scope of a dry eye complaint? Researchers are still struggling to determine the natural history of MGD. Without this knowledge, a cumbersome challenge remains for quantifying the clinical findings as a result of disease progression, or merely a function of aging.

So it is incumbent upon the optometric community not only to evaluate the definition of MGD and implications of the condition, but also to be vigilant in promoting use of these findings outside the hallowed halls of the research and clinical laboratories. That is the main goal of this continuing education module. Its contents will serve as a means to help optometrists better recognize and diagnose those with MGD, distinguishing them from patients with other ocular surface disorders, thus providing insight into the management of this particular dysfunction.

To that end, a group of optometrists, some of whom were directly involved in the TFOS/International Workshop on MGD, assembled to share their thoughts regarding MGD. Together, through their collective comments, this module will help optometrists evaluate the clinical relevance of the MGD Workshop’s findings, particularly as they apply to patients seeking treatment for CL-related or general ocular surface conditions, as well as to those requiring preoperative and postoperative ocular surgery care. The faculty of this module encourages eye care professionals and others interested in the topic to review the full MGD Workshop report for additional details not included here.

Kelly K. Nichols, OD, MPH, PhD
Gary N. Foulks, MD
The Importance of the Meibomian Gland

It is well documented that a primary reason patients present at an eye care specialist’s office is for complaints of dry eyes, “grittiness,” eyelid issues, and other symptoms with which practitioners are fairly well versed.1-5 People who wear CLs may be even more sensitive to subtle surface changes and may seek out treatment earlier in life than people who are not CL wearers. Ocular dryness is the leading complaint and a common reason people become intolerant to CLs.6-10

Researchers and clinicians are just beginning to understand the vital role the meibomian gland plays in a variety of ocular surface disorders. For example, MGD may complicate and adversely affect CL wear and lens deposition.3,11-13 The meibomian glands themselves are more numerous on the upper lid than on the lower lid and do not have direct contact with hair follicles, although they are sebaceous in origin.14 Further, recent studies suggest CL wear may cause atrophic changes to the meibomian gland.11,12 It remains unclear whether changes in meibomian gland structure result in alterations to lipid secretions, and whether those changes can be reversed or halted.

MGD in general is classified by its severity, its pathophysiologic or its anatomic changes.15 It remains unclear how the integrity of the meibomian glands affects the tear film surface and CL wear. We do know, however, that the gland’s function is often affected by hormones (androgen, estrogen, progestin, growth factors) and by all-trans retinoic acid.15 Androgen deficiency, in particular, has been cited as directly contributing to MGD (as well as to evaporative dry eye).16-20 As people age, the amount of meibomian cells expressing estrogen receptors in the lower lid increases, but this has not been shown to directly affect tear film stability.21 There is still much more we need to understand about meibomian gland regulation with age.

MGD prevalence has not been definitively determined either; it runs the gamut, depending on which criteria are used.22-24 In population-based studies, whites are at the lower prevalence range, with no study reporting more than 20%, although the studies were not designed to classify MGD according to race.20,21 Conversely, most studies evaluating MGD prevalence in Asians report upwards of 60%.24,25,26-27 One additional study noted a prevalence of 46.2% in Thailand,28 but because of inappropriate sampling methods, the study findings may require further validation. Only 2 studies24,26 evaluated the prevalence of MGD in CL wearers and non-CL wearers.

In most of these studies, subjects with clinical signs of MGD had overlapping symptoms of dry eye. Ophthalmic, systemic, and medication-related factors coexist and/or are codependent between MGD and dry eye. Undermining researchers’ and clinicians’ ability to easily quantify MGD from either form of dry eye is a lack of disease progression timelines, a definitive order on symptom development, and the processes that cause development of symptoms.

Because of its relationship to the tear film, MGD may very well be the most common cause of evaporative dry eye; it has also been linked to aqueous-deficient dry eye. The symbiotic relationship between the meibomian glands and the tear film indicates the former is able to alter the latter.

Optometrists would best serve their patients by consistently including gland expression tests in their standard dry eye evaluations to help differentiate MGD from other ocular surface and CL-related disorders. Improving patient symptoms—and therefore patient quality of life—should remain a primary treatment goal for optometrists.

Blepharitis and MGD: the need for differentiation

Discussion of major diseases of the ocular surface disease family—dry eye, blepharitis, MGD—has been hampered by the absence of a simple, widely accepted definition and by terminology that is ambiguous and inconsistently used. Some reports dating back to the 19th century describe diseases such as meibomian seborrhea or conjunctivitis meibomiana; we currently describe these as either MGD or blepharitis, respectively.4,6 McCulley first differentiated the relationship between meibomitis and gland dysfunction; Korb and Henriquez first introduced the term “meibomian gland dysfunction” in 1980.5

In the decades since, numerous criteria have been applied to describe MGD.6 The International Workshop on MGD noted that the dysfunction differs from the disease in that the latter is a broad category that encompasses dysfunction, neoplasia, and congenital diseases.6 Terms used in the last century, including meibomitis and meibomianitis, are more specific to a subset of disorders with inflammation at the core. In MGD, inflammation is not a necessary component.7

More recently, as issues affecting the ocular surface both in preoperative and postoperative states have been brought to the forefront with the increase in elective refractive corneal-based surgery, numerous articles have been published on ocular surface disorders. The most common overlap in terminology recently has been that between MGD and posterior blepharitis.8,11,30-34 It is critical that optometrists and other eye care professionals begin to differentiate between these 2 terms. Posterior blepharitis involves inflammation of the posterior lid margin; MGD is but 1 potential cause.33 As such, the 2 terms should not be used interchangeably.30 It should be noted that anterior blepharitis can occur simultaneously with MGD and may require concurrent management.

The most common category of MGD is obstructive MGD, cicatricial or noncicatricial.7 Additional categories include hyper-secretory, characterized by the release of a large volume of meibomian lipid at the lid margin, and hypo-secretory, characterized by decreased meibomian lipid secretion without gland obstruction. As previously stated, it is generally accepted that MGD is the leading cause of evaporative dry eye and plays a role in aqueous-deficient dry eye. Because MGD and dry eye are so intertwined, clinicians may have difficulty differentiating between the two.

If left untreated, MGD can lead to complications of the ocular surface, including tear film alterations and dry eye. There remains a lack of uniform agreement on the relationship between MGD and CL wear, with some studies showing that discomfort and dryness during CL wear can be attributed to MGD, but there is not enough evidence to suggest a direct correlation between CL wear and MGD.6

In short, alterations to the meibomian gland can lead to reduced delivery of meibum to the lid margin and tear film, can result in ocular surface irritation and transient visual blur and dry eye, and can negatively affect quality of life. MGD remains an underdiagnosed, underestimated, and undertreated disease.
The New Definition of MGD
The MGD Workshop group suggested the following new definition of MGD:

*Meibomian gland dysfunction (MGD)* is a chronic, diffuse abnormality of the meibomian glands, commonly characterized by terminal duct obstruction and/or qualitative/quantitative changes in the glandular secretion. This may result in alteration of the tear film, symptoms of eye irritation, clinically apparent inflammation, and ocular surface disease.31

The proposed new definition of MGD has provided clinicians with a definitive anatomic location for the abnormality, separate from other diseases of the lid margins. For optometrists, the nomenclature is being slowly integrated into daily clinical use. Research has already begun incorporating the definition into ongoing clinical studies (the results of which have not yet been completed). Following are relevant thoughts from the roundtable discussion.

**Dr Kelly Nichols:** What are your thoughts on the MGD Workshop’s new definition of MGD?

**Dr Paul Karpecki:** It’s been hugely advantageous. Before the report, terms had been thrown around arbitrarily and there was no consistency among them. Clinicians were using meibomitis, blepharitis, posterior blepharitis, meibomian gland disease dysfunction, and so forth. What this report solidified for me was finally getting everybody to look at the different terms and realize there are different meanings for each, and to create consistency.

It’s made it easier for me to manage patients, with meibomitis designating more the inflammatory level and MGD designating a larger overarching term.

**Dr Jason Nichols:** The waters were fairly muddy prior to the report in terms of terminology that might not have been correct. In the MGD Workshop, we were careful to ensure that the final report clearly defines anterior blepharitis, posterior blepharitis, and MGD. In turn, educators can use the proper terminology as they’re disseminating the report and teaching its findings.

**Definition differentiation of common terms**31

**Meibomitis/meibomianitis:** Subset of disorders with inflammation of the gland orifices as the key component

**Blepharitis:** Inflammation of anterior or posterior lid margin; can occur concurrent with MGD

**Posterior blepharitis:** Involves inflammation of the posterior lid margin; MGD may be 1 of several causes

**Meibomian gland dysfunction:** Inflammation is not mandatory; does not include neoplasia or congenital disease

Pathophysiology of MGD
Located in the tarsal plates of the eyelids, the meibomian glands are large sebaceous glands that act to synthesize and secrete lipids and proteins. These lipids are a crucial aspect in promoting tear film stability and in preventing tear film evaporation.

Numerous factors may potentially contribute to the pathophysiology of MGD—anterior blepharitis, CL wear, *Demodex folliculorum*, dry eye.5 Systemic diseases, such as rosacea, psoriasis, hypertension, and benign prostatic hyperplasia, could also be contributory factors, as are autoimmune diseases such as Sjögren syndrome.7 Hormone medications (antiandrogens, postmenopausal hormone therapy) also have been associated with MGD.15 According to the MGD Workshop report, “almost all of our understanding of the physiological, as well as pathophysiological, regulation of the meibomian gland originates from research exploring the effects of androgens, estrogens, prostaglandins, *trans* retinoic acid, and growth factors on this tissue and/or its epithelial cells.”15

Transient symptoms of dry eye became clinically relevant in patients who became CL-intolerant; Korb and Henriquez first described obstructive MGD as a result of hyperkeratinization in these same patients.3,37 CL intolerance dissipated after plug removal and gland expression. In those with symptomatic dry eye, excretory duct obstruction by increased keratinization signs were verified on histologic examination.35

Meibomian gland dysfunction, at its simplest, occurs when there is an obstruction sufficient enough to prevent the meibum from performing its job. Indeed, obstructive MGD appears to be the most common form of the condition.15 In its healthy form, meibum gland secretion is clear to light yellow; when in an unhealthy state, meibomian gland secretions may be cloudy, turbid, inspissated, or toothpaste-like in consistency.15

Here, roundtable panelists discuss MGD, its pathophysiology and the crucial potential overlap with CL intolerance.
**Dr Alan Kabat:** Like most ocular surface disorders, MGD is multifactorial in nature. I don’t think clinicians can determine a specific causative element for each patient with MGD. It’s more crucial to be able to recognize the features that can contribute to or exacerbate the symptomatology of MGD. For example, patients with dermatologic conditions like rosacea, seborrhea, or psoriasis should be scrutinized carefully. Likewise, CL wearers and people undergoing any form of incisional ocular surgery warrant more thorough investigation, because success or failure of the surgical procedure may hinge on appropriate management of MGD. Moreover, clinicians must be able to observe those findings that demonstrate staging of disease severity, so that appropriate measures can be initiated. The extent of meibomian gland obstruction and the quality of meibum in terms of expressibility and turbidity are the most useful diagnostic elements, in my opinion.

**Dr Jason Nichols:** Understanding MGD in the realm of CL use was an important piece of the MGD Workshop report. Practitioners generally report anecdotally that in clinical settings, approximately 60% of CL dry eye is more evaporative dry eye and 40% is more aqueous-deficient.28 38

**Dr Paul Karpcki:** It’s not unusual for optometrists to mistake MGD-related tear film issues for refractive errors or CL-related discomfort. So what presents as CL intolerance may have MGD as its cause. What we need to determine in these patients is the cause for the CL intolerance beyond first pointing at the CL as the cause. Regarding MGD itself, numerous questions still remain. Is it hormonal based? Is it androgen deficiency, menopause? Is it related to other factors like the environment? Is it diet based? Systemic disease related? There is not a lot of conclusive evidence that we truly understand the pathophysiology of MGD. Why does the Asian population have so much MGD? I do not think their diet is that different from or worse than the American diet. These observations raise a lot of good questions for future research while also providing an encompassing view of the current knowledge base of MGD.

**Dr Jason Nichols:** Without question, the primary reason people discontinue CL wear is that they experience dry eye symptoms—dryness, discomfort, etc—which usually happen in the age range of the mid 30s to early 40s. At the same time, I think many people in this age group are successfully treated with refractive surgery, but will likely have dry eye postoperatively. The 2 things are correlated: the discomfort/dryness and the CLs. Sometimes refractive surgery is a patient’s choice, when the CL intolerance might have been able to be managed by treating the underlying cause of the CL dry eye, the MGD.

**New Directions for Diagnosing MGD**

Historically speaking, the majority of MGD diagnosis was undertaken as part of clinical or basic research studies, yet the condition remained underdiagnosed or potentially overlooked altogether in clinical practice. Creating an algorithm that would be applicable in both clinical and research settings has been challenging. From a clinical perspective, MGD diagnosis should be done within the confines of diagnosing any ocular surface disorder, with attention to potential causal relationships to both aqueous-deficient and evaporative dry eye.15 This can be accomplished by performing 2 sets of tests. In the first, distinguish those patients with dry eye of any type from those without dry eye. In the second, differentiate between MGD-related evaporative dry eye and aqueous-deficient dry eye. In general, it has been recommended that meibomian gland expression be added to the cadre of testing in asymptomatic adults to detect asymptomatic or mildly symptomatic, nonobvious MGD.39 The International Workshop on MGD group proposed a set sequence for testing to quantify those with MGD-related diseases, beginning with a questionnaire and then adding physical testing of the surface and eyelids that gradually becomes more invasive. An evidence-based approach for the management of MGD has been proposed (see Table 1). At each level, a lack of response dictates a move to the subsequent level.

**Dr Kelly Nichols:** One of the diagnostic tools the MGD Workshop report highlighted was the routine expression of the meibomian glands. Since the report has been published, I’ve heard more clinicians are incorporating expression into their ocular surface examinations earlier in the assessment process than they had in years past. What are your thoughts?

**Dr Alan Kabat:** I always quote the statistic that at least 75% of the patients we see who have dry eye as a diagnosis have some degree of MGD either as the primary source or as secondary involvement. The MGD Workshop report gives the clinician who would like to begin expressing meibomian glands a pattern to follow and some guidelines. For others who have been expressing more routinely, the report can help them step up and be able to treat MGD a little bit more consistently and, I hope, get better results.

**Dr Kelly Nichols:** In the more severe cases of ocular surface disease, whether it is aqueous-deficient or not, there is generally a meibomian gland component. Regarding those practitioners who fit lenses, they should be evaluating the lids at the same time. I think specialty lens fitters are using the diagnostic tools, but the emphasis here is that at every stage of referral or management, we should be looking at the lid margins.

**Dr Jason Nichols:** Relative to the CL patient, clinicians look at symptoms first. With CLs, there is a phenomenon wherein the patient has a very large diurnal variation in symptoms. The CL patient has a significant increase in the severity of symptoms from midnight into the evening hours. Comparatively, the non-CL MGD patient tends to have worse symptoms in the morning. In this group, you need to examine the lid margins, express, look for plugged glands. There have been studies showing that CL wear can lead to some atrophy of the meibomian glands.60 40 In the CL wearer, symptoms could be indicative of primary MGD, or the MGD could be induced by CL wear—this has yet to be determined.

**Dr Gary Foulks:** Let’s further discuss eyelid expression. Has the MGD Workshop report changed your approach to the way you express, or whom or how you express?

**Dr Alan Kabat:** I express the lower lid. Some like to use the Mastrota paddle, or cotton tip applicators, but I use my thumb. I find that by using a thumb technique consistently, I typically can find what I need. I differentiate between diagnostic meibomian gland expression and therapeutic gland expression. Diagnostically, I’m trying to simulate minimal pressure on the lids to get an idea of the quality of the meibum (turbid, thick, etc) as well as its ease of delivery from the glands in the patient’s natural state. Korb and Blackie discuss this,41 and described the device they invented to simulate the pressure of a ‘normal blink’.
In more severe cases in which we see meibomian capping and/or in which the patient is highly symptomatic, I may be inclined to perform a therapeutic expression. In this, I am using much greater pressure than would be involved in the natural blinking process. Here, my goal is to clear the meibomian ductules of stagnant meibum, so that we’re starting with a ‘clean slate’, of sorts. I believe this is the same goal as is involved with the LipiFlow® device or the Maskin probing technique, but my methods are far more rudimentary.

Dr Gary Foulks: Clinicians who aren’t currently expressing need to know how easy it is to do. (See below for Dr Karpecki’s technique on how to express.)

Dr Alan Kabat: As a result of the MGD Workshop report, for any patient who comes in with that itchy, burny, gritty type of history as a chief or peripheral complaint, the general dry eye workup with vital dye staining, evaluation of tear break-up time (TBUT), etc, now also includes gland expression.

Expressing Lids: One Clinician’s Technique
Paul M. Karpecki, OD

Some clinicians are not yet comfortable with how or when to express lids. Expressing just the lower lid will yield valuable information about glandular health. I’ve found oftentimes expressing the lower lid—in the central to nasal aspects—is more than sufficient to detail what’s occurring on the upper lid as well. If you’ve never expressed a lid before, begin with patients who are symptomatic or in whom you notice some signs as you are looking at the eyelid with slit-lamp examination.

Briefly, you’ll need to gauge what gets expressed. Is the substance clear and oil-like? That would indicate a very healthy gland. Or is it turbid, paste-like; or is the gland nonexpressive? (See Figures 1-3).

It can be helpful to apply topical anesthesia before expressing. First I pull down the lower eyelid. Then I place either a wetted cotton tip applicator or Mastrota paddle behind the lower eyelid. A wet cotton tip swab can help avoid dessicating the conjunctiva. Then, with my index finger, I press toward that cotton tip applicator or paddle. In my experience, some patients prefer that technique to pressing directly on their eye. (Note: if you are performing expression behind the slit-lamp, the technique is easier without the aid of the applicator or paddle behind the lid.) Hold enough pressure to ensure the patient notices the pressure but is not in extreme pain, as this is just for diagnostic purposes—if you’re pinching, it’s too much pressure.

Typically, the pressing position should be held for about 15 seconds—by 10 seconds, you should start to see something coming out of the gland. I prefer to begin in the central lower eyelid and then move between the nasal and central areas. I usually select 3 areas to express.

There are probably about 10 to 15 glands from the central aspect to the medial. I gauge how many of those expressed and what they expressed. Typically I will see 2 to 4 that express, and then I check for the quality of the expressed material. If you get no production, however, and see a lot of blood vessels or telangiectasia, a thickened eyelid margin or posterior facing meibomian glands, that’s clearly indicative of advanced stages of MGD.

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It can be helpful to apply topical anesthesia before expressing. First I pull down the lower eyelid. Then I place either a wetted cotton tip applicator or Mastrota paddle behind the lower eyelid. A wet cotton tip swab can help avoid dessicating the conjunctiva. Then, with my index finger, I press toward that cotton tip applicator or paddle. In my experience, some patients prefer that technique to pressing directly on their eye. (Note: if you are performing expression behind the slit-lamp, the technique is easier without the aid of the applicator or paddle behind the lid.) Hold enough pressure to ensure the patient notices the pressure but is not in extreme pain, as this is just for diagnostic purposes—if you’re pinching, it’s too much pressure.

Typically, the pressing position should be held for about 15 seconds—by 10 seconds, you should start to see something coming out of the gland. I prefer to begin in the central lower eyelid and then move between the nasal and central areas. I usually select 3 areas to express.

There are probably about 10 to 15 glands from the central aspect to the medial. I gauge how many of those expressed and what they expressed. Typically I will see 2 to 4 that express, and then I check for the quality of the expressed material. If you get no production, however, and see a lot of blood vessels or telangiectasia, a thickened eyelid margin or posterior facing meibomian glands, that’s clearly indicative of advanced stages of MGD.

Expressing Lids: One Clinician’s Technique
Paul M. Karpecki, OD

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Implications for Treatment
The treatment of MGD has previously lacked a consensus, with varied treatments and therapies across continents. Because MGD has been acknowledged as both underdiagnosed and under-reported, the varied treatment practices add to the inability to accurately assess practice patterns. Similarly, without a consensus on severity levels for MGD, developing treatment plans can be problematic. The International Workshop on Meibomian Gland Dysfunction incorporated a treatment algorithm in its findings (see Table 1) to help address the problem.

Table 1. Treatment Algorithm for MGD Reprinted with permission from Investigative Ophthalmology & Visual Science.

<table>
<thead>
<tr>
<th>Stage</th>
<th>Clinical Description</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>No symptoms of ocular discomfort, itching, or photophobia</td>
<td>Inform patient about MGD, the potential impact of diet, and the effect of work/home environments on tear evaporation, and the possible drying effect of certain systemic medications</td>
</tr>
<tr>
<td></td>
<td>Clinical signs of MGD based on gland expression</td>
<td>Consider eyelid hygiene including warming/expression as described below (±)</td>
</tr>
<tr>
<td></td>
<td>Minimally altered secretions: grade ≥2–4</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Expressibility: 1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>No ocular surface staining</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Minimal to mild symptoms of ocular discomfort, itching, or photophobia</td>
<td>Advise patient on improving ambient humidity; optimizing workstations and increasing dietary omega-3 fatty acid intake (±)</td>
</tr>
<tr>
<td></td>
<td>Minimal to mild MGD clinical signs</td>
<td>Institute eyelid hygiene with eyelid warming (a minimum of four minutes, once or twice daily) followed by moderate to firm massage and expression of MG secretions (+)</td>
</tr>
<tr>
<td></td>
<td>Scattered lid margin features</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mildly altered secretions: grade ≥4–&lt;8</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Expressibility: 1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>None to limited ocular surface staining; DEWS grade 0–7; Oxford grade 0–3</td>
<td>All the above, plus (±)</td>
</tr>
<tr>
<td></td>
<td>Artificial lubricants (for frequent use, nonpreserved preferred)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Topical azithromycin</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Topical emollient lubricant or liposomal spray</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Consider oral tetracycline derivatives</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Moderate symptoms of ocular discomfort, itching, or photophobia with limitations of activities</td>
<td>All the above, plus</td>
</tr>
<tr>
<td></td>
<td>Moderate MGD clinical signs</td>
<td>Oral tetracycline derivatives (+)</td>
</tr>
<tr>
<td></td>
<td>↑ lid margin features: plugging, vascularity</td>
<td>Lubricant ointment at bedtime (±)</td>
</tr>
<tr>
<td></td>
<td>Moderately altered secretions: grade ≥8 to &lt;13</td>
<td>Anti-inflammatory therapy for dry eye as indicated (±)</td>
</tr>
<tr>
<td></td>
<td>Expressibility: 2</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mild to moderate conjunctival and peripheral corneal staining, often inferior: DEWS grade 8–23; Oxford grade 4–10</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Marked symptoms of ocular discomfort, itching or photophobia with definite limitation of activities</td>
<td>All the above, plus</td>
</tr>
<tr>
<td></td>
<td>Severe MGD clinical signs</td>
<td>Anti-inflammatory therapy for dry eye (+)</td>
</tr>
<tr>
<td></td>
<td>↑ lid margin features: dropout, displacement</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Severely altered secretions: grade ≥13</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Expressibility: 3</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Increased conjunctival and corneal staining, including central staining: DEWS grade 24–33; Oxford grade 11–15</td>
<td></td>
</tr>
<tr>
<td></td>
<td>↑ signs of inflammation: moderate conjunctival hyperemia, phlyctenules</td>
<td></td>
</tr>
</tbody>
</table>

“Plus” disease Specific conditions occurring at any stage and requiring treatment. May be causal of, or secondary to, MGD or may occur incidentally:

1. Exacerbated inflammatory ocular surface disease
2. Mucosal keratinization
3. Phlyctenular keratitis
4. Trichiasis (e.g. in cicatricial conjunctivitis, ocular cicatricial pemphigoid)
5. Chalazion
6. Anterior blepharitis
7. Demodex-related anterior blepharitis, with cylindrical dandruff

At each treatment level, lack of response to therapy moves treatment to the next level. A ± sign means that the evidence to support the use of the treatment at that level is limited or emerging; thus its use should be based on clinical judgment. A + sign indicates that the treatment is supported by the evidence at that stage of disease.
Following are the roundtable participants’ comments regarding the MGD Workshop recommended algorithm and its role in their clinical practices.

**Dr Kelly Nichols:** The treatments in the algorithm are paired with the different stages. Does that resonate with what you had done previously?

**Dr Jason Nichols:** For stage 1, I would add a “1-plus” with a focus on the CL, obviously. If it is a simple issue like a poorly fitting lens, or a patient not caring for the lenses properly with a rub and rinse, not cleaning the lipids from the surface, things of that nature, that would be an easy remedy to recommend before moving on to a more complicated treatment like a pharmaceutical. Hygiene and compliance in the lens wearer should be assessed early on.

**Dr Alan Kabat:** I now tend to look more critically at the staging patterns, particularly in terms of advanced or severe changes. The classifications I used prior to the MGD Workshop report were certainly less specific and probably broader overall with regard to what might constitute a “stage 1” or a “stage 2” presentation. Regrettably, quite a bit of MGD can be overlooked because its presentation is so subtle. With the consensus view and a finite definition of what constitutes each stage of the disease, it’s easier to communicate with our eye care colleagues. Likewise, it gives us a straightforward and reliable algorithm for initiating therapy at each level.

**Dr Jason Nichols:** For stage 1, I would add a “1-plus” with a focus on the CL, obviously. If it is a simple issue like a poorly fitting lens, or a patient not caring for the lenses properly with a rub and rinse, not cleaning the lipids from the surface, things of that nature, that would be an easy remedy to recommend before moving on to a more complicated treatment like a pharmaceutical. Hygiene and compliance in the lens wearer should be assessed early on.

**Dr Paul Karpecki:** Before reading the MGD Workshop report, I had not used treatments like the liposomal (lipid) spray extensively. I had used topical azithromycin and combination agents quite a bit before. The report helped me refine what points in the earlier stages of MGD I positioned treatments. I’ll still recommend warm compresses in the milder stages, but now I add liposomal spray at the next stage, in addition to tear supplements.

So now I’m going to put the patient on topical azithromycin, a combination agent such as loteprednol/tobramycin or tobramycin/dexamethasone at almost every level, and doxycycline the moment I start to see telangiectasia or MGD in the presence of rosacea, for example. Before prescribing any medications with steroids in them, baseline IOP should be measured. IOP should be monitored and measured within 3 to 4 weeks of use to see if a patient is a steroid responder; patients should be educated to call the office if they experience pain, redness, or a decrease in vision. As much of a believer in steroids as I am, and as often as we use them in clinical practice—as well as new safer advances such as ester-based steroids—I still write “no refills” when prescribing a steroid so I can be sure that the follow-up appointments are met and IOP is monitored.

Comorbidity with anterior blepharitis often exists in presence with MGD. A spray can break up a lot of the debris on the lashes, letting the liposomes penetrate into the ocular surface. In mild cases with turbid expression, I will use lipid-containing tears and hot compresses to achieve a palliative response. Once there’s more disease or inflammation, I very quickly add in therapeutics, including oral tetracyclines, and may use a tear supplement that works better on hyperosmolarity.

**Dr Kelly Nichols:** There was some controversy as to where to place mention of lipid sprays and lipid-containing tears in the algorithm, as much of the evidence for these products was emerging or pending publication. Ultimately, the products were recommended for stage 2 MGD, often considered mild to moderate symptomatic cases. Since publication of the MGD Workshop report, many practitioners have started using lipid-containing products.

**Dr Jason Nichols:** CL wearers tend to be more symptomatic in the evenings. Practitioners used to change the material or lens fit first, then add a rewetting drop or tear supplement, then change the lens care solution. If none of those regimens worked, then they’d consider prescribing oral or topical treatments. I think the MGD Workshop report treatment recommendations may change how practitioners treat MGD in the CL wearer, with the MGD treatments being recommended and prescribed earlier.

**Dr Kelly Nichols:** What about omega-3s? The MGD Workshop report recommends dietary modification and/or supplementation to include omega-3 in early stages of MGD. The general consensus was that while evidence is limited or emerging, omega-3s are for the most part considered safe and may be effective.

**Dr Gary Foulks:** Although there is not randomized clinical trial evidence for dosing of omega-3 in ocular disorders, it’s probably best not to exceed the American Heart Association guidelines of 3 grams daily, particularly if the patient is on anticoagulants.

**Dr Paul Karpecki:** Omega-3 supplementation depends on a person’s current health, the quality of his or her current diet, and the state of ocular disease present. Someone who has a poor diet, who never eats any vegetables, fruits, or fish, for example, eats meats, and thus has a very high level of unhealthy omega-6s in the regular diet, and who has stage 3 MGD is probably going to require at least 2000 milligrams of an omega-3 supplement or nutritional supplements that combine omega-3 with healthy omega-6 and other nutrients. Someone who eats fish several times a week, is very good about eating vegetables and fruits, and who has stage 2 MGD could be well served with lower amounts of essential fatty acid supplements.

**Dr Gary Foulks:** The typical US diet includes ample, if not excessive, amounts of omega-6 fatty acids. The addition of omega-3 supplements is intended to restore the balance between omega-3 and omega-6, the ratio of which is an important influence on inflammation. If omega-6 is supplied in a supplement, then omega-3 should be included in that same supplement.

**Dr Alan Kabat:** I had not been a strong proponent of the inclusion of fatty acids in the diet, and have used them for patients who were not really amenable to tetracyclines. With the MGD Workshop consensus report recommending these nutrients for patients as early as stage 1 MGD, I will reconsider.

**Final Impressions**

Clinicians and researchers alike agree that the emphasis on lipid expression will contribute to diagnosis of patients with milder levels of MGD. The MGD Workshop clarified the differences between MGD and other lid margin diseases (such as blepharitis); using consistent terminology in clinical trials and in comanaged patients needs to be more rapidly integrated into daily occurrence. The emphasis by the MGD Workshop to stratify treatment options by a stepwise approach should allow clinicians to more effectively treat this common disorder. Adding assessment of the eyelids and meibomian glands can fine-tune a dry eye workup, and will benefit the patient and the optometrist’s practice alike.
There are approximately 35 million CL wearers in the United States, with about half of those suspected of having dry eye symptoms. Perhaps 20% (7 or 8 million people) have severe symptoms or are at risk for CL discontinuation. The numbers are similar to the overall prevalence of dry eye in the general population. MGD seems to be more prevalent in the long-term contact lens wearer, but MGD may be easier to manage in the CL patient than in someone with a systemic disorder such as rosacea.

Dr Paul Karpecki: MGD in a long-term CL wearer tends to be present, but I have not noticed it to be as severe. I still use therapeutics, and I’m still aggressive in treatment, but I might not be as likely to use an oral tetracycline in my CL MGD patients unless the condition displays the same severity as that seen in other non-CL-wearing cases.

Dr Jason Nichols: Clinicians have to look at the CL aspects of these patients’ care as well—the lens itself on the eye—in addition to what the patient does with that lens, how he or she cares for the lens. In the CL wearer, osmolarity was shown to be associated with dry eye. In theory, the lipids in the tears or meibum can bind to the CLs, and that effectively removes them from their normal functions in the outer lipid layer. There’s a faster rate of evaporation of the tears in a CL wearer than in a non-CL wearer. That higher evaporation leads to a higher osmolarity.

The MGD Workshop report has really helped raise awareness about the potential for problems with CLs, dry eye, and MGD, and it may help move us toward a medical approach to CL dry eye, but we still need to rule out an ill-fitting lens or damaged lens. Clinicians need to then look at the ocular surface, specifically the cornea/conjunctiva. The palpebral conjunctiva can show signs of inflammation, redness, or hyperemia that might be indicative of the lens edges rubbing too much. That, in turn, might ultimately lead to atrophy of the glands or plugged glands.

The role of CL solutions in dryness is not well understood. Generally speaking, for a CL wearer, rewetting drops are used often, but we know their efficacy is not maintained over the long term. Specifically regarding topical drops, patients should dose 10 to 15 minutes before inserting the lens. There is some thought that an emollient lubricant or lipid emollient could be better for a CL wearer if more of the lens-related dry eye is related to MGD.

For the CL-intolerant MGD patient, clinicians could consider topical azithromycin or a soft steroid for a short period of time, perhaps to quiet the inflammation while the patient is not wearing the lenses; after stopping the treatment, then start the refitting process. There has been some evidence of a benefit from either azithromycin or cyclosporine in CL wearers with dry eye (although those studies did not specify the presence of MGD).
CASE ILLUSTRATIONS OF MGD DIAGNOSIS AND TREATMENT

Case 1

A 50-year-old white male with history of intermittent irritative symptoms and red eyes presents with swelling of the eyelids in the morning. He is taking no systemic medications but has used topical vasoconstrictors without relief. On physical examination there is facial erythema and swelling of the eyelids OU without conjunctival discharge. The eyelashes are normal, but there is inspissation (plugging) of some of the meibomian gland orifices. Conjunctival vascular engorgement is present with a tear meniscus of 0.7 mm. Rapid tear break-up time (TBUT) is present in both eyes. Faint micropunctate fluorescein staining of the inferior cornea is present OU with fine superficial vessels crossing the limbus. [Tear film osmolarity is 308 mOsm/Kg OU.]

Dr Alan Kabat: Initially, this sounds like a rosacea patient. A dermatology consult is always a consideration for such a patient. This could be stage 3 MGD because there is conjunctival and corneal involvement with regard to staining. The patient does have the plugging, the vascularity, and the thickening in the lid margin.

In terms of treatment, I would start with the recommendations for MGD stages 2 and 3, and, of course, talking about education, lid hygiene, with a strong emphasis on more compresses and massage for this patient.

I would consider topical azithromycin treatment early, along with support of the tear film using appropriate lubrication. In patients with MGD, we understand that the oil component of the tears is deficient, so it makes sense to use a lipid-restorative agent. For these individuals, I currently use Systane® Balance; my dosing is 4 times daily to start, and then I’ll modify the regimen based on symptomatic improvement.

If the patient does have rosacea, then I’d aggressively consider oral tetracycline. I would elect to recommend omega-3 fatty acids, but I do not feel that they have the same effect as other options. So, in these lower-staged patients, I would treat topically for the first 2 weeks and then reevaluate before moving to systemic treatment.

Dr Paul Karpecki: For these kinds of milder patients who may not consider their MGD symptoms to be a serious issue, I encourage patient compliance by imaging the eye. In my experience, a large image of even a relatively good-looking lid margin will help people understand the reasons for therapy. With eyes that have MGD, a large image can really drive the point home. I’d initially start with warm compresses. I tell patients that even if they can get just 5 minutes of good warm heat to their eyelids, it will make things a little better. I prefer commercially available warm compresses—ThermoEyes® or TranquilEyes® or the Bruder Eye Hydrating Compress. When patients make an investment, they’re more likely to comply, and these agents are not very expensive.

Dr Jason Nichols: I’d classify this patient as a 2, maybe 2-plus, because both signs and symptoms are mild, and there is trace staining.

Dr Kelly Nichols: There are 2 scenarios in which a patient might present: the patient presents during a routine eye examination, or the patient comes to your office because of the irritative symptoms and red eyes. How does your treatment differ in those examples?

What diagnostic codes should we use for the medical treatment?

Dr Alan Kabat: There is no MGD diagnostic code, and most clinicians erroneously use the code for internal hordeolum. Blepharitis is still acceptable as a medical diagnosis, and carries a diagnostic code. The problem with a blepharitis diagnosis is it’s a 00 code. In this case, blepharconjunctivitis might work, so enter CPT code 372.2. Dry eye syndrome (CPT code 375.15) is a possible choice because the patient does have secondary dry eye. I suspect rosacea in this case—the patient does have involvement of the conjunctiva, both bulbar and palpebral; rosacea conjunctivitis could be an appropriate diagnosis (CPT code 372.31).

Dr Paul Karpecki: Make sure to include rosacea itself (CPT code 695.3). Sometimes we overlook the systemic disease that’s playing a role in the ocular portion. In this case, if it is MGD stage 2, I would use loteprednol/tobramycin (Zylet®) or topical azithromycin (Azasite®) and then palliative therapies such as warm compresses, lipid-based tear supplements, and liposome spray, and follow up in a month. If it is MGD stages 2 plus or 3, and rosacea, I would include oral doxycycline and would ask the patient to return in 4 to 6 weeks. If the situation is acute, with significant erythema and swelling, one might argue infection is present. A combination anti-infective/steroid such as loteprednol/tobramycin or tobramycin/dexamethasone (TobraDex® ST) may be appropriate. In those cases, I bring the patient back in less than a month to monitor the intraocular pressure.

Dr Gary Foulks: If you do use topical azithromycin, our studies showed that 4 weeks was the point at which you saw the most response.
A 45-year-old white female who has previously worn gas-permeable and hydrogel CLs, and more recently hydrogel multifocal lenses to correct moderate myopia (4.50 D) and presbyopia (15 years of CL wear), reports gradual onset intolerance of the lenses beginning in the middle of the day with some intermittent blurring of vision, especially late in the day, and conjunctival redness upon removal of the lenses, which clears during the ensuing hour. Examination confirms good CL fit and movement but rapid TBUT. Meibomian gland plugging is seen on slit-lamp examination. None-to-trace ocular surface staining is present. There is a very small amount of CL-related staining present, but nothing obvious or significant.

Dr Jason Nichols: We need to concentrate on the MGD or posterior blepharitis component. We need to ascertain the CL material and CL care history. This patient does have a few issues that might be related to tear film instability as she goes through the day.

In terms of treatment, pharmaceuticals, omega-3s, punctal plugs—they are not usually a primary consideration for practitioners for CL patients. For this patient, I would consider a different hydrogel or a silicone hydrogel, but we might be limited because she needs a multifocal lens. I don’t think the MGD Workshop report has taken us into changing the care of the CL-intolerant/blepharitis patient yet. But it is on the horizon.

Dr Jason Nichols: There’s a link between the two. CL wearers have dry eye symptoms, but questions arise about the etiology. Some work conducted in Japan has shown that lens wear is associated with an atrophy of the meibomian glands and increased plugging of the glands, but we need more evidence regarding a causal relationship.

Dr Alan Kabat: I tend to look at things more in terms of disease than I do from a CL perspective. I’m more likely to classify this as MGD in a CL patient and treat it from that point of view. Others may see a problem with tear stability and insert punctal plugs in this patient. In my opinion, that would be the wrong way to go. If you have not treated the MGD, you are going to make this patient worse by going straight to plugs.

Dr Paul Karpecki: Plugging may exacerbate certain conditions, such as allergies, inflammatory dry eye, and MGD, and should be instituted after the condition is under control. But in other cases, punctal occlusion is the best primary treatment: for mild dry eye cases in CL wearers, or neurotrophic keratitis, plugging works really well early on. This patient seems like a very low-level case. Always get the MGD or any sign of inflammation under control first. If this is more severe MGD, then I would use plugs as a back-up step. In this patient, I’d start with topical azithromycin, and if that doesn’t work, then I’d move to switching lenses or solutions.

Dr Jason Nichols: We did a clinical trial comparing plugs to a sham procedure in about 20 symptomatic CL wearers, and after 6 weeks, the symptoms scores of both groups decreased, but no other objective tear film-based differences were detected. There have been other studies on topical treatments—2 using cyclosporine with inconsistent results. We did a trial with topical azithromycin this past year and showed fairly significant improvements in comfortable wear time compared with the control group, but no much change in some of the objective findings.

Dr Alan Kabat: What is the consensus on using lipid-based tear supplements such as a Systane Balance or Refresh* Optive™ Advanced in what clearly seems to be lipid-deficient, secondary dry eye in a CL wearer?

Dr Paul Karpecki: I’ve found patients with mild MGD tend to prefer the lipid-based tears more than patients who have more advanced or systemic disease-associated conditions. The average practitioner is looking for a tear supplement that does everything.

Dr Jason Nichols: The lipid layer in a CL wearer is about half the thickness it is in a non-lens wearer, approximately 15 to 17 nm when you put a lens in the eye. The idea of adding something is appealing. The challenge is getting the right mix of aqueous to interface with the lipid, but also with this piece of plastic. We need the right sort of lipids that will not just bind to the CL polymer without forming the necessary lipid layers. But theoretically, the idea is appealing.

Dr Kelly Nichols: I like the combination of trying to approach this from both a CL and a medical approach. One achievement the MGD Workshop report attempted is to encourage colleagues to think of this as 1 patient rather than as 2 separate issues of MGD and CL problems. In the patient in this case, what about follow-up?

Dr Jason Nichols: If I’m going the traditional route of just materials/lens care, I’d see this patient after 2 weeks. If I opt for the medical route—warm compresses and, potentially, azithromycin—I’d wait for 30 days to ensure I could see an effect.

Dr Alan Kabat: I’m a little more cautious and would have the patient back in 2 or 3 weeks, based on how symptomatic the patient is. If very symptomatic, and seemingly very proactive, I would bring the patient back sooner so we could validate or see if progress is being made. If not as symptomatic, if not as vocal, then I would certainly defer back to about 30 days.
References


1. Recently, the Tear Film and Ocular Surface Society/International Workshop on Meibomian Gland Dysfunction developed a new definition for meibomian gland dysfunction (MGD), introduced recommendations for the diagnosis and classification of the condition, and presented a management algorithm. Why were these actions undertaken?
   a. To replace outdated MGD guidelines
   b. To improve insurance reimbursement for the diagnosis and treatment of MGD
   c. To provide a consensus for the previously ambiguous condition
   d. To help improve the efficiency of eye care practice settings

2. The International Workshop on Meibomian Gland Dysfunction proposed a set sequence for testing to quantify those with MGD-related diseases, beginning with a questionnaire and then adding the use of which diagnostic assessment?
   a. Fluorescein staining
   b. Meibomian gland expression
   c. Osmolarity testing
   d. Schirmer test

3. Which of the following is a common cause of intolerance to contact lens wear?
   a. Ocular dryness
   b. Musca volitans
   c. Ocular tearing
   d. Hordeolum

4. In which location are meibomian glands most abundant?
   a. Palpebral fissure
   b. Lower eyelid
   c. Hair follicles
   d. Upper eyelid

5. Systemic deficiency of which substance directly contributes to MGD?
   a. Insulin
   b. Thyroid
   c. Androgen
   d. Vitamin D

6. Which racial population group has the highest prevalence of MGD?
   a. White
   b. Asian
   c. Hispanic
   d. African American

7. Meibomian gland dysfunction is:
   a. Synonymous with meibomian gland disease
   b. A cause of ocular neoplasia
   c. Synonymous with meibomitis
   d. A cause of posterior blepharitis

8. Which is the most common form of MGD?
   a. Obstructive meibomian gland dysfunction
   b. Meibomian sicca
   c. Hypersecretory meibomian gland dysfunction
   d. Meibomian seborrhea

9. Of which condition is MGD the leading cause?
   a. Anterior blepharitis
   b. Phlyctenular keratitis
   c. Evaporative dry eye
   d. Conjunctivitis

10. According to emerging evidence, which of the following factors contributes to the pathogenesis of MGD?
    a. Eyeglass wear
    b. Contact lens wear
    c. Artificial sunlight exposure
    d. Natural sunlight exposure

11. Based on the International Workshop on Meibomian Gland Dysfunction’s proposed new definition, which of the following best describes the location or extent of meibomian glands affected by MGD?
    a. Chronic diffuse
    b. Limited diffuse
    c. Upper eyelids
    d. Lower eyelids

12. Which best describes healthy meibomian gland secretion (meibum)?
    a. Turbid
    b. Inspissated
    c. Clear to light yellow
    d. Toothpaste-like in consistency

13. Significant qualitative or quantitative changes in meibum typically result in alteration of the _________ of the eye?
    a. Aqueous humor
    b. Tear duct
    c. Vitreous humor
    d. Tear film

14. Which statement best describes the relationship between inflammation and MGD?
    a. Inflammation is a clinical consequence of MGD
    b. Inflammation is required for the diagnosis of MGD
    c. Inflammation is a leading etiological factor of MGD
    d. There is no relationship between inflammation and MGD

15. Which systemic disease would most likely contribute to the pathogenesis of MGD?
    a. Osteoporosis
    b. Depression
    c. Asthma
    d. Rosacea

16. Which component of meibum is most crucial to tear stability?
    a. Erythrocytes
    b. Proteins
    c. Lipids
    d. Leukocytes

17. Historically, which of the following terms best describes the state of MGD in clinical practice?
    a. Overdiagnosed
    b. Underdiagnosed
    c. Overtreated
    d. Well understood
18. A 61-year-old female presents with ocular discomfort and photophobia. Which of the following is most likely the best first approach to the diagnosis, based on the International Workshop on Meibomian Gland Dysfunction’s guidance?
   a. Differentiate between evaporative dry eye and aqueous-deficient dry eye
   b. Determine if the patient has signs of anterior blepharitis
   c. Determine if the patient has dry eye
   d. Differentiate between meibomian gland dysfunction and meibomian gland disease

19. A 57-year-old female complains of contact lens intolerance. She is taking estrogen replacement therapy. Which of the following is likely the most appropriate first assessment that should be performed, based on the International Workshop on Meibomian Gland Dysfunction’s guidance?
   a. Administration of a symptom questionnaire
   b. Meibography
   c. Meibomian gland expression
   d. Measurement of TBUT

20. Which diagnostic assessment did the International Workshop on Meibomian Gland Dysfunction Group emphasize the most in terms of determining MGD staging?
   a. Measurement of the blink rate and calculation of the blink interval
   b. Schirmer test
   c. Measurement of TBUT
   d. Routine meibomian gland expression

21. In contact lens-wearing patients with MGD, during which time of the day do symptoms tend to be most troublesome?
   a. Evening hours
   b. When first awaking
   c. Overnight while sleeping
   d. Early afternoon

22. Which patient-reported ocular complaint warrants the performance of meibomian gland expression?
   a. Itching
   b. Burning
   c. Grittiness
   d. All the above

23. The International Workshop on Meibomian Gland Dysfunction incorporated within its findings a treatment algorithm. With which of the following criterion did the algorithm pair treatments?
   a. Disease stage
   b. Etiology
   c. Gender
   d. Race/Ethnicity

24. An 81-year-old male is diagnosed with stage 2 MGD. The patient is on anticoagulant therapy for atrial fibrillation. Which therapeutic option is most likely LEAST appropriate for the patient?
   a. Omega-3 fatty acid supplements
   b. Topical liposomal spray
   c. Lipid-based tear supplement
   d. Topical antibiotic

25. A decision has been made to treat stage 2 MGD with topical azithromycin. How many days, most likely, is the most appropriate follow-up period?
   a. 3
   b. 7
   c. 30
   d. 120

26. A 42-year-old female presents with contact lens intolerance. The patient does not complain of any other ocular symptoms. She has mild to moderate plaque psoriasis. Meibomian gland expression releases minimally altered secretion quality (grade 3), with an expressibility score of 1. No ocular surface staining is found. Which stage of MGD is most likely included in the diagnosis?
   a. 1
   b. 2
   c. 3
   d. 4

27. A 61-year-old female presents with complaints of ocular discomfort and photophobia that limit her daily activities. Slit-lamp examination finds meibomian gland plugging and vascularization of the posterior lid margin. Meibomian gland expression releases moderately altered secretion quality (grade 12), with an expressibility score of 2. She has moderate conjunctival and peripheral corneal staining. Which stage of MGD is most likely included in the diagnosis?
   a. 1
   b. 2
   c. 3
   d. 4

28. A 58-year-old male is diagnosed with stage 2 MGD and anterior blepharitis. Which of the following is/are likely an appropriate treatment?
   a. Topical azithromycin
   b. Lipid-based tear supplement
   c. Liposomal spray
   d. All the above are likely appropriate treatments

29. A 44-year-old female presents for a routine eye examination reporting slight intolerance to contact lenses. She has no other ocular symptoms. Her medical history includes hypertension and Sjögren syndrome. Meibomian gland expression releases minimally altered secretion quality (grade 2). No ocular surface staining is observed. Which of the following is likely the most appropriate treatment?
   a. Healthy, omega-3-rich diet, and eyelid hygiene
   b. Topical emollient lubricants and topical azithromycin
   c. Oral tetracycline derivative and lubricant ointment at bedtime
   d. Liposomal spray and topical steroid

30. A 74-year-old male is seen 1 week prior to cataract surgery. He complains of ocular itching and photophobia. There is marked redness of the eyelids, a tear meniscus of 0.8 mm, and rapid TBUT is present. Meibomian gland expression releases moderately altered secretion quality (grade 11). Which of the following is likely the most appropriate treatment plan?
   a. Prescribe eyelid hygiene, topical emollient lubricants, and topical azithromycin; recommend that the patient proceed with surgery
   b. Prescribe eyelid hygiene, topical emollient lubricants, and topical oral doxycycline; recommend that the patient proceed with surgery
   c. Prescribe eyelid hygiene, topical emollient lubricants, and tea tree oil scrubs; recommend that the patient delay surgery
   d. Prescribe eyelid hygiene, lipid-based tear supplements, and topical azithromycin; recommend that the patient delay surgery