

Commentary: Meibomian gland dysfunction and ocular surface analysis

Meibomian gland dysfunction (MGD) is characterized by obstruction of the meibomian gland terminal ducts and/or changes in their glandular secretion, resulting in changes in tear film stability, inflammation, and symptoms of irritation.^[1] It is one of the most common conditions encountered by ophthalmologists and is a leading cause of evaporative dry eye disease. MGD causes certain morphological changes in the meibomian glands, including dropout, shortening, truncation, distortion, and dilation. Changes to lid morphology are a hallmark of severe MGD; however, the goal is to diagnose these patients before this end-stage presentation. Meibography is an objective and repeatable examination method. When taken together with subjective symptoms and lid margin findings, it allows highly reliable diagnostic evaluation of MGD.

Arita *et al.*^[2] described the first noncontact infrared meibography as a lamp with an infrared charge-coupled device video camera and infrared transmitting filter. Since then, meibography has come a long way over the past decade. Now, computerized noncontact infrared meibography systems are available as attachments with corneal topographers and fundus photographers, or as independent mobile devices. These devices detect tear film lipids through interferometry and are capable of 3-D meibography and measurement of noninvasive tear break up time and tear meniscus height. Spectral domain optical coherence tomography (OCT) can also be used for infrared meibography. These OCT devices can contribute to the field of meibography with increased visualization, which will help elucidate anatomic details of meibomian gland dropout.^[3] Laser scanning confocal microscopy is another method of assessing meibomian gland dropout and involves everting the lids and placing the device on the conjunctiva to scan *in vivo* in both vertical and horizontal movements. This technique can be used to measure acinar unit diameter, acinar unit density, and periglandular inflammatory cell infiltrate. Acinar diameter is significantly larger in MGD patients, and acinar unit density is significantly lower in MGD patients.^[4]

A better understanding of meibomian gland morphology and ocular surface imaging has led to the incorporation of various in-office procedures to treat MGD. These procedures primarily target inspissated meibomian glands by using heat or mechanical energy to express glands and restore the natural flow of meibum. Intense pulsed light (IPL) is one such technique that involves targeting pigmented or vascular lesions with visible and infrared light, which, upon absorption, is converted to destructive heat.^[5] IPL therapy involves direct application of 500-nm light to the skin; this coagulates the underlying blood vessels. Proposed mechanisms of action include reduced inflammatory mediators and bacterial overgrowth by the destruction of eyelid telangiectasias. It also causes the melting of viscous meibum, allowing for its improved flow. Vectored thermal pulsation is another technique that combines meibomian gland expression with heat. During this procedure, the device applies heat over the palpebral conjunctiva of the upper and lower eyelids, while providing pulsatile external pressure.^[6]

Although noncontact meibography techniques are becoming increasingly popular, there is a lack of definitive evidence linking meibography findings to the true structure and composition of meibomian glands. In addition, meibography is not always sufficiently sensitive or specific to account for the patients' symptoms. The current study^[7] by the authors is thus an important addition to the existing literature to supplement early diagnosis and treatment of MGD.

Abhishek Dave

Consultant, Cornea and Refractive Surgery Services,
Centre for Sight, Safdarjung Enclave, New Delhi, India

Correspondence to: Dr. Abhishek Dave,
Consultant Cornea and Refractive Surgery Services,
Centre for Sight, B-5/24, Safdarjung Enclave,
New Delhi - 110 029, India.
E-mail: abhishek_dave@yahoo.co.uk

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