Intense, Pulsed Light for Dry Eye Syndrome

The light acts as a warm compress that liquifies the meibomian gland's secretions and ultimately unplugs them.

BY ROLANDO TOYOS, MD

ry eye syndrome is one of the most common disorders seen in any general ophthalmology practice. The popularity of refractive procedures, such as laser vision correction and the implantation of premium IOLs, has increased ophthalmologists' need to treat dry eyes in order to achieve satisfactory surgical outcomes.

The most common problem that ophthalmologists see in cases of dry eye disease is a failure of the meibomian glands to function properly. The meibomian glands are responsible for secreting the lipid layer of the tear, which prevents the premature evaporation of the tear complex. Meibomian gland dysfunction (MGD) causes meibum to accumulate, which leads to inflammation and bacterial infiltration of the gland. The bacteria contain lipases that alter the polarity of the tear and allow its quick evaporation as well as further block the meibomian gland. Poor vision, pain, redness, and swollen eyelids ensue.

Diagnosis is easy upon examination of affected patients' lids and glands. Additional tests include corneal staining and tear breakup time. Curing dry eye disease, however, has proven difficult. Drops can alleviate inflammation and infection, improve the tear film, and perhaps the meibomian gland itself. The routine use of warm compresses and lid scrubs is also beneficial. In addition to these measures, I have my patients with MGD undergo intense, pulsed-light treatments, which break the cycle of blepharitis and dry eye syndrome.

THE CONNECTION BETWEEN INTENSE, PULSED LIGHT AND MGD

I first suspected that therapy with intense, pulsed light could improve MGD in 2003 when my colleagues and I opened an aesthetics clinic. I would see patients with rosacea who had various ocular disorders and would refer them to the aesthetics clinic to have their telangiectasia and facial redness treated with intense, pulsed light.

The treatment had become an established therapy.¹⁻³ These patients returned reporting improvement in their skin, but they also said that their eyes felt better and their vision had improved. Upon examination, I found improvement in their lid margins, meibomian glands, and tear breakup time. My colleagues and I began to study the link. We determined that light therapy not only worked for patients with rosacea but also for most individuals who had MGD and were eligible for intense, pulsed light. We received an ASCRS grant in 2004 for our research and have presented our ongoing data at several meetings including the ASCRS annual meeting.

Six years later, I have treated hundreds of patients successfully with intense, pulsed light. I have developed a nonproprietary standard regimen for treating patients to achieve maximum results that any ophthalmologist with the proper training should be able to follow. To my knowledge, no one besides myself is using this technique.



Figure 1. The author performs intense, pulsed-light therapy on a patient.

THE BASICS

Intense, pulsed light uses a flash lamp that emits energy in a band from a base of the visible spectrum (400 nm) to the border between the near and midinfrared (1,300 nm). The flash lamp is directed through a crystal to skin tissue. A filter limits the transmission discharged in order to protect the tissue. Blocking portions of the spectrum can safeguard the tissue not targeted and avoid complications (Figure 1).

The first set of patients my colleagues and I treated had obvious telangiectasias on their lid margin next to the meibomian glands (Figure 2). We used a vascular setting that allowed light in the 500-nm range to target the hemoglobin in red blood cells. The hemoglobin absorbs the energy and coagulates, causing thrombosis of the blood vessel. One reason why intense, pulsed light may work is that the closed blood vessels can no longer send inflammatory mediators to the gland. These mediators may cause the gland's dysfunction.

The first thing we noticed in patients treated with intense, pulsed light is that their telangiectasias disappeared, as did the erythema and swelling in the gland. Consequently, the eyelids themselves appeared cleaner. One month after treatment, meibomian glands that had not functioned at all began to operate. Instead of resembling toothpaste, the secretion we observed was thinner and more normal.

TREATMENT REGIMEN

When patients first present to our clinic with MGD, we have to determine if they have active blepharitis. We have found that intense, pulsed light prevents blepharitis. The therapy alone, however, cannot treat an active infection. If the patient is a candidate for therapy but has active blepharitis, he undergoes treatment with Azasite (azithromycin ophthalmic solution 1%; Inspire Pharmaceuticals Inc., Durham, NC) once daily at night until the bottle is empty. We instruct patients to rub in any excess medicine on their eyelids after application. They also scrub their lids in the morning with baby shampoo when they shower. When they come in for light therapy, the MGD persists, but the active infection has been treated. All medications are stopped prior to the treatment.

We are currently using the newest Dermamed Q4 (DermaMed International, Inc., Lenni, PA) intense, pulsed-light system. We set the system to the acne treatment platform in the Fitzpatrick type 1 skin setting. We set the fluence anywhere from 8 to 12 J/cm² with a pulse width of 20 to 30 milliseconds, depending on the skin's response. We place an external instrument within the ray of the intense pulsed light to shield the patient's eyelids. Treatment begins at one tragus of the ear and moves across to the other tragus before repeating to complete a double pass. We apply a cool ultrasound gel on the skin before performing the



Figure 2. A patient with MGD presents to the author's practice with extensive erythema, telangiectasias, notching of the lid margin, closed meibomian glands, and a poor tear film.

treatment. When the light reaches the lower eyelid, we have patients look up and then apply treatment right below the eyelash margin. The upper meibomian glands are not treated directly, but we have seen an improvement in the upper glands from indirect therapy.

Upon completion of the second pass, we remove all of the gel and shields. We tell patients to apply sun block and avoid sun exposure for 1 week. They may have some mild redness of the skin, which normally goes away in a few hours. Sometimes, the redness may last a few days. To help decrease the mild acute inflammation of their eyelids after intense, pulsed-light therapy, patients use Xibrom (bromfenac; Ista Pharmaceuticals, Inc., Irvine, CA) drops twice daily for 4 days.

We inform patients that they will receive four to six treatments spaced 1 month apart. Our endpoint is the proper functioning of 90% of the lower lid glands or three successive treatments with no increase in the number of glands working. A small minority of patients may not respond to treatment. We increase the fluence in these cases. If their meibomian glands do not improve after three treatments, we consider them to be nonresponders. During the last year, fewer than 1% of our treated patients have been nonresponders.

In my experience, each treatment increases the tear breakup time. The maximum increase occurs after four treatments. All patients report a subjective improvement in their dry eye symptoms (no steroids were prescribed after treatment). On examination, we find that patients with telangiectasia at the lid margin will have less after only one treatment. Glands will show more fluid secretions when pressed. We also find less capping of the meibomian glands. The lid margins are less erythematous and clearer. After several treatments, the anatomy of the lid and skin has a better cosmetic appearance. In some patients, glands that were

not functioning at all and seemed to have "mushroom capping" of the opening will still have some capping, but they will have normal secretions that can be expressed at the edges of the cap. In these cases, I recommend the use of warm compresses (applied for 3 minutes once daily) to help open the gland to improve expression. Although compresses may have been ineffective before therapy, they often alleviate symptoms afterward.

CONCLUSION

I believe that intense, pulsed light works in three ways to improve meibomian gland function. First, it acts as a powerful warm compress to liquefy the toothpaste-like secretion plugging the gland. Second, the intense, pulsed light closes the microvasculature that is feeding the inflammatory mediators to the gland that inhibit normal function. Third, the light improves lid apposition and thus the pumping mechanism of the meibomian gland during blinking. When the function of the meibomian gland improves, recurrent ble-pharitis ends.

After patients complete the four- to six-treatment regimen, they periodically return for maintenance therapy as needed. Some of our patients come in every 3 months, whereas others will not return for 1 year. Most of our pa-

tients no longer need eye drops after they have finished their initial course of treatment. They usually return for a maintenance treatment when they feel that their symptoms are coming back.

We only treat patients with Fitzpatrick skin types 1 and 2, although we will consider some type 3 patients. If we are unsure whether a patient can undergo intense, pulsed-light therapy because we are not sure of their skin type, we will place a test spot on their skin. Patients with dark skin tones cannot receive this therapy, because it will discolor their skin.

Many treatments for dry eye only help with some of the symptoms. Intense, pulsed-light therapy immediately improves symptoms and the meibomian gland's function.

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