Paralysis of the Orbicularis Oculi is Associated with Pathologic Changes in Meibomian Gland Morphology on Infrared Meibography

Ryan J. Wise, B.S., B.A.; Conley B. Call, M.D.; Marlan Hansen, M.D.;
Keith D. Carter, M.D.; Randall E. Verdick, BA, FOPS; Richard C. Allen, M.D., Ph.D.

University of Iowa Health Care
Department of Ophthalmology and Visual Sciences

The University of Iowa

BACKGROUND

Tears are composed of aqueous, mucous, and lipid layers. The lipid layer of tears is produced by meibomian glands located within the tarsal plate of the eyelid. Meibomian gland dysfunction (MGD) leads to abnormal tear function resulting in ocular irritation and surface disease.

The orbicularis oculi primarily protracts the eyelid during the blink cycle. Although the mechanism responsible for inducing the meibomian glands to secrete lipids is unknown, previous studies suggest that a secondary function of the oribicularis is compression of the glands during muscle contraction. Consequently, the orbicularis may play a dual role in ocular health by facilitating eyelid blink and promoting normal tear function.

Orbicularis weakness is seen in various conditions including myotonic dystrophy, chronic progressive external ophthalmoplegia, periocular botulinum toxin injections, and facial nerve palsy. Patients with orbicularis weakness commonly complain of dry eye presumably due to exposure keratopathy; however, MGD would aggravate any dry eye condition.

Video infrared (IR) meibography is a non-invasive technique for the direct observation of meibomian gland morphology *in vivo*. IR meibography permits the investigator to quantitate meibomian gland dropout and other morphological abnormalities of the glands. Previous meibography studies found a 25% or greater gland dropout and other morphological abnormalities in cases of MGD compared to 5% gland dropout in controls.

We believe an overlooked etiology of MGD is orbicularis weakness which can be demonstrated using IR meibography.

PURPOSE AND HYPOTHESIS

Hypothesis: Orbicularis weakness leads to changes in meibomian gland morphology.

Purpose: To determine whether or not there is an association between meibomian gland morphology and orbicularis weakness in the setting of facial nerve palsy using IR meibography.

MATERIALS AND METHODS

Design: Non-randomized, cross-sectional study

Methods:

• Population: Patients with a unilateral facial nerve palsy. Involved eyelids served as the experimental group. Fellow normal eyelids served as controls.

• Exclusion criteria: Any history of eye surgery, ocular allergies, use of eye drops

• Data collected: Patient age, sex, date of diagnosis of facial nerve palsy

• IR meibography: A 1 inch IR probe was used to evert and transilluminate the eyelid which was recorded by an IR camera (Fig. 1). Meibography images were captured using Adobe Premier Elements.

• Data Analysis: Meibomian gland images were quantitated using the meibograde method. The meibograde scores meibomian glands based on three categories: distortion, shortening, and dropout. Each category is scored 0 -3 based on the area of eyelid involved: score 0, no area involved; 1, < 33% involved; 2, 33%-66% involved; 3 , >66% of area involved. Category summation gave an overall eyelid score termed meibograde (Fig. 2).

Infrared Meibography Studies

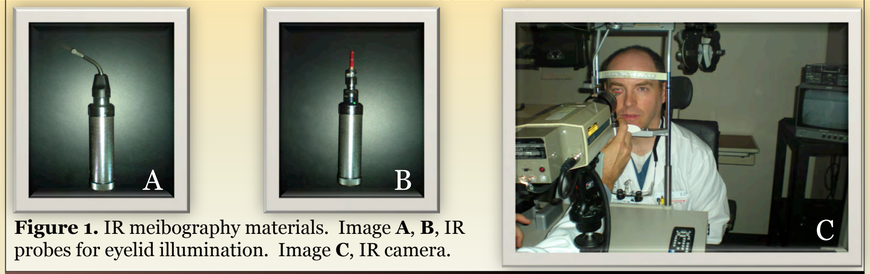

Figure 1. IR meibography materials. Image **A**, **B**, IR probes for eyelid illumination. Image **C**, IR camera.

Infrared Meibography Imaging

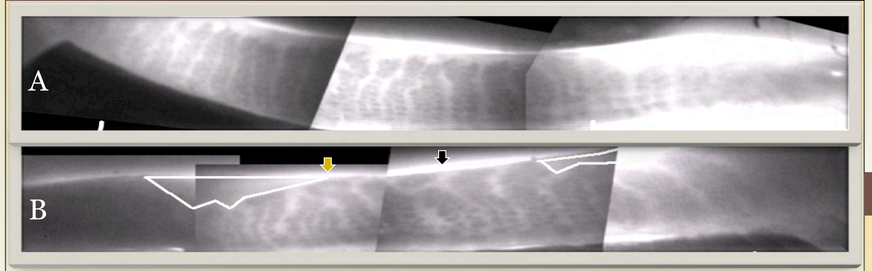

Figure 2. IR meibography of the lower eyelids in a patient with facial nerve palsy at 6 year follow up. Meibomian gland lipids scatter IR light causing the glands to appear as dark streaks along the palpebral conjuctiva. **A**, **B** Images of the control and affected eyelids. The control eyelid **(A)** corresponds to a meibograde of 0. The eyelid affected by facial nerve palsy **(B)** corresponds to a meibograde of 5. Note that the affected eyelid shows signs of glandular distortion (black arrow) and shortening (yellow arrow) as well as gland dropout (circled white).

Infrared Meibography Meibograde

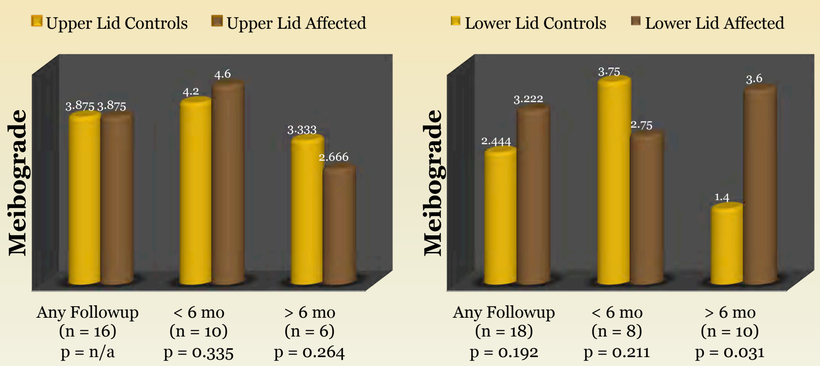

Figure 3. Graph of mean meibograde scores in control and facial nerve palsy eyelids at any, less than 6 months, and greater than 6 months follow up time. Graph on left shows upper eyelid meibogrades. Graph on right shows lower eyelid meibogrades.

RESULTS

A total of 34 eyelids (9 females, 2 males; mean age 58.0 years) with or without pre-existing facial nerve palsy were imaged using IR meibography. Control and experimental eyelids were categorized into groups based on follow up time since the date of diagnosis: any time, <6 months, >6 months. The meibograde was used to score eyelids (figure 3).

1. At any follow up, there was no statistically significant difference in the mean meibogrades between the facial nerve palsy and control groups in either the upper eyelids (3.875 vs. 3.875, respectively; p = n/a) or lower eyelids (3.222 vs. 2.444, respectively; p = 0.192).
2. At less than 6 months follow up, there was no statistically significant difference in mean meibogrades between the facial nerve palsy and control groups in either the upper eyelids (4.6 vs. 4.2, respectively; p = 0.335) or lower eyelids (2.75 vs. 3.75, respectively; p = 0.211).
3. At greater than 6 months follow up, there was no statistically significant difference in the mean meibogrades between the facial nerve palsy and control groups in the upper eyelids (2.666 vs. 3.333, respectively; p = 0.264).
4. At greater than 6 months follow up, there was a statistically significant difference in the mean meibogrades between the facial nerve palsy and control groups in the lower eyelids (3.6 vs. 1.4, respectively; p = 0.031).

DISCUSSION

Meibomian glands are located in the tarsal plate of the eyelid and produce lipids important for tear function. The mechanism inducing meibomian glands to release lipids is unknown but dysfunction can lead to ocular irritation and surface disease. Studies suggest that a region of the orbicularis oculi called the muscle of Riolan contracts around the meibomian glands causing them to expel lipids into the tear film. We hypothesize that orbicularis weakness secondary to facial nerve palsy would therefore lead to loss of meibomian gland activity and changes in gland morphology over time.

Results of this study demonstrate an association between orbicularis weakness and progressive changes in meibomian gland morphology compared to controls (Fig. 3). Morphologic changes observed in the meibomian glands included distortions, shortening, and dropout (Fig. 2).

Interestingly, we found that patients with >6 months of orbicularis weakness had a significant difference in meibomian gland morphology within *lower* eyelids but not upper eyelids . This may reflect anatomical differences in the muscle of Riolan between the upper and lower eyelids. It could also result from a greater meibomian gland density in the upper eyelids which would require more time for changes to have a significant effect.

CONCLUSIONS

1. **Patients with orbicularis weakness for less than 6 months are not likely to show pathologic changes in meibomian gland morphology.**
2. **Pathologic changes in meibomian gland morphology in the setting of orbicularis weakness for greater than 6 months preferentially involve the lower eyelid.**
3. **Meibomian gland dysfunction should be included in the differential diagnosis for patients presenting with orbicularis weakness and ocular irritation.**