

Optimizing Treatment for Diabetic Macular Edema With Laser

Combination therapy can enhance the effects of anti-VEGF agents.

BY RAHUL N. KHURANA, MD

Although anti-VEGF monotherapy is effective for treating diabetic macular edema (DME), the burden of monthly injections weighs heavily on patients and, often, is not a sustainable model.

Combination therapy with anti-VEGF and laser photocoagulation is particularly effective in patients with either chronic or diffuse DME because data have shown that anti-VEGF monotherapy may be less successful for these patients.¹

I have found several benefits to combination therapy. First, I can rapidly reduce some thickening of the retina with anti-VEGF therapy and follow this with laser photocoagulation. Second, the laser can be applied more precisely and at lower power settings on nonedematous retinal tissue. This is an important advantage because, when an edematous retina is treated with laser, the spot size increases at the level of the retinal pigment epithelium compared with laser spots in a nonedematous retina. The thickened retina then reacts like a prism and spreads out the light, resulting in a larger, more diffuse burn. Furthermore, after I initially reduce the macular edema with anti-VEGF therapy, I can apply the laser photocoagulation more precisely and with less collateral damage to the surrounding retina.

NEW SOFTWARE FOR TITRATION

I have had success with a recently developed software application called Endpoint Management (EpM; Topcon Medical Laser Systems) that is designed to increase control over laser titration. The software includes a feature for placing visible landmarks while applying treatment. I can now treat closer to the fovea without worrying about causing the collateral damage that occurs with more traditional laser photocoagulation systems.



Figure 1. Before laser. Fundus photography shows cystoid macular edema with lipid exudates, intraretinal hemorrhages, and cotton wool spots.

My preoperative evaluation consists of a dilated fundoscopic examination, a fluorescein angiogram, and quantitative measurement with an optical coherence tomography scan. These imaging models reveal the perfusion status of the retina and the amount of macular edema, and they provide a baseline with which to compare results.

I use a contact lens (Area Centralis, Volk) to deliver the laser. After I anesthetize the eye with a topical drop, I apply

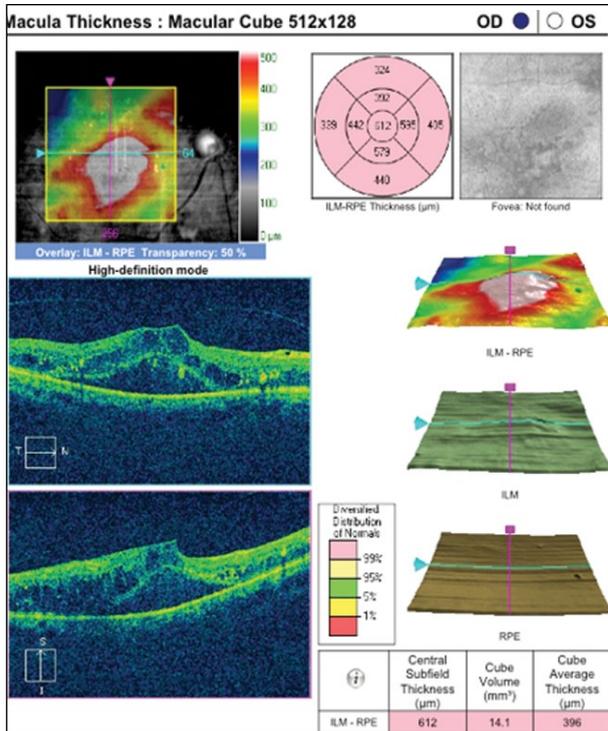


Figure 2. Before laser. Visual acuity was 20/400 and central subfoveal thickness was 612 μm with diffuse retinal thickening.

Doing a test burn maximizes the benefit of the laser and minimizes damage that can occur from overtreatment.

hydroxypropyl methylcellulose to the contact lens and place it on the eye.

I titrate the laser, always starting at a low power setting for my test burns. Doing a test burn maximizes the benefit of the laser and minimizes damage that can occur from overtreatment. I start with a power of 100 mW and increase the power to achieve a satisfactory burn. I fire a few test shots into the periphery until I see a light laser burn on the retina.

CASE STUDY

A 55-year-old man with diabetes was referred with 1 year of vision loss in his right eye. The patient had no other ocular history. He had significant diabetes and



Figure 3. The screen of the PASCAL Laser System with Endpoint Management software. The central pattern shows landmark spots in red, while endpoint spots are shown in yellow. Endpoint power is approximately half of the power used for landmarks.

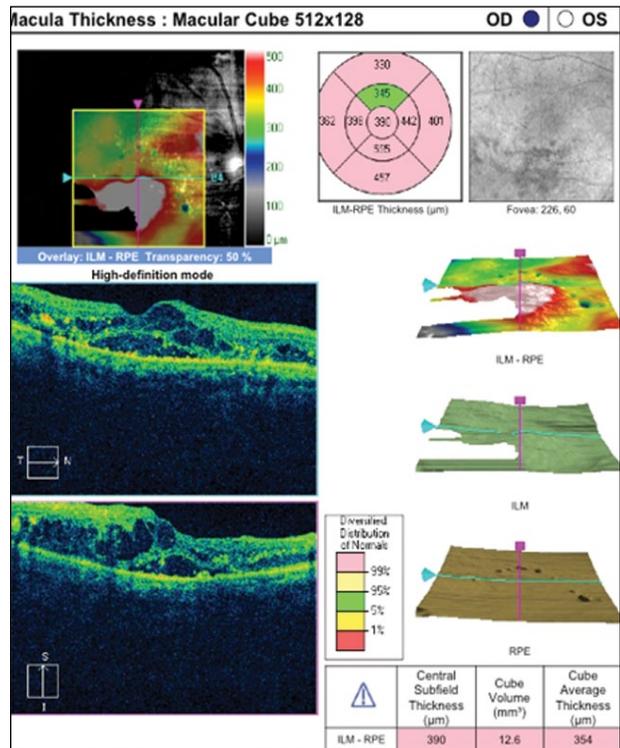


Figure 4. Five weeks postlaser. Visual acuity remained at 20/400, while central subfoveal thickness decreased to 390 μm.

hypertension. His medications included metformin and lisinopril.

On examination, visual acuity was 20/400 and intraocular pressure was 11 mm Hg in the right eye. Slit-lamp examination was unremarkable. Dilated funduscopic

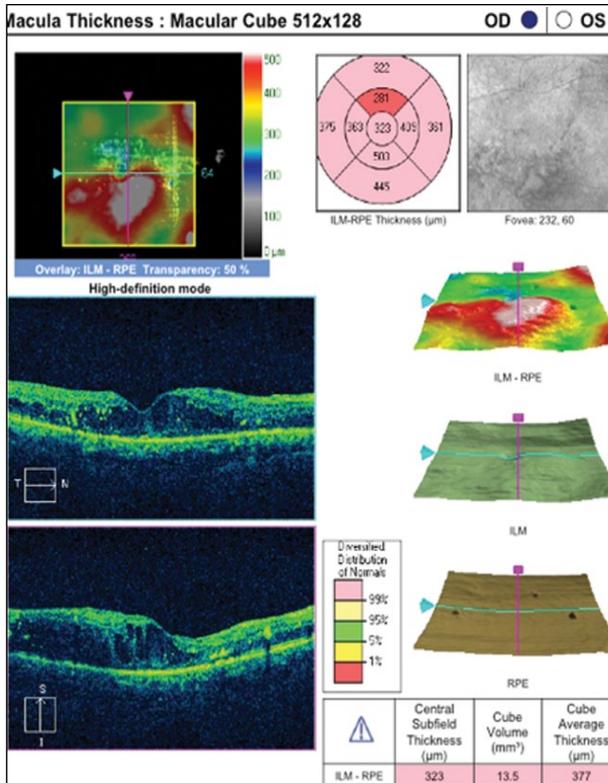


Figure 5. Nine weeks postlaser. Visual acuity improved to 20/100, and central subfoveal thickness decreased to 323 µm.

examination revealed cystoid macular edema with lipid exudates. Fundus photography showed cotton wool spots and intraretinal hemorrhages (Figure 1). Fluorescein angiography showed numerous microaneurysms leaking on late frames. Optical coherence tomography scans showed diffuse retinal thickening with subretinal fluid. The central subfoveal thickness was 612 µm (Figure 2).

Various treatment options—including anti-VEGF therapy, steroids, and laser treatment—were discussed with the patient. The patient declined any intravitreal injections and decided in favor of focal laser photocoagulation. The PASCAL laser (Topcon) with EpM software was utilized. Using a single-spot setting, the laser was titrated to 200 mW for 15 ms for a light burn. Using the EpM software with the landmark function enabled, laser photocoagulation was completed (Figure 3).

Five weeks later, visual acuity remained at 20/400, but the central subfoveal thickness decreased to 390 µm (Figure 4). Nine weeks after laser photocoagulation, visual acuity improved to 20/100, and the central subfoveal thickness decreased to 323 µm (Figure 5). Eighteen weeks after laser photocoagulation, the visual acuity remained at 20/100, and the central subfoveal thickness decreased to 256 µm (Figure 6).

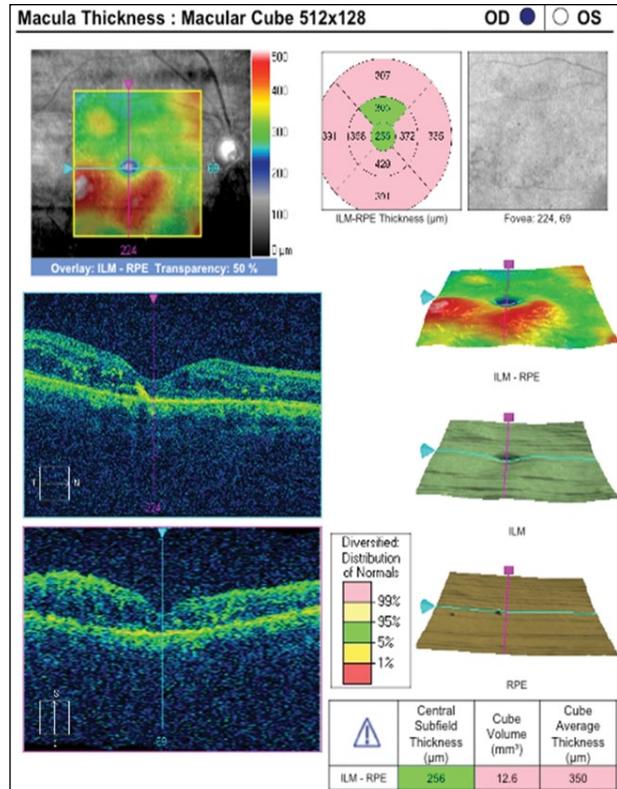


Figure 6. Eighteen weeks postlaser. Visual acuity remained at 20/100, and central subfoveal thickness decreased to 256 µm.

CONCLUSION

The efficacy and duration of a single laser photocoagulation treatment is illustrated in this challenging case of chronic diffuse DME. Laser photocoagulation continues to have an important place in the management of DME and is complementary to anti-VEGF treatment. ■

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1. Solaiman KA, Diab MM, Dabour SA. Repeated intravitreal bevacizumab injection with and without macular grid photocoagulation for treatment of diffuse diabetic macular edema. *Retina*. 2013;33(8):1623-1629.

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