Laser Photocoagulation of Diabetic Macular Edema

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Diabetic retinopathy is a major cause of blindness, accounting for 10% of new cases of blindness at all ages and 20% of new cases of blindness between the ages of 45 and 74 in the United States.¹ Ocular complications of diabetes include nonproliferative and proliferative retinopathy and diabetic maculopathy, which can accompany either one. Diabetic maculopathy may present as macular edema, macular ischemia, or a combination of the two. Whereas diabetic eyes with nonproliferative disease generally have good visual acuity and a good prognosis, macular edema remains the major cause of visual loss in diabetics.²⁻⁵ However, approximately 50% of the visual loss from macular edema in diabetics can be reduced by laser photocoagulation. Prevention of visual loss from diabetic macular edema by laser photocoagulation is one of the goals of the “Diabetes 2000 Project” of the American Academy of Ophthalmology, whose overall goal is elimination of preventable blindness from diabetes by the year 2000.

HISTORY

Information regarding the incidence of macular edema in diabetics comes from select populations,⁶⁻⁹ clinical trials,¹⁰⁻¹² or population-based studies.¹³⁻¹⁵ In a report by Fatz and Fine, macular edema was the greatest single cause of visual impairment in 1100 consecutive patients with diabetic retinopathy referred to the Diabetic Retinopathy Center at Johns Hopkins Hospital.¹⁶,¹⁷ The Wisconsin Epidemiologic Study of Diabetic Retinopathy, which was initiated in 1980 and which provided 4-year follow-up, found that the overall incidence of macular edema in a group of younger-onset, insulin-dependent diabetics was 8.2%; in a group of older-onset diabetics using insulin, the rate was 8.4%; and in a group not using insulin, the rate was 2.9%.¹⁵ Macular edema was found to be most strongly associated with the duration of diabetes.¹⁸ The incidence of macular edema was 0% at 5 years and 29% at 20 years or more if diabetes had been diagnosed before the age of 30, and 3% at 5 years and 28% at 20 years if diabetes had been diagnosed after the age of 30.¹⁸ The study also found that 38% of diabetics with diagnosed edema had central macular involvement.¹⁸

Diabetic macular edema (DME) is defined as thickening of the retina within one disc diameter of the center of the macula in the Diabetic Retinopathy Study (Table 1).¹⁹ Olk defined diffuse diabetic macular edema (DDME) as two or more disc areas of retinal thickening involving the foveal avascular zone,²⁵,20⁻²⁴ and recommended modified grid laser photocoagulation in DDME.²⁰,²¹,²⁴ The Early Treatment Diabetic Retinopathy Study (ETDRS) recommended focal or grid photocoagulation for clinically significant diabetic macular edema (CSDME).¹² The ETDRS definition of CSDME is based on the presence of any one of the following three characteristics: (1) thickening of the retina at or within 500 µm of the center of the macula; (2) hard exudates at or

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TABLE 1
Definitions of Diabetic Macular Edema

| 1. Diabetic macular edema (DRS\textsuperscript{19}) | Thickening of the retina within one disc diameter of the center of the macula. |
| 2. Diffuse diabetic macular edema (Olk\textsuperscript{20}) | Two or more disc areas of retinal thickening involving the foveal avascular zone. |
| 3. Clinically significant diabetic macular edema (ETDRS\textsuperscript{12}) (any one of the three) | 1. Thickening of the retina at or within 500 μm of the center of the macula. |
| | 2. Hard exudates at or within 500 μm of the center of the macula "if associated" with thickening of the adjacent retina. |
| | 3. A zone or zones of retinal thickening one disc area or larger, any part of which is within one disc diameter of the center of the macula. |

within 500 μm of the center of the macula “if associated” with thickening of the adjacent retina; or (3) a zone or zones of retinal thickening one disc area or larger, any part of which is within one disc diameter of the center of the macula.\textsuperscript{12} The ETDRS has shown that photocoagulation for CSDME reduces the risk of visual loss from macular edema by 50%.

DIAGNOSIS
Detection of DME necessitates careful binocular fundusscopic examination by slit-lamp biomicroscopy. Treatment, on the other hand, is based on clinical and fluorescein angiography (FA) findings. Diagnosis can be made using either a contact lens or one of the high power +78-D or +90-D noncontact lenses. Diagnosis of DME is clinical and does not depend on FA findings.

INDICATIONS FOR SURGERY
Laser photocoagulation for DME is indicated if a patient has DDME or CSDME. Treatment should be applied regardless of the visual acuity. Even eyes with 20/20 or better vision should be considered for treatment if DDME or CSDME is present.

TABLE 2
Preoperative Evaluation

| 1. Complete ophthalmologic examination |
| 2. Fundus photography |
| 3. Fluorescein angiography |
| 4. Systemic evaluation by family physician, internist, or diabetes specialist |

Patients with macular ischemia alone will not benefit from laser photocoagulation. However, macular ischemia may also coexist with DME. In Olk’s studies, patients with DDME associated with parafoveal ischemia of 6 clock hours or less did equally well with modified grid laser photocoagulation regardless of the laser wavelength, either argon blue–green (488 nm),\textsuperscript{20} argon green (514 nm), krypton red (647 nm),\textsuperscript{21} or diode laser (810 nm).\textsuperscript{24}

Furthermore, our clinical experience confirms that patients with large areas of parafoveal ischemia involving more than 6 clock hours of the foveal avascular zone (FAZ) associated with DDME may also benefit from modified grid laser photocoagulation, as macular edema contributes to decreased vision to a certain extent in those patients. Additionally, although previous studies of the treatment of DME have included patients with 20/200 or better vision, our clinical experience has shown that patients with extensive DDME and visual acuity less than 20/200 may occasionally benefit from modified grid laser photocoagulation.

PREOPERATIVE EVALUATION
Preoperative evaluation should consist of a complete ophthalmologic examination and fundus photographs. If a clinical decision to treat the patient has been made, then FA is also performed (Table 2). FA provides a basis for confirming which areas to treat. Focal laser photocoagulation implies the treatment of areas of focal leakage seen in the early phases of the FA. Grid laser photocoagulation is performed by applying equally spaced spots throughout areas of diffuse thickening and capillary nonperfusion. Modified grid laser photocoagulation is grid treatment of areas of diffuse thickening and capillary nonperfusion, in addition to individual treatment of focally leaking areas.

Patients who are to be treated should be as stable as possible systemically. Increased glycosylated hemo-
globin and blood sugar levels of 200 mg/dl or greater have been shown to be associated with a significantly increased frequency of microvascular complications. The Wisconsin Epidemiologic Study of Diabetic Retinopathy showed an increasing severity of diabetic retinopathy and hard exudates with increasing serum cholesterol in insulin-dependent persons. We have recently shown that patients with evidence of systemic vascular disease (defined as a positive history of any of the following: intermittent claudication; amputation of one or more digits; peripheral bypass surgery; stroke, carotid endarterectomy, or carotid bypass surgery; or myocardial infarction or coronary artery bypass surgery) did poorly after modified grid laser photoagulation for DDME.

A recent study, the Diabetes Control and Complications Trial (DCCT), has demonstrated that intensive insulin therapy can delay the onset and slow the progression of retinopathy, nephropathy, and neuropathy in patients with type I diabetes mellitus. The DCCT advised caution in employing intensive therapy in type II diabetes mellitus. There is also some evidence that rapid improvement of long-standing poor control may increase the risk of retinopathy progression in some type I patients as well, and such patients should be monitored more frequently for progression of retinopathy.

Therefore, we think that patients should be in an optimal state of systemic diabetic control prior to laser photoagulation for DME. Patients whose diastolic pressure is greater than 100 mm Hg or whose hemoglobin A-1C is greater than 10 mg/dl should be referred for further medical management prior to laser photoagulation for DME and should be stabilized systemically in regard to hypertension, blood sugar control, and renal failure.

**TECHNIQUE**

Focal, grid, or modified grid laser photoagulation can be used to treat DME.

Treatment is performed as an outpatient procedure with topical anesthesia. Usually, unilateral treatment is performed; however, bilateral treatment can also be performed, especially under extenuating circumstances (e.g., a patient’s travel difficulties). A frame of a recent FA projected on a viewer adjacent to the patient serves as a reference during the treatment session. For treatment, we prefer the Mainster standard contact lens (Ocular Instruments, Bellevue, WA), which provides excellent magnification of the poste-

![Figure 1. Focal treatment (Early Treatment Diabetic Retinopathy Study) to the areas of focal leakage.](image)

or pole and stereopsis. This lens gives an inverted reverse view of the fundus.

In the ETDRS, the goal of focal treatment was closure or obliteration of leaks, using an argon green (514 nm) laser (Fig. 1). Focal leaks were initially treated lightly with 100- to 200-μm spots to create a light gray background burn. Closure of the focal leak with a 50- or 100-μm burn completed the treatment. Exposure time was 0.05 to 0.1 second. A 0.05-second exposure was suggested for leaks within 500 μm of the fovea. Power was adjusted to whiten the microaneurysm with the closure technique. At the first session, all areas of focal leakage between 500 μm and two disc diameters from the center of the macula were treated. Treatment of the lesions between 300 and 500 μm from the center was considered when the patient's visual acuity was worse than 20/40 without parafoveal capillary nonperfusion. Eyes were examined 3 to 4 months later to detect areas of retinal thickening and CSDME. FA was repeated to detect new or residual areas of focal or diffuse leakage. Together, the findings were used to determine whether additional laser therapy was needed. This regimen was repeated at 3- to 4-month intervals thereafter until all areas of leaking microaneurysms were occluded.

Grid treatment was performed if areas of diffuse leakage were detected in the mid to late phase of FA. This leakage was thought to be secondary to permeability abnormalities within dilated capillaries. It resulted in areas of retinal thickening usually without readily identified areas of focal leakage. Treatment involved placing light burns of argon green, with an
exposure time of 0.1 second, 100 to 200 μm in size, in the areas of leakage that resulted in retinal thickening that were more than 500 μm from the center of the macula and 500 μm from the outer margin of the optic disc. Power was set at 50 mW initially and was increased slowly to obtain a light gray–white lesion. Burns were spaced at one spot size width apart.

Modified grid laser photocoagulation for DDME consists of applying grid treatment to areas of retinal thickening and capillary nonperfusion in the posterior pole outside the parafoveal area (Fig. 2). Two or three rows of laser spots are applied in the parafoveal region up to and including the edge of the FAZ. Spot size is 100 μm or 125 μm for argon green and diode laser, respectively, in the parafoveal area. Spots are placed one spot size apart from each other. Then, 200-μm spots spread 200 μm apart from each other are applied throughout all remaining areas of retinal thickening and in all areas of capillary nonperfusion. In areas of obvious focal leakage, additional spots are applied focally. Laser parameters are similar to what was described above for focal or grid argon green photocoagulation, but differ for longer wavelength lasers such as the diode laser. Argon green spots are applied with powers ranging from 100 to 300 mW; for primary treatment with the diode laser, powers ranging from 200 to 700 mW are used. The goal of treatment is to keep the burns as light as possible, obtaining burns just barely visible at the level of the outer retina or retinal pigment epithelium. One hundred–millisecond duration is used for argon green and 200-millisecond duration is used for the diode laser (Table 3). Patients are seen every 3 to 4 months. If clinical examination demonstrates residual retinal thickening involving the FAZ, then supplemental modified grid photocoagulation is applied to those areas where residual edema or nonperfusion is present. For any supplemental treatment, duration, power, and spot size parameters are similar to those of the primary treatment.

Absorption and thermal effects of various laser wavelengths, including argon blue–green (488 to 514 nm), argon green (514 nm), krypton red (647 nm), and diode (810 nm) lasers, in macular photocoagulation have been widely studied. Hemoglobin, melanin, and xanthophyll are the major pigments that absorb the laser light. Hemoglobin

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<td>Average Laser Parameters Used for Treatment</td>
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absorbs blue-green, green, and yellow wavelengths well, but poorly absorbs red wavelength. Xanthophyll that is located in the inner and outer layers of the macula is good at absorbing the blue-green light; this is one of the reasons why the blue-green laser should not be used in macular photocoagulation. Melanin absorbs all of the wavelengths well and is mainly located in the retinal pigment epithelial cells. The absorption and thermal effect of the lasers with longer wavelengths are more pronounced in the deeper retinal layers. This may attribute to them a theoretical advantage of sparing the inner retinal layers. However, modified grid laser treatment has been effective in treating DME regardless of whether argon blue-green, argon green, krypton red, or diode laser has been used. 20, 21, 24, 37

INTRAOPERATIVE AND POSTOPERATIVE COMPLICATIONS

The most disastrous complication of macular laser treatment is foveal photocoagulation, which occurs when the clinician loses sight of the fovea. Deferring the treatment of the juxtafoveal areas of edema until some extrafoveal resolution of edema occurs delineates the foveal structure better and helps to avoid inadvertent treatment of the FAZ. Meticulous observation of the posterior pole and frequent comparison with the FA permit precise localization of the fovea before and during the treatment. The clinician should correlate the reversed, inverted image through the Mainster lens or the upright image of the Goldmann (Ocular Instruments) posterior pole lens with the projected reference FA at the side of the laser to check vascular landmarks and accurately locate the fovea at all times.

Most patients experience transient blurring of vision immediately after macular photocoagulation. Those who are treated with grid or modified grid therapy usually complain of paracentral scotomas, which tend to diminish with time. However, most patients adapt quickly to these scotomas and report that they gradually diminish or disappear. 2 In the ETDRS, the development of mild paracentral scotomas to the I-2e visual field test object in treated patients was not statistically significant compared with the visual fields of untreated controls. 12 It has been found that the blue-green wavelength used by some investigators damages the nerve fiber layer more than the pure green wavelength does. 36 Thus, green argon or longer wavelength lasers are recommended to reduce the development of these scotomas and to decrease absorption of the blue light by the yellow xanthophyll pigment. When treating eyes with DDME close to the FAZ, longer wavelength lasers such as the diode laser (810 nm) may have a theoretical advantage because the burns will affect deeper layers with relative sparing of the inner neurosensory retina. 31 This, in turn, may reduce the degree of parafocal scotomas usually experienced by patients undergoing laser photocoagulation for DDME. However, to date, clinical studies have not confirmed such a significant advantage of longer wavelength lasers over argon green.

Occasionally, a patient will complain of mild color defects and loss of hue discrimination. Some diabetic patients may have preexisting imperfections in color vision, but this problem may also be exacerbated by macular photocoagulation. 39, 40

In all reports describing the development of choroidal neovascular membranes after focal treatment of diabetic microaneurysms, the argon green or blue-green laser has been applied, particularly with a 50-μm spot size (Fig. 6). 41-44 Small spot size concentrates power in a density sufficient to disrupt Bruch's membrane. Low power and spot size no smaller than 200 μm help to reduce this complication.

Subretinal fibrosis may also result from high-density, small spot size laser photocoagulation. 45, 46 This may also be avoided by using lower power and larger spot size.

Progressive enlargement of photocoagulation scars and atrophy of retinal pigment epithelium have been reported in 5% of patients treated for DME with grid photocoagulation (Table 4). 47, 48 Some of these patients had associated vision loss.

POSTOPERATIVE COURSE AND OUTCOME

Laser photocoagulation for DME is an outpatient procedure. Patients can resume routine activities immediately after the laser photocoagulation. Caution should be advised for driving, as the patients will have blurred vision after the procedure for a transient period of time. No patching of the eye is necessary, unless retrobulbar anesthesia is used. Patients do not require any topical medicine after the procedure.

The ETDRS has shown that laser photocoagulation decreases visual loss from DME by more than 50%. The visual loss was defined as at least doubling of the visual angle on the ETDRS visual acuity chart. In patients with CSDME and no involvement of the center of the macula, visual loss was 13.2% in the treated group versus 22.1% in the control group at 3
years. When the center was involved, the rates were 13.8% and 33.0%, respectively.12

Modified grid laser photoocoagulation, regardless of the laser wavelength used, at least stabilized vision in approximately 80% of the eyes over 2 years. DDME was reevaluated every 3 to 4 months and most of the patients required more than one treatment session.20,21,24

### SPECIAL CASES

**Cataract and DME.** It has been shown that diabetic retinopathy is likely to deteriorate following cataract extraction, mostly in the form of significant macular edema.49-52 Jaffe and Burton studied the progression of diabetic retinopathy in eyes with nonproliferative diabetic retinopathy that had not had previous laser photoocoagulation.49 Six of the eight eyes in their study had vision worse than the preoperative vision and none achieved 20/50 or better vision. Other studies also indicated that visual acuity of 20/40 or better could be achieved in less than 50% of the eyes with existing diabetic retinopathy prior to the surgery.51,52 However, diabetic eyes with little or no retinopathy have a good prognosis.52-54 Recently, Henriksson et al. reported that 89% of diabetics with minimal or no retinopathy achieved 20/40 or better vision following cataract extraction.55 Poor glycemic control was an important factor in progression of retinopathy and patients who progressed had a significantly higher incidence of macular edema.

The incidence of pseudophakic cystoid macular edema (CME) is also higher in diabetics undergoing cataract extraction, and the CME lasts longer than in nondiabetics.56 The pattern of leakage in the FA in these patients can help differentiate pseudophakic CME from DME, although macular edema in these cases is usually a combination of the two.

Treatment of CSDME prior to cataract extraction is not a topic of debate. Additionally, the fact that CSDME appears or progresses significantly following cataract extraction has led us to intervene with treatment for DME even when the edema is not yet "clinically significant" if the patient is scheduled for cataract extraction. We believe that laser photoocoagulation of DME prior to cataract extraction may prevent or decrease both the incidence and the severity of CSDME postoperatively.

**Proliferative diabetic retinopathy and DME.** Various treatment regimens have been advocated for patients with both high-risk proliferative diabetic retinopathy and CSDME. Focal or grid macular photoocoagulation alone has been used in eyes with minimal proliferative disease, with panretinal scatter laser photoocoagulation performed later as proliferative retinopathy progresses.19,21,24 This regimen was developed to avoid exacerbation of the macular edema by the panretinal scatter treatment, which might result from inflammation or altered blood flow in the retinal circulation.37,58

Recently, a combination of modified grid photoocoagulation to the macular area and panretinal scatter treatment to the peripheral retina has been reported to provide good results in eyes with DDME and moderate to advanced proliferative diabetic retinopathy (two or more high-risk factors as defined by the Diabetic Retinopathy Study).59 Modified grid photoocoagulation to the macula and panretinal scatter treatment to the inferior 180° of peripheral retina is performed in one session (Fig. 3); then, 2 to 4 weeks later, a second session of panretinal scatter to the superior 180° of peripheral retina is performed (Fig. 4). Macular edema and proliferative diabetic retinopathy are reassessed by clinical examination and FA at 3- to 4-month intervals to determine whether supplemental grid and/or panretinal scatter treatment is required. A recent report by Lee and Olk demonstrated that after 2 years, macular edema had resolved in 93% of the eyes and proliferative diabetic retinopathy had been reduced in 86% of the eyes when combined modified grid and panretinal photoocoagulation had been performed.59

Treatment of proliferative retinopathy with scatter photoocoagulation first and treatment of the macular edema after the proliferative disease has been stabilized has been advocated as an alternative to combined treat-
ment in eyes with both macular edema and proliferative disease. Interestingly, Gardner et al. reported “improvement” of macular edema after panretinal photocoagulation for proliferative diabetic retinopathy. However, it is important to recognize that macular edema is more likely to be exacerbated during the interval of delay between the treatments using this regimen.

Pregnancy and DME. Progression of diabetic retinopathy during pregnancy is well known. However, the extent of progression seems to be dependent mostly on the severity of the diabetic retinopathy at the onset of pregnancy. Diabetics with mild or no diabetic retinopathy at the onset of pregnancy do not seem to progress significantly. Sunness was able to identify only one case that progressed to proliferative disease in a review of the literature that included 484 patients with mild or no retinopathy at the onset of pregnancy. Slight progression of mild background retinopathy may occur in approximately 10% of pregnant women and is likely to regress in more than 50% of the cases once pregnancy is completed.

Moderate to severe background diabetic retinopathy present prior to pregnancy is likely to progress significantly in the second trimester and regress in the late third trimester and postpartum, especially in the first 6 months. Macular edema can be a significant component of this progression. Fluctuations in the vision of these patients can be attributed partly to exacerbation of macular edema. These patients should be examined during each trimester.

Proliferative retinopathy is the type of retinopathy that is most likely to progress in pregnant women. The panretinal photocoagulation indications that apply to pregnant patients with proliferative retinopathy also apply to nonpregnant patients. The response of the proliferative disease to panretinal photocoagulation is similar in pregnant and nonpregnant diabetic patients. However, proliferative retinopathy in pregnant patients is likely to regress in the postpartum period even without the treatment. Furthermore, a recent study by Kaaja et al. has shown a protective effect of pregnancy on the long-term progression of retinopathy. These factors should not alter the decision to apply panretinal photocoagulation, if the proliferative retinopathy reaches the high-risk stage.

SURGICAL TREATMENT OF DME

Pars plana vitrectomy with stripping of the posterior hyaloid has recently been shown to be beneficial in selected patients with diabetic macular edema. The patients who are likely to benefit the most are those with DDME and attached, thickened, taut and glistening posterior hyaloid membranes. Visual acuity was at least stabilized in approximately 90% of those patients, with two lines or more of improvement occurring in about 50% of the cases.

CASE REPORTS

Case 1. A 35-year-old man had had type 1 diabetes mellitus for the past 20 years. He had had xenon arc
Figure 5. (Case 1.) (A–D) Diffuse diabetic macular edema with marked cystoid macular edema bilaterally.

Panretinal photocoagulation applied to both eyes approximately 5 years earlier. He had experienced a progressive decline in his visual acuity in both eyes over the past 6 months. Visual acuity was 20/100 in each eye. On presentation, he had DDME with marked CME bilaterally (Figs. 5A–D). The patient underwent modified grid laser photocoagulation in both eyes, the right eye with argon green (Fig. 5E) and the left eye with krypton red (Fig. 5F). During a follow-up visit at 3 to 4 months, partial resolution of DDME was seen in both eyes (Figs. 5G–J). Supplemental modified grid laser photocoagulation was given to each eye, with argon green in the right eye (Fig. 5K) and krypton red in the left eye (Fig. 5L). A follow-up visit 3 to 4 months after the second laser treatment revealed complete resolution of DDME clinically and no residual edema in the FA (Figs. 5M–P). Visual acuity had improved to 20/63 in the right eye and 20/50 in the left eye.

Case 2. A 65-year-old woman had been a type 2 diabetic for 15 years. The patient also had systemic hypertension that was controlled with medications. The patient had had argon green panretinal photocoagulation applied to both eyes 1 year earlier. On presentation, visual acuity was 20/63 in the right eye and 20/40 in the left eye. An examination revealed DDME with CME in both eyes (Figs. 6A–D). In addition, a small island of residual neovascularization was seen along the supertemporal arcade in the left eye. The patient underwent modified grid laser photocoagulation with krypton red laser in the right eye (Fig. 6E) and argon green laser in the left eye (Fig. 6F). During a follow-up visit 3 to 4 months later, partial resolution of macular edema was seen in both eyes, but residual thickening involving the FAZ was still present in both eyes (Figs. 6G–J). Supplemental modified grid laser photocoagulation was given to each eye, with krypton red in the right eye.
Figure 5. (Case 1.) (E and F) Modified grid laser photocoagulation with argon green in the right eye and krypton red in the left eye. (G–J) Partial resolution of diffuse diabetic macular edema 3 to 4 months later.
Figure 5. (Case 1.) (K and L) Post-treatment photographs of supplemental modified grid laser photocoagulation given to each eye (with argon green in the right eye and krypton red in the left eye, respectively). (M–P) There was complete resolution of diffuse diabetic macular edema clinically and no residual edema 3 to 4 months after supplemental laser treatment.
Figure 6. (Case 2.) (A–D) Diffuse diabetic macular edema with cystoid macular edema in both eyes and one small island of residual neovascularization along the superotemporal arcade in the left eye. (E and F) Modified grid laser photocoagulation with krypton red laser in the right eye and argon green in the left eye, respectively.
(Fig. 6K) and argon green in the left eye (Fig. 6L). A follow-up visit 3 to 4 months after the second laser treatment showed resolution of DDME clinically and no residual edema in the FA (Figs. 6M–P). Visual acuity had improved to 20/40 in both eyes.

Case 3. A 32-year-old man had had type 1 diabetes mellitus for the past 18 years. He complained of blurred vision in the right eye of 3 months’ duration. Visual acuity was 20/25 in the right eye. An examination showed CSDME (Fig. 7A), with multiple focal leaks seen on the FA (Fig. 7B). Focal treatment was applied to areas of focal leakage using argon green (Fig. 7C). A follow-up visit 3 to 4 months later showed resolution of CSDME (Fig. 7D) and no leakage on the FA (Fig. 7E).

Case 4. A 50-year-old man had been a type 1 diabetic for the past 28 years. The patient had received focal argon green photocoagulation for CSDME 5 years earlier. The patient currently complained of blurred vision in both eyes of 6 months’ duration. Visual acuity was 20/40 in the right eye and 20/32 in the left eye. An examination revealed DDME in both eyes in addition to multiple areas of neovascularization elsewhere (NVE) and preretinal and vitreous hemorrhage in both eyes (DDME combined with high-risk proliferative retinopathy in both eyes) (Figs. 8A–D). The patient underwent the first stage of combined modified grid and inferior 180° panretinal laser photocoagulation with argon green in the right eye (Figs. 8E and 8F) and with krypton red in the left eye (Figs. 8G and 8H). Two to 4 weeks later, the patient underwent a second-stage treatment involving superior 180° panretinal laser photocoagulation with argon green in the right eye (Figs. 8I and 8J) and with krypton red in the left eye (Figs. 8K and 8L). Three to 4 months later, the patient showed complete involution of proliferative retinopathy in both eyes (Figs. 8M and 8N), but residual DDME in the right eye (Fig. 8O).
Supplemental modified grid laser photocoagulation with argon green was given (Fig. 8P). Three to 4 months after the final laser treatment, the patient showed complete resolution of DDME in both eyes (Figs. 8Q–T). One year later, the patient had a completely stable picture in both eyes, with visual acuity...
of 20/40 in the right eye and 20/25 in the left eye (Figs. 8U and 8V).

Case 5. A 58-year-old woman had type 2 diabetes mellitus. She complained of blurred vision in the right eye of 4 months’ duration. Visual acuity was 20/25 in the right eye. An examination demonstrated DDME with intraretinal exudate (Figs. 9A and 9B). Modified grid laser photocoagulation was applied using the diode laser (810 nm) (Fig. 9C). Three to 4 months later, DDME and intraretinal exudate had resolved (Fig. 9D) and minimal leakage in the late frames of FA was demonstrated (Fig. 9E). Visual acuity had improved to 20/20 in the right eye.

REFERENCES


Figure 8. (Case 4.) (E–H) The first stage of combined modified grid and inferior 180° panretinal laser photocoagulation with argon green in the right eye (E and F) and krypton red in the left eye (G and H). (I and J) The second stage of treatment involving the superior 180° panretinal laser photocoagulation with argon green in the right eye.

Figure 8. (Case 4.) (K and L) The second stage of treatment involving the superior 180° panretinal laser photocoagulation with krypton red in the left eye. (M and N) There was complete involution of proliferative retinopathy in both eyes 3 to 4 months later, but residual diffuse diabetic macular edema in the right eye (O).


Figure 8. (Case 4.) (P) Supplemental modified grid laser photocoagulation with argon green. (Q–S) There was complete resolution of diffuse diabetic macular edema 3 to 4 months after the last laser treatment in the right eye.


Figure 8. (Case 4.) (T) There was complete resolution of diffuse diabetic macular edema 3 to 4 months after the last laser treatment. (U and V) There was a completely stable picture in both eyes 1 year later.


Figure 9. (Case 5.) (A and B) Diffuse diabetic macular edema with intraretinal exudate. (C) Modified grid laser photocoagulation applied using the diode laser (810 nm). (D) Diffuse diabetic macular edema and (E) intraretinal exudate that have resolved with minimal leakage seen in the late frames of fluorescein angiography. No further treatment was recommended at this time.


