Laser photocoagulation control of diabetic macular oedema without fluorescein angiography

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Abstract

This study included 40 eyes in 22 diabetic patients with focal macular oedema. Laser photocoagulation was directed at decompensated or leaking microvascular lesions clinically detected without using pretreatment fluorescein angiograms. Post-treatment fluorescein angiograms performed after adequate clinical control of disease showed complete resolution of the macular oedema in 25 eyes (62.5%), whereas persistent leakage from microvascular lesions closer than 500 μ m from the centre of the foveola was noted in 15 eyes (37.5%). These were clinically detected during the pretreatment examination and were found not to impair or threaten the patient's vision. Our data confirm the clinical impression that fluorescein angiography is not necessary for effective treatment and should be used only if necessary.

The beneficial effect of photocoagulation for focal or diffuse diabetic macular oedema has been demonstrated.¹⁻⁷ In most studies fluorescein angiography was performed for pretreatment and post-treatment evaluation.³⁻⁵⁻⁷ However, it is our impression that routine fluorescein angiography is not necessary for effective treatment or adequate clinical control.

We undertook the study of diabetic patients who had focal macular oedema and were treated with argon laser to demonstrate that the majority of the patients could be satisfactorily treated without fluorescein angiography.

Patients and methods

This study comprised 40 eyes of 22 diabetic patients that had focal macular oedema and hard yellow exudates involving or threatening the fovea (Fig 1).

The patients were six men and 16 women whose ages ranged from 85 years to 34 years, mean 57 years. The diagnosis of diabetes had been made in these patients from age 4 to 53 years, average 19.7 years. Sixteen of them used insulin and six of them oral hypoglycaemic agents.

The pretreatment ocular examination consisted of best corrected visual acuity; slit-lamp examination; Goldmann applanation tonometry; examination of vitreous and retina by direct and indirect ophthalmoscopy and contact or non-contact lenses. During the initial examination care was taken to identify the leaking lesions. Decompensated or leaking microvascular abnormalities were clinically identified by means of the direct ophthalmoscope. Lesions which were characterised by transudation or extravasation were observed to be surrounded by oedema of the neurosensory retina, often with an irregularity of the internal limiting membrane light reflex. With a longer course of extravasation the decompensated retinal microvascular abnormalities were bordered by hard yellow exudates, or with a greater duration by cholesterol crystals. If treatment was indicated because of decompensation, other microaneurysms in the area which might later leak were also treated. Pretreatment fluorescein angiograms were performed but were not examined prior to or used during photocoagulation.

All microaneurysms and dilated capillaries, some of which were telangiectatic in areas of oedematous retina, were treated. The treatment was with the 50 to 100 μ m argon blue-green laser of 0.05 to 0.1 second duration, with adequate power to obtain definite whitening within the wall of the microvascular lesion. Care was taken to focus precisely at the level of the lesion to be treated. Repeated burns with adjustment of focus were usually needed to obtain actual whitening in the wall of the leaking lesion itself. Treatment of lesions closer than 500 μ m to the centre of the macula was not done initially if they were not affecting or threatening the patient's vision. When treatment of these lesions was indicated, a spot size of 50 μ m with a 0.02 to 0.05 second duration was used to minimise the size of the laser burn near the foveola.

Follow-up examinations identical to the pretreatment examination were performed every four to six weeks. Subsequent treatment was performed for lesions that had not been sealed during the initial treatment. Post-treatment fluorescein angiography was performed when it was determined clinically that the focal macular oedema was controlled.

The pretreatment and post-treatment fluorescein angiograms were compared for absence or persistence of leakage.

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Figure 1 Pretreatment photograph of the right eye of a 54-year-old woman with diabetes mellitus of 15 years' duration, showing background diabetic retinopathy with decompensated microaneurysms and capillaries with a serous elevation of the neurosensory retina surrounded by hard yellow exudates encroaching on the foveola.

Figure 2 Fluorescein angiogram of the eye showing early hyperfluorescence of the microaneurysms and blockage of the fluorescence by haemorrhage.

Figure 4 Four months post-treatment fluorescein angiogram of the eye showing that the leaking microvascular lesions have been effectively treated.

Results

The pretreatment fluorescein angiograms showed focal leakage from microvascular lesions in all the eyes included in the study (Figs 2 and 3). The pretreatment visual acuity was 20/20 to 20/30 in 20 eyes, 20/40 to 20/60 in nine eyes, 20/70 to 20/100 in six eyes, and 20/200 in five eyes. The average follow-up period was six months.

The post-treatment fluorescein angiograms showed complete resolution of the macular oedema in 25 eyes (62.5%) (Fig 4), while persistent leakage was noted in 15 eyes (37.5%) despite satisfactory clinical control of the disease.

In all these eyes the leakage was from microaneurysms closer than 500 μ m from the foveola that were identified clinically during the pretreatment examination and at the time of treatment.

A change in vision by at least two lines on the chart was required to be considered better or worse. A change of one line was considered the same. At the final follow-up examination visual acuity was found to be improved in six eyes (15%) and unchanged in 34 eyes (85%). Among the improved eyes, the pretreatment visual acuity was 20/30 in one eye, 20/100 in three eyes, and 20/200 in two eyes.

Thirty eyes (75%) had single laser treatment and 10 eyes (25%) had two treatments.

Discussion

In this study 25 eyes (62.5%) showed complete

Figure 3 Late phase fluorescein angiogram of the eye showing extravasation of fluorescein dye round the leaking microaneurysms.

angiographic resolution of the focal macular oedema, whereas 15 eyes (37.5%) showed persistent angiographic leakage. This leakage was from microvascular lesions closer than 500 μ m from the centre of the foveola, which were judged not to interfere with or threaten the patient's vision. On subsequent examinations there was a satisfactory clinical control of the disease. Complete resolution of macular oedema was achieved in only 3% of the treated eyes in a previous study because of microaneurysmal leakage too close to the fovea.⁵ In our cases treatment of lesions closer than 500 μ m from the centre of the macula was undertaken only if they were impairing or threatening the patient's vision. With treatment of these lesions there is some risk of damaging the subfoveal retinal pigmented epithelium or creating symptomatic scotomas.

Our data support the clinical impression that routine fluorescein angiography is usually not necessary for effective treatment and control of focal macular oedema. Microaneurysms and damaged retinal capillaries may leak plasma and be surrounded by oedema and hard yellow exudate. Careful ophthalmoscopy and contact or non-contact lens examination will reveal these foci of microvascular incompetence. Rarely, when leakage persists after photocoagulation or if there is an occult lesion, fluorescein angiography may reveal the refractory area. In patients with profound nuclear sclerosis, which decreases contrast and complicates the visualisation of microvascular detail, fluorescein angiography may be required to detect decompensated microvasculature. Moreover, in patients who had previous laser treatments, areas of chorioretinal atrophy may obscure leaking microaneurysms and require fluorescein angiography.

Background diabetic retinopathy may have several subcategories of classification. Although this study dealt with focal and multifocal patterns of macular oedema caused largely by decompensated microaneurysms, a diffuse telangiectatic pattern resulting from a widespread decompensated microangiopathy may also occur, prompting a more guarded prognosis. An ischaemic background retinopathy is not amenable to laser therapy. It should be empha-

sised that the prognosis also depends on the patient's co-operation and positioning of the head at the time of laser treatment.

During treatment it is of critical importance to focus the laser beam at the level of the microvascular lesion. The treatment may have to be repeated until a white reaction is seen within the wall of the microvascular lesion. It should be emphasised that multiple treatment sessions may be required to achieve adequate clinical control of the disease.

Finally our results show that fluorescein angiography is not absolutely necessary in planning and carrying out photocoagulation in diabetic eyes with focal macular oedema and should not be used routinely, but only if necessary.

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