Treatment of diabetic maculopathy by argon-laser

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Diabetic retinopathy may cause practical blindness in both the proliferative and the background stages of the disease. In the latter, visual impairment is caused by a direct affection of the macular area, a condition to which the term diabetic maculopathy is often applied.

Diabetic maculopathy is composed of two basic types of lesion: lipid deposits and foci of pathological capillaries (Dobree, 1970). Lipoid deposits may appear as clusters of hard exudates, large and small rings (circinate retinopathy), or plaques of lipid material (King, Dobree, Kok, Foulds, and Dangerfield, 1963). In addition to the lipid deposits, macular oedema can be seen. It has been suggested (Gass, 1968; Maumenee, 1968) that lipid deposits, in any form, result from leakage from the diseased capillaries.

Systemic treatment by drugs or local treatment by photocoagulation have been suggested. Drugs to enhance the absorption of the deposits have been used (Caird, Pirie, and Ramsell, 1969), but the visual results were doubtful. Recently, Xenon-arc photocoagulation has been recommended by several authors. Welch (1969) and Spalter (1971) photocoagulated the centres of circinate formations, and claimed remarkable success in improving vision in some cases. Rubinstein and Myska (1972) treated diabetic maculopathy by Xenon-arc photocoagulation of leaking capillaries either at the centre of ring formations or elsewhere, or by perimacular (around the macula) or paramacular (temporally to the macular area) "bombing". Xenon-arc photocoagulation of diabetic maculopathy has also been used by Gass (1971) and by Krill, Archer, Newell, and Chishti (1971).

The use of argon-laser photocoagulation has recently been suggested. Patz (1972) and Patz, Maumenee, and Ryan (1971) advised that it should be used to treat mainly intraretinal microvascular abnormalities (IRMA) in the paramacular area. Zweng, Little, and Peabody (1972) treated 35 eyes with diabetic maculopathy and claimed visual improvement in 71 per cent. Behrendt (1972) treated six cases of diabetic maculopathy by partial occlusion of the small arterioles approaching the macula and claimed visual improvement in all.

It is the purpose of this paper to describe our experience with argon-laser treatment of diabetic maculopathy. The results were evaluated in each case by visual acuity and fluorescein angiography.

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Materials and methods

21 consecutive patients suffering from visual impairment due to diabetic maculopathy were selected for treatment by argon-laser. The examination of each patient included visual acuity, measured several times before and after treatment, fundus drawing, fundus photography, and fluorescein angiography. After treatment each patient was examined at first every second week and then once a month. Results were evaluated by comparison of the preoperative and postoperative visual acuity, clinical fundus picture, and fluorescein angiogram. Each patient had one eye treated, except for two (Cases 3 and 18) in whom both eyes were treated.

Argon-laser therapy was carried out in a similar manner to that suggested by Rubinstein and Myska (1972) for Xenon-arc photocoagulation. Photocoagulation was performed in each case according to the findings in the fluorescein angiogram. It included closure of leaking capillaries, applications to the centre of each circinate formation, and the destruction of areas of IRMA, usually found in the paramacular area along the temporal raphe. In addition, perimacular "bombing" was applied along the upper and lower macular vessels and in the oedematous area. The nearest application to the fovea was 500 μ .

Patients were re-examined 2 weeks after treatment. If the repeated angiogram showed leakage or IRMA not destroyed by treatment, a second treatment was applied. A third treatment was necessary in three cases.

The argon-laser settings were adjusted to each individual case. Generally, however, the upper and lower limits were as follows:

Diameter of argon-laser beam:	50–200 μ
Duration	: 0.05–0.1 sec.
Power	: 75–250 mW

Results

These are shown in the Table (opposite). Of the 23 eyes (21 patients) the visual acuity improved in twelve and remained the same in five. In five other eyes the visual acuity initially improved and then returned to the preoperative level. Only in one case did the visual acuity deteriorate. Improvement in the visual acuity was most marked 4 to 6 weeks after the treatment. Further follow-up of the patients showed a deterioration of the visual acuity in several cases. The improvement in the visual acuity was always reflected in the fundus picture by diminution or disappearance of the macular oedema and regression of the macular and perimacular exudates.

Three patients (Cases 2, 3, and 9) are described in detail to illustrate the findings. All three had marked improvement in the visual acuity of the treated eye.

Case 2, a 61-year-old woman, had suffered from diabetes for 14 years. She was known to have had diabetic retinopathy for 1 year and there had been rapid deterioration in vision for the last 6 months in her left (and only) eye. The right eye was blind from an old-standing rhegmatogenous retinal detachment.

Examination

The visual acuity in the left eye was counting fingers at 1 metre; this was attributed to a diabetic maculopathy consisting of a circinate formation close to the macula, encroaching on the macular area, and causing macular oedema (Fig. 1A, overleaf). The fluorescein angiogram showed multiple spots of leakage and microaneurysms, particularly in the centre of the circinate formation.

Treatment

The patient was treated at three separate sessions.

Table Results in 21 patients

Patient no.	Age (yrs)	Eye	Duration of visual impairment	Pre-operative visual acuity	Postoperative examination				Remarks
					I		II		
					Time post-op. (mths)	Visual acuity	Time post-op. (mths)	Visual acuity	
1	72	L	?	6/60	4	6/24	8	6/60	Temporary improvement
2	61	L	4 mths	1/60	2	6/30 +	9	6/30 +	Marked improvement
(Figs 1 and 2)									
3	66	{ R L	3 yrs	2/60	2	2/60	9	2/60	No change
			2 mths	3/60	2	6/12	9	6/12	Marked improvement
4	60	L	2 yrs	6/60	1	6/21 -	9	6/60	Temporary improvement
5	60	L	?	6/21	2	6/12	7	6/12	Improvement
6	69	R	?	6/60	3	6/18	6	6/60	Temporary improvement
7	59	R	2 yrs	6/60 +	4	6/21 +	6	6/21	Improvement
8	57	R	?	6/60	4	6/21	7	6/60	Temporary improvement
9	50	L	2 mths	6/30	6	6/9	9	6/12	Marked improvement
(Fig. 3)									
10	62	L	6 mths	6/21	1	6/21	4	6/21	No change
11	64	L	6 mths	6/30	1	6/15	6	6/21	Improvement
12	60	L	1 yr	4/60	1	4/60	3	5/60	No change
13	70	L	1 yr	6/30	1	6/15	8	6/21 +	Improvement
14	79	R	5 mths	6/30	$\frac{1}{2}$	6/30	7	6/30	No change
15	75	R	2 mths	2/60	1	6/60	5	6/60	Improvement
16	65	R	1 yr	4/60	2	6/30	6	6/60	Temporary improvement
17	62	R	1 yr	6/60 -	$\frac{1}{2}$	6/60	4	6/21	Improvement
18	61	{ R L	1 yr	1/60	1	4/60	6	6/60	Improvement
			1 yr	4/60	1	4/60	4	4/60	No change
19	69	R	?	6/60	1	6/15	6	6/7.5	Marked improvement
20	51	R	1 mth	6/30	3	5/60	9	3/60	Deterioration
21	69	R	6 mths	6/60	1	6/30	4	6/21	Improvement

Results

The visual acuity increased to 6/30 + and remained so for the 9 months of follow-up. Fig. 1B shows the clinical appearance of the fundus 6 weeks after the first treatment and Fig. 1C 6 months after the last treatment.

Figs 2A and 2B (overleaf) show the post-treatment fluorescein angiograms.

Case 3, a 68-year-old woman, was known to have had diabetes mellitus for 8 years and diabetic retinopathy for 3 years.

Treatment

The visual acuity in the right eye had been 2/60 for the last 3 years because of a macular plaque. This eye was treated by argon-laser but vision remained unchanged.

In the left eye the visual acuity was good until 2 months before treatment, when it was found to be 3/60 because of macular oedema and exudates. This eye was treated by argon-laser, and 1 month later the paramacular circinate formation showed signs of regression with disappearance of the macular oedema.

FIG. 1 Case 2. Left eye
(A) Before treatment;
(B) 6 weeks after first treatment;
(C) 6 months after third treatment. Note marked regression of circinate formation

FIG. 2 Case 2. Fluorescein angiogram of left eye: arteriovenous phase
(A) 6 weeks after first treatment;
(B) 6 months after third treatment. Note increase of area showing a central "curtain effect", without retinal leakage. The black dots inside the paramacular area are laser marks

Results

The visual acuity increased to 6/12 with a parallel improvement in the fluorescein angiogram.

Case 9, a 50-year-old physician, had been known to suffer from diabetes mellitus for 10 years, and to have had diabetic retinopathy for 2 years.

Examination

The visual acuity was 3/60 in the right eye and 6/30 in the left. Fundoscopy showed a macular plaque in the right eye and a circinate formation with macular oedema in the left (Fig. 3A).

(A) Before treatment

(B) 7 months after treatment

FIG. 3 Case 9. Left macular area

Treatment

No treatment was suggested for the right eye. The left eye was treated by argon-laser.

Results

6 weeks later the ring of exudates showed signs of regression, followed by dissolution (Fig. 3B).

The fluorescein angiogram showed a great number of intraretinal microvascular abnormalities, mainly temporal to the macula. After treatment marked diminution of these changes was noted and 1 month later the visual acuity increased to 6/9 with a parallel disappearance of the macular oedema. The visual acuity was 6/12 7 months after treatment.

Discussion

Photocoagulation treatment for diabetic retinopathy has become increasingly popular in recent years and even more so with the introduction into clinical use of the argon-laser. Only large-scale clinical studies conducted for many years will enable us to evaluate its efficacy as regards the proliferative types of diabetic retinopathy, but in our opinion the evaluation of argon-laser photocoagulation in cases of diabetic maculopathy can be done much more rapidly.

Many of the patients described here had improved visual acuity after treatment, with absorption of all forms of exudates: clusters, rings, or plaques. This absorption was much faster than their natural absorption which, for large rings (the most common form of diabetic maculopathy we noticed), takes 2 to 5 years (Dobree, 1970).

At the time of the greatest visual effect of the treatment, which was usually 4 to 6 weeks after application, the absorption of the exudates from the macular area was only in its initial stage (see Figures). Thus, the rapid improvement in the visual acuity should be attributed to some other factor, most probably to the resolution of the macular oedema which, in most cases, is found in background diabetic retinopathy with macular involvement. Clinically, the elimination or disappearance of the macular oedema was noted during the first few weeks after treatment.

The exact rationale of argon-laser treatment in diabetic maculopathy is not very clear. It is conceivable that its major effect is achieved by closing abnormal capillaries, which are the source of the macular oedema and of the lipid exudates (Gass, 1968; Maumenee, 1968; Welch, 1969; Dobree, 1970; Spalter, 1971). The resultant diminution in the intraretinal microvascular abnormality was clearly demonstrated by fluorescein angiography. This destruction of abnormal retinal capillaries could diminish, or even abolish, the deposition of new exudates, thus increasing the speed of dissolution of the rings.

The disappearance of the macular oedema could also be due to the destruction of abnormal and leaking retinal capillaries with resulting decrease in the formation of oedema fluid. It is also possible that the argon-laser beam causes an increase in the absorption of the macular fluid. Peyman, Spitznas and Straatsma (1971) showed that photocoagulation disrupted the normally tightly closed zonulae occludentes, thereby forming new pathways through which retinal or subretinal fluid could be absorbed by the choroid.

No bundle-branch defects were encountered in our patients, even in those cases in which photocoagulation was performed nasally to the macula and in the maculo-papillary bundle area. This agrees with the findings of François and Cambie (1971) and Patz and others (1971). The complication was reported by Apple, Goldberg, and Wyhinny (1973), but it may occur only if strong argon-laser photocoagulation is used.

Patz (1972) suggested the use of a macular fixation device for photography before treatment in macular disease. We did not use the fixation device except in a few patients because we feared its unreliability. The foveal area could nearly always be recognized by biomicroscopy of the fundus.

There was an inverse relationship between the visual results after treatment and the preoperative duration of the visual impairment. Marked improvement was seen in all cases in which the visual deterioration had lasted for only 2 to 4 months. It appears that old-standing macular plaques should not be treated since the chances for improvement are extremely poor.

Summary

21 patients suffering from diabetic maculopathy were treated by argon-laser photocoagulation and were afterwards followed for up to 9 months.

Improvement in visual acuity was obtained in twelve patients, and in five an initial improvement later deteriorated to the preoperative level. In five cases the visual acuity did not change and in one patient it deteriorated.

The improvement in visual acuity was accompanied by the disappearance of the macular oedema and a diminution of the lipid exudates.

The rationale of treatment of diabetic maculopathy by argon-laser photocoagulation is discussed.

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References

- APPLE, D. J., GOLDBERG, M. F., and WYHINNY, G. (1973) *Amer. J. Ophthalm.*, **75**, 595
- BEHRENDT, T. (1972) *Arch. Ophthalm. (Chicago)*, **87**, 629
- CAIRD, F. I., PIRIE, A., and RAMSELL, T. G. (1969) "Diabetes and the Eye". Blackwell, Oxford
- DOBREE, J. H. (1970) *Brit. J. Ophthalm.*, **54**, 1
- FRANÇOIS, J., and CAMBIE, E. (1971) *Ann. Ophthalm.*, **3**, 1201
- GASS, J. D. M. (1968) *Arch. Ophthalm. (Chicago)*, **80**, 583
- (1971) *Trans. Amer. Acad. Ophthalm. Otolaryng.*, **75**, 580
- KING, R. C., DOBREE, J. H., KOK, D'A., FOULDS, W. S., and DANGERFIELD, W. G. (1963) *Brit. J. Ophthalm.*, **47**, 666
- KRILL, A. E., ARCHER, D. B., NEWELL, F. W., and CHISHTI, M. I. (1971) *Amer. J. Ophthalm.*, **72**, 299
- MAUMENEE, A. E. (1968) *Trans. ophthalm. Soc. U.K.*, **88**, 529
- PATZ, A. (1972) *Surv. Ophthalm.*, **16**, 249
- , MAUMENEE, A. E., and RYAN, S. J. (1971) *Trans. Amer. Acad. Ophthalm. Otolaryng.*, **75**, 569
- PEYMAN, G. A., SPITZNAS, M., and STRAATSMA, B. R. (1971) *Invest. Ophthalm.*, **10**, 489, 181
- RUBINSTEIN, K., and MYSKA, V. (1972) *Brit. J. Ophthalm.*, **56**, 1
- SPALTER, H. F. (1971) *Amer. J. Ophthalm.*, **71**, 242
- WELCH, R. B. (1969) "The Treatment of Diabetic Retinopathy" in "Symposium on the Treatment of Diabetic Retinopathy, Airlie House, Warrenton, Virginia, 1968", ed. M. F. Goldberg and J. L. Fine, pp. 563–568. U.S. Department of Health, Education and Welfare.
- ZWENG, H. C., LITTLE, H. L., and PEABODY, R. R. (1972) *Trans. Amer. Acad. Ophthalm. Otolaryng.*, **76**, 990