OUR EXPERIENCE WITH LASER PHOTOCOAGULATION IN DIABETIC MACULAR OEDEMA

Lt Col DP VATS^{*}, Col M DESHPANDE⁺,^{VSM},
Lt Col A BHARADWAJ[#],^{VSM}, Maj R PHOOKEN^{**},
Lt Col A BANERJI⁺⁺, Brig JS SAINI^{##}, Col RG DASH^{***}

(Army Hospital, Delhi Cantt)

ABSTRACT

Diabetic macular oedema, focal or diffuse, is primarily responsible for visual loss in cases of diabetic retinopathy. When to start photocoagulation in such cases has often been debatable. Over a 3 year period, 80 eyes of 80 patients were subjected to argon green macular grid photocoagulation in a randomized clinical study keeping the other eye (80 eyes) as controls in patients with diabetic macular oedema. It was observed that in 5 per cent of such eyes the vision did improve while in 6.3 per cent of patients deterioration of vision was present as compared to 16.3 per cent in control group. It is concluded that laser photocoagulation has a definite role in arresting the progression of diabetic maculopathy and early intervention should be resorted to in case of clinically significant macular oedema.

MJAFI 1997; 53 : 195-198

KEYWORDS: Argon laser photocoagulation; Diabetic retinopathy.

Introduction

Diabetic retinopathy leads to painless, progressive, and permanent blindness but it is preventable. It has been reported that 60-80 per cent of all diabetics develop retinopathy 15-20 years after the diagnosis [1]. This retinopathy may either be of background type (BDR) or of the proliferative type (PDR). Coexisting with both these types of retinopathy there may be macular oedema which is the main cause of visual loss.

It is interesting to note that the prevalence of PDR is higher in younger patients with type I diabetes while macular oedema is more prevalent in older age group with type II diabetes [2]. Diabetic retinopathy is the major cause of blindness in the West due to increased longevity of diabetics as a result of better therapeutic control. Hypertension, associated nephropathy and pregnancy are risk factors in diabetic retinopathy.

The micro-anatomical changes seen in diabetic retina include capillary basement membrane thickening, loss of microvascular intramural pericytes,

formation of capillary microaneurysms, capillary acellularity, break down of blood retinal barrier and neovascularisation.

Diabetic maculopathy can be divided into: (a) ischemic maculopathy and (b) macular oedema, which may be focal or diffuse.

Clinically significant macular oedema is due to either presence of retinal thickening with or without hard exudates involving the centre of fovea or presence of retinal thickening or hard exudates within 500 microns of centre of fovea or presence of retinal thickening or hard exudates of 1 disk diameter (DD) size some part of which is within 1 DD from the centre of fovea.

Laser photocoagulation is the essential modality of treatment for diabetic retinopathy. Laser photocoagulation converts the hypoxic areas to anoxic areas thus reducing the release of vasoproliferative factor, diverts blood supply to vital parts of the retina by changing the haemodynamics, restores blood retinal barrier thus reducing the retinal oedema. It also prevents advanced

^{*}Reader, ⁺Ex-Prof & Head, [#]Associate Prof, ^{***}Prof & Head, Dept of Ophthalmology, Armed Forces Medical College, Pune 411040; ^{*}RMO, President Body Guards, New Delhi; ⁺⁺Classified Specialist (Ophthalmology), Army Hospital, Delhi Cantt 110010; ^{##}Consultant (Med & Endocrinology), Command Hospital (Southern Command), Pune 411040.

complications like vitreous haemorrhage, retinal detachment and neovascular glaucoma.

At what stage of retinopathy laser should be used has remained controversial. A few trials with different treatment protocols including early treatment of diabetic retinopathy have recommended photocoagulation in cases of clinically significant macular oedema with or without visual loss and reported variable results.

We report the details of 80 such cases with diabetic macular oedema subjected to grid and focal photocoagulation.

Material and Methods

This study was conducted at Army Hospital (Referral and Research) Delhi Cantt. A total of 80 patients (160 eyes) were selected for this study from patients attending eye out-patient and diabetic clinics.

All cases were documented cases of type I or type II diabetes mellitus. All patients had either focal or diffuse macular oedema in both eyes diagnosed by ophthalmoscopy and three-mirror contact lens examination. Oedema was confirmed with intravenous 20 per cent sodium fluorescein angiography. No patient was aphakic and none had evidence of central cataract. Eyes with insignificant peripheral lenticular opacities insufficient enough to affect fundus visibility and vision were also included in the study. Patients with other associated causes of visual loss like age-related macular degeneration, high myopia, corneal opacities, glaucoma, previous history of photocoagulation, or age below 20 years were excluded from the study.

A detailed medical history in relation to treatment and duration of diabetes, hypertension, pregnancy, renal failure and a comprehensive systemic examination was done by an endocrinologist.

Ocular exam

Baseline ocular examination assessing the best-corrected visual acuity (VA) at 6 metres distance using the Snellens visual acuity chart was recorded and the patients were divided into 3 groups.

Group A - Visual acuity of 6/9 or better (Good Visual Acuity)

Group B - Visual acuity between 6/12 and 6/18 (Moderate VA)

Group C - Visual acuity between 6/24 and 6/60 (Poor VA)

The macular oedema was classified as focal or diffuse according to fluorescein leakage pattern to decide on the mode of laser treatment. Seven or more leaks or a diffuse leak in the macular area 60 seconds after injection of fluorescein was classed as diffuse. One eye of each patient was selected for laser treatment while the other eye served as its control. If macular oedema and vision were almost equal in both eyes than the right eye of the patient was taken for laser treatment. If there was a visual difference, then the worse eye was taken for treatment.

Treatment techniques

Pupil of the eye to be treated was dilated with a combination of cyclopentolate 1 per cent and phenylephrin 5 per cent. Topical 4 per cent xylocaine drops were used as anaesthesia. Nidek argon laser machine was used for photocoagulation along with Meinster or 3-mirror Goldmann lens.

The treatment techniques consisted of either direct treatment of focal fluorescein leaks and microaneurysms or grid treatment of macular area for diffuse oedema. Direct treatment was done by placing confluent 100 micron burns over the area of discreet leaks. All leaks up to 2 DD away from the centre of macula were treated this way. Any leak closer than 1/2 DD from macular centre was not touched. Time and power were adjusted to obtain faint burns. Microaneurysms were first surrounded by burns and then directly treated.

For diffuse oedema of the macula grid treatment was done.

The fovea was surrounded by very light photocoagulation burns leaving an area of 1/2 DD from the centre of fovea. Inner rows were of 50 micron spot size and outer rows were 100-200 microns. Time was limited to 0.1 s and power was adjusted to obtain grade 1 light burns. All haemorrhages were carefully avoided.

Follow-up

Follow-up was done every 3 months for 1 year and 6 monthly for subsequent years. Visual acuity was recorded in both eyes. Direct and indirect ophthalmoscopy and 3-mirror Goldman lens examination was carried out. Angiography was

repeated and fresh leaks were retreated in the treated eyes.

Results

The pretreatment visual status of the subjects is given in Table 1. Anterior segment and IOP had no significant change in all eyes except a few peripheral lenticular opacities.

TABLE 1

Pretreatment visual status (best corrected visual acuity)

Group	ET group eyes	EC group eyes
Good vision (6/6-6/9)	16	18
Moderate vision (6/12 - 6/18)	36	42
Poor vision (6/24 - 6/60)	28	20

ET - eyes randomised for treatment; EC - eyes randomised for no treatment (control)

At the end of 3 years vision remained the same in 88.8 per cent of eyes while it improved in 5 per cent and deteriorated in 6.2 per cent of the eyes. Three patients had associated hypertension and nephropathy and progressed into proliferative stage of retinopathy in both eyes.

Visual results after 3 years are given in Table 2. In the control group of 80 eyes it was observed that in none of the eyes visual acuity improved while in 83.8 per cent acuity remained the same. There was significant decrease in visual acuity in 16.2 per cent of the eyes.

On comparing the visual acuities in pre-treatment, post-treatment and in control group after 3 years, it was seen that eyes which had good to moderate vision (Gp A & B) before laser treatment

fared better after treatment than eyes which had poor vision to start with. No eye in poor vision group had improvement of vision after laser treatment. The untreated eyes tended to have an increase in the exudates and macular oedema as compared to treated eyes which showed arrest or regression or slow progress of oedema.

There was no major complications except nausea during fluorescein angiography in 10 patients and transient blurring of vision in 6 eyes.

Discussion

The Early Treatment Diabetic Retinopathy Study (ETDRS), till now the largest and longest multicentric follow-up study, has advocated direct treatment of focal fluorescein leaks or grid treatment to areas of diffuse oedema or non-perfused areas to prevent further visual loss. This study further recommended grid treatment in cases of clinically significant macular oedema despite good vision to get the maximal beneficial effect of laser [4-6].

In an another study, Lee and Olk, who followed up 185 patients for 9 years after grid laser for macular oedema showed statistically significant improvement in vision in some cases. Argon green is the ideal wave length (514 nm) for macular grid as it is not absorbed by the xanthophyll in macula thereby preventing phototoxicity [7].

Our study was based on 80 patients of type I or type II diabetes (predominantly Type II) with diabetic macular oedema where one eye was taken as control. The study was conducted to observe and evaluate the effects of grid/focal lasers on diabetic macular oedema in arresting the progress of visual

TABLE 2

Visual results after 3 years in treatment (ET) and in control (EC)

Vision group	Pre treatment	Treated eyes at 3 years			Before treatment	Control eyes at 3 years		
		U	I	D		U	I	D
Good (6/6 - 6/9)	16	13	3	Nil	18	16	Nil	2
Moderate (6/12 - 6/18)	36	34	1	1	42	38	Nil	4
Poor (6/24 - 6/60)	28	24	Nil	4	20	13	Nil	7
Total	80	71	4	5	80	67	Nil	13

U - unchanged; I - improved; D - deteriorated

loss and micro anatomical changes. Majority of our cases had type II diabetes for a mean period of 12-16 years.

In the treated eyes in Group A it was observed that vision remained static in majority of eyes while it improved in 3 eyes after laser treatment which is attributable to the regressing of early macular oedema after laser. While in control eyes of the same patients there was deterioration in 2 eyes and vision remained static in the others. It signifies that early visual loss may improve if laser is started early as observed by Lee and Olk.

When we compare the percentage of visual gain and loss at the end of 3 years in treated and untreated eyes in all the groups we find that visual acuity remained static in 88.8 per cent in treated eyes, against 83.8 per cent in control eyes. Visual improvement was seen in 5 per cent of treated eyes as compared to one in control eyes. Visual deterioration was in 6.2 per cent in treated eyes as compared to 16.2 per cent in control eyes. Though the difference is not statistically significant yet it shows a definite trend towards beneficial effect of laser and may become statistically significant in a larger and wider study corroborating with ETDRS trials. Treated and control eyes of patients with associated hypertension and nephropathy in both groups deteriorated towards proliferative retinopathy thereby subsequently requiring panretinal photocoagulation at the end of study period signifying the poor prognosis in associated hypertension and nephropathy in diabetes.

Following conclusions can be drawn from the observations :

1. Grid/Focal argon (green) laser photocoagulation helps in reducing observable macular oedema and exudates in cases of diabetic macular oedema.
2. In a small percentage of cases, if diagnosed and lasered early, the vision may improve despite the initial loss.
3. Laser photocoagulation slows down the rate of visual loss in the natural course of diabetic retinopathy (maculopathy).
4. Eyes with better visual acuity before photocoagulation have a better visual prognosis after laser, hence photocoagulation should be started early despite normal vision in cases of clinically significant macular oedema.
5. Diabetes with associated hypertension and nephropathy have a comparatively poor visual prognosis despite laser.

This was a moderate term study in a single centre predominantly on Type II diabetes. A long-term, multicentric study on a wider group of patients will bring more information about the management of diabetic retinopathy as the advantage of laser in such patients in undisputed.

It has led us to believe that early laser photocoagulation is beneficial in cases of diabetic retinopathy with all types of macular oedema. We also think that in the future, considering the normal course of the disease, it may become standard practice to laser the retina even in the preproliferative stages especially in young patients with years of productive life ahead of them.

REFERENCES

1. Albert DM, Dryja TP. The Eye, In: Cotran RS, Kumar V, Robbins SL, editors. Robbins Pathological basis of disease, 4th ed. Philadelphia: WB Saunders 1989; 829-30.
2. Klein R, Klein BEK. The Wisconsin epidemiologic study of diabetic retinopathy - II & III. Prevalence age related and risk of diabetic retinopathy. Arch Ophthalmol 1984; 102: 526-32.
3. Daniel MA, Jakobiec FA. Principles and practise of Ophthalmology, 1994 Vol II and V; 747-79 and 2925-33.
4. Early Treatment Diabetic Retinopathy Study Research Group. Treatment techniques and clinical guidelines for photocoagulation of diabetic macular oedema; ETDRS Report No. 2. Ophthalmology 1987; 94: 761-74.
5. Early Treatment Diabetic Retinopathy Study Research Group. Early Photocoagulation for diabetic retinopathy: ETDRS Report No. 9. Ophthalmology 1991; 98: 766-85.
6. Early Treatment Diabetic Retinopathy Study Research Group. Photocoagulation for diabetic macular oedema. ETDRS Report No. 4. Int Ophthalmol Clin 1987; 27: 265-72.
7. Lee CM, Olk RJ. Modified grid laser photocoagulation for diffuse diabetic macular oedema. Ophthalmology 1991; 98: 1594-602.