

Course of Diabetic Retinopathy After Cataract Extraction

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Abstract

The study included 25 diabetic patients with symmetric non-proliferative diabetic retinopathy. Their average age was 58.2 years. The mean duration of diabetes mellitus was 9.7 years. In all cases an uneventful extracapsular cataract extraction was performed on the cataractous eye and the fellow eye was left as a control, to study the course of diabetic retinopathy after cataract extraction. Accelerated retinopathy developed in 16 out of 25 patients (64%). Cataract extraction was associated with asymmetric progression of non-proliferative diabetic retinopathy. In 9 patients (56.2%) accelerated retinopathy developed in the operated-on eye only, while in 7 patients -14 eyes- (43.8%) it developed bilaterally, being more progressive in the operated-on eye. Data analysis throws light on the importance of preoperative and postoperative detailed retinal examination in diabetic patients with non-proliferative diabetic retinopathy who are subjects for cataract surgery, for early detection and management of accelerated retinopathy.

Introduction

DIABETES mellitus is a common disease affecting 1-2% of the population. Cataract occurs earlier in diabetics than in non diabetics [1-2]. The visual prognosis for patients with preexisting diabetic retinopathy who undergo extra-capsular cataract extraction is less favorable than for patients without retinopathy [2-3]. Jaffe et al [4]

reported that cataract extraction was highly associated with asymmetric progression of non-proliferative diabetic retinopathy, being more clinically evident in the operated-on eye than in the fellow eye. In contrast, Sebestyen [5] found that progression of diabetic retinopathy in the operated-on eye occurred concurrently with the same progression in the fellow eye. The aim of this

work is to study the effect of cataract extraction on the course of diabetic retinopathy.

Material and Methods

The study included 30 diabetic patients with symmetric non-proliferative diabetic retinopathy with no evidence of rubeosis iridis, neovascular glaucoma or proliferative diabetic retinopathy. The average age of the patients was 58.2 years. The mean duration of diabetes mellitus was 9.7 years.

All the patients underwent a pre-operative examination including a medical and ophthalmic history. Ophthalmological examinations were performed prior to cataract surgery and included measurement of the visual acuity, slit-lamp biomicroscopy, measurement of the intraocular pressure by applanation tonometry and direct and indirect ophthalmoscopy. Fluorescein angiograms were obtained pre-operatively, 6 months post operatively and at the end of the follow up period.

The preoperative visual acuity of the operated-on (cataract) eyes ranged from 6-60 to hand movement, while the visual acuity of the fellow eye ranged from 6/9 to 6/36.

In all cases an uneventful extra-capsular cataract extraction was performed on the cataract eye and the other eye was left as a control. Neither rupture of the posterior capsule nor vitreous loss was recorded during surgery. Fundus examination was done one week after surgery as a baseline

examination for future comparison with the postoperative retinal course.

The first follow up period (F_1) was from the beginning of the second week after surgery to the end of the first month postoperatively. The second (F_2) was 6 months after surgery and the third (F_3) was 12 to 18 months postoperatively. The average follow up period was 13 months. All the data were recorded in a uniform manner on a standardized reporting sheet.

Accelerated retinopathy was defined as the development of any of the following during the follow up period: New clinically significant macular edema, increased hard exudates or intraretinal hemorrhages or new-onset proliferative diabetic retinopathy. The risk of accelerated retinopathy in the operated-on eye was compared to that in the non-operated-on eye.

Results

The clinical data of 5 patients were excluded from the results of the study as they did not show-up during the follow up period. Therefore data were evaluated from the remaining 25 patients to assess progression of diabetic retinopathy in the operated-on eye compared to the fellow eye.

Accelerated diabetic retinopathy developed in 16 out of 25 patients included in the study during the follow up period (64%). Among these 16 patients, accelerated retinopathy developed only in the operated-on eyes with no clinical evidence of accelerated retinopathy in the remaining 7

patients (14 eyes) accelerated diabetic retinopathy developed in both the operated-on as well as in the fellow eye (Group II) (43.8%).

Among group (I) new clinically significant macular edema developed in 5 eyes, vitreous hemorrhage in 3 eyes, rubeosis iridis in one eye and neovascular glaucoma in one eye. Proliferative diabetic retinopathy developed in 2 eyes of group (I) and was detected during the follow up period (F₂).

The incidence of accelerated retinopathy in group II was 43.8%. This incidence of accelerated diabetic retinopathy was highly asymmetric being more advanced in the operated-on eye compared to the fellow eye. Among group (II), new clinically significant macular edema developed in 5 operated-on eyes and in 4 of the fellow eyes. Vitreous hemorrhages developed in 3 of the operated-on eyes and in only one of the fellow eyes. Rubeosis iridis developed in 2 operated-on eyes and in one of the fellow eyes, neovascular glaucoma developed in only one operated eye.

Proliferative retinopathy developed in 2 operated-on eyes compared to one of the fellow eyes. None of the patients had acceleration of retinopathy in the fellow eye only during the follow up period.

9 of 25 patients (36%) did not develop accelerated retinopathy in either the operated-on eye or in the fellow eye during the follow up period. Among these patients 3 developed transient cystoid macular edema

detected by fluorescein angiography during the follow up period (F₂).

Discussion

It has previously been suggested that removal of the lens in diabetic patients contributes to worsening of the preoperative diabetic retinopathy in operated on eye [6-7].

In the present study the overall incidence of progression of diabetic retinopathy after extra-capsular cataract extraction was 64%. This incidence is slightly lower than that reported by Jaffe et al [4] (74%) and higher than that reported by Pollack et al [8] which was 39%.

On the other hand, Sebestyen [5], in a retrospective study found that progression of retinopathy in the operated-on eye occurred concurrently with progression in the fellow eye. But owing to the fact that Sebestyen's study was retrospective, he most probably was unable to determine the true incidence of progression of retinopathy. Furthermore an objective measure of accelerated retinopathy was not provided.

In the present study, it is clearly evident that if progression of diabetic retinopathy were to develop it always affects the operated-on eye either solely, as in group I, (56.2%) or concurrently with the fellow eye as in group II (43.8%). Moreover, in cases where accelerated retinopathy developed in both eyes, it was always more aggravated in the fellow eye. This fact is

evidenced by the occurrence of higher incidence of vitreous hemorrhage, rubeosis iridis, neovascular glaucoma and even progression to the proliferative stage in the operated-on eye more than in the fellow eye.

Although this study was not intended to compare the relationship between the degree of non-proliferative diabetic retinopathy present preoperatively, the rate of retinopathy progression and the final visual acuity postoperatively, yet progression of retinopathy was minimal in patients with the least amount of preoperative retinopathy and these patients had the best visual prognosis postoperatively.

In the present study, cystoid macular edema developed in 10 out of the 25 operated-on eyes (40%) during the follow-up period. The incidence of cystoid macular edema varied according to the postoperative course of diabetic retinopathy, being more clinically significant in eyes with markedly accelerated postoperative retinopathy. Moreover, the incidence of development of cystoid macular edema is higher in the operated-on eyes than in the fellow eyes.

This incidence is in accordance with the results of Pollack et al [8] who reported that cystoid macular edema developed in 39% of eyes following cataract surgery. On the otherhand, other authors reported that cystoid macular edema developed only in 8% of operated-on eyes with

diabetic retinopathy following cataract extraction [9].

In this instance, it is important to differentiate pseudophakic cystoid macular edema from diabetic macular edema after cataract surgery. The presence of increasing hemorrhages and lipid deposits suggests that the edema is at least partly related to diabetes, so, prompt Laser photocoagulation is indicated during the early postoperative period.

In contrast, in pseudophakic cystoid macular edema, the retinal hemorrhages and lipid deposits usually do not change significantly and fluorescein angiography shows the petalloid pattern of foveal hyperfluorescence characteristic of pseudophakic cystoid macular edema. In this instance, delayed Laser photocoagulation for three to four months should be considered to allow for spontaneous resolution.

Although the mechanism by which cataract extraction contributes to progression of diabetic retinopathy remains to be determined, yet it has been suggested that surgical trauma may contribute to breakdown of the blood retinal barrier beyond that present in diabetic patients leading to development of macular edema, retinal hemorrhages and hard exudates [4].

Although Alpar [10] reported that the presence of an intact posterior capsule may offer some protection against progression of diabetic retinopathy postoperatively. Yet, Jaffe et al [4] reported a high

incidence (74%) of retinopathy progression following cataract extraction in eyes with intact posterior capsule. In the present study none of the patients had a capsular rent at the time of cataract extraction, however retinopathy progressed despite the presence of an intact posterior capsule.

After analysis of the results of this study, the following advices may be considered:

1. All diabetic patients, subjects for cataract surgery should be thoroughly examined both medically and ophthalmologically with detailed preoperative retinal examination. The patients should be informed that if non-proliferative diabetic retinopathy is present preoperatively, there is a high possibility of progression of retinopathy postoperatively which is specifically related to cataract extraction. Accordingly the visual prognosis is significantly worse than if no retinopathy is present.
2. It may be wise to delay cataract extraction as long as possible in patients with non-proliferative diabetic retinopathy preoperatively, for fear of worsening of the retinal status postoperatively.
3. Detailed postoperative retinal examination, within the first postoperative month - after cataract extraction is advised in patients with preoperative diabetic retinopathy to detect and treat any

acceleration of retinopathy as early as possible.

4. Prompt Laser Photocoagulation is advised during the early postoperative period in patients who develop significant macular edema postoperatively.

References

1. KLEIN BEK, KLEIN R, MOSS S. E.: Prevalence of cataract in a population - based study of persons with diabetes mellitus. *Ophthalmology*, 92: 1191 - 6, 1985.
2. STRATSMAN B.R., PETTIT T., WHEELER N., and MIYAMASA, W.: Diabetes mellitus and intraocular lens implantation. *Ophthalmology*, 90 : 366, 1983.
3. CHENG H. and FRANKLIN S. L.: Treatment of cataract in diabetics with and without retinopathy. *Eye*, 2: 607, 1988.
4. JAFFE G. J., BARTON T.C., KUHN E., PRESCOTT A. and HARTZ A.: Progression of non proliferative diabetic retinopathy and visual outcome after extracapsular cataract extraction and intraocular lens implantation. *Am. J. Ophthalmol.*, 114 : 448 - 456, 1992.
5. SENESTUEN J. G.: Intraocular lenses and diabetes mellitus. *Am. J. Ophthalmol.*, 10 : 452, 1986.
6. JAFFE G. J., and BARTON T. C.: Progression of non-proliferative diabetic retinopathy following cataract extraction. *Arch. Ophthalmol.*, 106: 745, 1988.

7. POLLACK A., DOTAN S., and OLIVER M.: Progression of diabetic retinopathy after cataract extraction. *Br. J. Ophthalmol.*, 75: 547, 1991.
8. POLLACK A., LEIBA H., BUKELMAN A., ABRAHAMI S., and OLIVER, M.: The course of diabetic retinopathy following cataract surgery in eyes previously treated by Laser photocoagulation. *Br. J. Ophthalmol.*, 76: 228- 231, 1992.
9. RUIZ T. S., SAATCI O. A.: Posterior chamber intraocular lens implantation in eyes with inactive and active proliferative diabetic retinopathy. *Am. J. Ophthalmol.*, 111 : 158-162, 1991.
10. ALPAR J. J. : Cataract extraction and diabetic retinopathy. *Am. Intraocular Implant Soc. J.*, 10: 433, 1984.